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THE EFFECTS OF STIMULUS REPETITION, PAIRING, INTENSITY AND INTERTRIAL INTERVAL ON THE CARDIAC RATE REPONSE IN *MACACA MULATTA*

by

Paul Ronald Bindler

A dissertation submitted to the Graduate Faculty in Psychology in partial fulfillment of the requirements for the degree of Doctor of Philosophy, The City University of New York.

1978

This manuscript has been read and accepted for the Graduate Faculty in Psycology in satisfaction of the dissertation requirement for the degree of Doctor of Philosophy.

December 20, 1978
date

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ABSTRACT

THE EFFECTS OF STIMULUS REPETITION, PAIRING, INTENSITY, AND INTERTRIAL INTERVAL ON THE CARDIAC RATE RESPONSE IN MACACA MULATTA

by

Paul R. Bindler

Adviser: Professor William N. Schoenfeld

Changes in the cardiac rate response to repetitions of the unconditional stimulus (UCS) alone, and pairings of the conditional stimulus (CS) and UCS, at different intensities of UCS were studied in twelve chair-restrained rhesus monkeys. CS consisted of a 10-sec light, while UCS was an electric shock. Three groups received either 13.5, 4.0, or 0.5 mA as UCS intensity. Each subject was exposed to all experimental conditions. The experiment consisted of six main experimental stages: 1) 10 sessions of UCS-alone presentations, 2) 10 sessions of CS-UCS pairings, 3) 1 session of UCS-alone presentations, 4) 10 sessions of CS-alone presentations, 5) 10 sessions of UCS-alone, and 6) 5 sessions of CS-UCS pairings in which UCS intensity was reversed for the 0.5 and 13.5 mA groups. An additional group (denoted NP-4.0 mA) received a 4.0 mA UCS, and received all the experimental manipulations excluding stage 2. The two 4.0 mA groups underwent a partial replication of the first three stages with the intertrial interval (ITI) time reduced.

During every trial, cardiac interbeat time (IBI) was recorded in successive 2-sec time periods, beginning with 2-sec pre-CS, 10-sec during CS, and 30-sec post-UCS. Measures of the magnitude, temporal form, and latency of the peak cardiac

conditional response (CR) and unconditional response (UCR) were based on mean IBI values for successive 2-sec recording periods. When CS was not present, only the mean IBI 2-sec prior to UCS was determined.

The magnitude of the acceleratory phase of the UCR decreased over sessions of UCS-alone presentations, but not within sessions. Longer ITIs facilitated intersession habituation. The amount of decrease in the magnitude of the UCR was positively related to the overall number of UCS-alone presentations. Furthermore, mean IBI pre-UCS level decreased over sessions. When pre-UCS levels were statistically equated over sessions the decrease in UCR magnitude was larger than the unadjusted raw data. Finally, the form of the UCS intensity-habituation function depended on the measure of habituation: 1) UCR magnitude in the final session of UCS-alone presentations showed an inverse relationship between UCR habituation and UCS-intensity, and 2) percentage or absolute amount of change scores showed a positive relationship between UCR habituation and UCS intensity.

The effects of UCS-alone presentations and conditioning on UCR magnitude could be differentiated. UCS-alone presentations resulted in a larger decrement in UCR magnitude than conditioning. Furthermore, UCR diminution during conditioning was primarily a function of UCS repetition during conditioning. The CS attenuated the effect of UCS presentations through its effect on baseline (pre-UCS) level: the CR elevated pre-UCS levels; the greater the increase in pre-UCS level, the smaller the UCR magnitude. The amount of UCR diminution for the moderate and high UCS intensity groups during conditioning, as well as the

amount of UCR recovery to UCS-alone presentations after conditioning, was positively related to both UCS intensity and the number of UCS-alone presentations. The finding that UCR diminished during conditioning as the CR increased in magnitude accounts, in part, for reports in the literature that CR and UCR differ in many reponse characteristics. It was found that after UCR habituation CR and UCR were similar in temporal form, magnitude, and latency.

Presentations of CS-alone (extinction) after conditioning did not affect UCR magnitude when UCS-alone presentations were resumed. This finding was attributed to either a) the difference in the generalization gradient between CS and UCS, or b) the possibility that UCR reached a lower limit by this phase of the experiment, in which case additional changes in UCR magnitude would not be detected.

In the final experimental stage of reconditioning, the group initially conditioned at the low UCS intensity showed an increase in CR magnitude when reconditioned at the high UCS intensity. The group initially conditioned at the high UCS intensity showed a decrease in CR magnitude when reconditioned at the low UCS intensity.

Acknowlegments

I would like to thank my teacher and adviser, Professor William N. Schoenfeld, for his advice, support, and encouragement throughout my years as a graduate student. I am especially thankful for having the opportunity to be exposed to his thinking in the experimental analysis of behavior, as well as for the time and guidance he has devoted to me personally. I would also like to thank Dr. Ronald M. Kadden for the many hours he has spent reviewing and editing this work, and discussing the ideas herein.

I would like to dedicate this thesis, firstly, to my father, of blessed memory. His guidance in my earlier years has constantly remained with me. Above all, this work is dedicated to my mother. It is to her that the majority of the credit must go for whatever positive qualities I may have. Her supervision and encouragement ultimately made this work possible. She is truly, as the Psalmist states, a "Woman of valor."

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Introduction

The effects of stimulus repetition have been studied in a wide variety of contexts. The literature on stimulus repetition can be categorized into two broad categories. One comprises those stimulus conditions in which repetition results in an increase in response magnitude (e.g., "acquisition", "sensitization"); the second comprises conditions in which repetition results in a decrease in response magnitude (e.g., "habituation", "extinction", "UCR diminution"). The main interest of the present study is in the second category.

Historically, stimulus operations resulting in a decrease in responsiveness were of interest to theorists since they represented a means of eliminating responses from the behavioral repertoire (Keller and Schoenfeld, 1950; Pavlov, 1927;) While many of the conditions under which repetition of a stimulus results in a decrement in response magnitude are operationally similar, theorists have often thought that these operations produce distinct and unique behavioral phenomena. An instance of this is the classic distinction between habituation and extinction. The response decrements subsumed under these two categories result from the same stimulus operation, that is, the repetition of a single stimulus. However, habituation is typically associated only with unconditional responses (UCR) while extinction is associated with conditional responses (CR) (Kimmel, 1973).

Operational similarities among different procedures do not compel the conclusion that the behavioral effects are similar. However, as many of the parameters of the procedures that result in response diminution have not been systematically explored, it

remains to be seen whether or not the behavioral distinctions are tenable (cf., Schoenfeld, 1972, 1976). One purpose of the present study was to clarify the operational similarities of various procedures to repetitively present stimuli, and to determine whether they result in systematic changes in behavior.

The literature pertinent to the present paper is quite diverse. For the sake of coherence, the review of the literature has been partitioned into two sections based primarily on the manner in which these phenomena have been categorized in the literature. The first pertains to response decrements occurring as a result of repetitions of a single stimulus; the second concerns decrements in response magnitude resulting from paired stimulus presentations.

Single Stimulus Presentations

Repeated presentations of a single stimulus constitutes the simplest form of stimulus schedule. The finding that response magnitude often decreases ("habituates") with successive presentations of a single stimulus has been confirmed in studies employing a wide variety of organisms, response measures, and stimulus modalities (Peeke and Herz, 1973; Ratner, 1970). While the ubiquity of the phenomenon is well established, nevertheless there are conditions under which response magnitude does not change, and may, in fact, increase with repeated stimulation (e.g., Geer, 1964; Raskin, Kotses, and Bever, 1969). This diversity of effect has led to the investigation of the stimulus parameters determining the effects of single stimulus repetition on response magnitude.

The effects of stimulus repetition, particularly on

autonomic nervous system responses, has assumed importance in a number of contemporary theoretical formulations (Graham, 1973; Graham and Clifton, 1966; Lacey, 1967; Sokolov, 1963; Thompson, Groves, Teyler, and Roemer, 1973). Autonomic responses have been of interest to theorists since they are considered to reflect organismic states or processes, such as "arousal", "orienting", and "defensiveness" (Graham, 1973; Pribram and McGuinness, 1975). Of particular interest to theorists is the cardiac rate response because it is considered to be a good measure of these underlying states and it is a biphasic response (Graham and Clifton, 1966; Lacey, 1967; Sokolov, 1963). The bifurcation of the cardiac rate response into two clearly differentiable phases has been thought to represent two separate responses reflecting distinct behavioral processes (Graham and Clifton, 1966; Sokolov, 1963). Schoenfeld (1972, 1976) has criticized "process" oriented explanations of behavior on a variety of theoretical grounds. He suggests, as a theoretical alternative, consideration of the functional relationships between changes in behavior and the systematic manipulation of the physical and temporal parameters of stimuli (Schoenfeld, 1972).

One stimulus variable receiving attention has been the intensity of the eliciting stimulus, since this variable affects the temporal form of the cardiac response as well as its magnitude and direction of change to repeated presentations of the stimulus. Over a period of approximately 7-10 sec after stimulus onset the temporal form of the cardiac rate response in a number of different species is typically biphasic (acceleration-deceleration). Both response phases are positively related to stimulus intensity. These effects have been reported for

tones in the range of 60-120 db (Berg and Graham, 1970; Chase and Graham, 1967; Fehr and Stern, 1965; Hart, 1974; Jackson, 1974; Jackson and Graham, 1973; Keefe and Johnson, 1970; Lang and Hnatiow, 1962; McDonald, Johnson, and Hord, 1964; Raskin, et al., 1969; Roessler, Collins, and Burch, 1969; Smith and Strawbridge, 1969; Uno and Grings, 1965), and electric shocks in the .8-20 mA range (Church, LoLordo, Overmier, Solomon, and Turner, 1966; Deane, 1961; Deane and Zeaman, 1958; Ginsberg and Thysell, 1966; Katcher, Solomon, Turner, LoLordo, Overmier, and Rescorla, 1969; Lykken, Macindoe, and Tellegen, 1972; Schneiderman, Van Dercar, Yehle, Manning, Golden and Schneiderman, 1969; Wilson, 1964). The range of species in these studies includes humans, rabbits, rats, monkeys, and dogs. Studies employing tones in the range of 20-60 db report only monophasic decelerations(Berg and Graham, 1970; Black, 1964; Davis and Buchwald, 1957; Jackson and Graham, 1973; Notterman, Schoenfeld, and Bersh, 1952; Raskin et al., 1969; Uno and Grings, 1965; Wilson, 1964). Monophasic decelerations have also been reported for low intensity shocks (approximately .5 mA) (Ginsberg and Thysell, 1966; Stern and Word, 1961).

In the studies cited above, when the stimulus was repeatedly presented, the acceleratory phase of the response was generally found to either remain unchanged or increase in magnitude, while the deceleratory phase habituated rapidly (e.g., Jackson, 1974; Uno and Grings, 1965). Studies of stimulus repetition were primarily those conducted with tones. Studies finding only monophasic decelerations to tones in the 20-60 db range report

habituation within a few trials (e.g., Davis and Buchwald, 1957; Jackson and Graham, 1973).

Graham has suggested that the studies of the effects of single stimulus repetition on the cardiac rate response support the view of cardiac acceleration and deceleration representing two response classes (Graham, 1973; Graham and Clifton, 1966). She feels that cardiac decelerations meet Sokolov's (1963) two main criteria for the "orienting response" (OR): elicitation by low stimulus intensities and rapid habituation. Cardiac accelerations, on the other hand, qualify for the "defensive response" (DR), since they are elicited by high intensity stimuli and remain unchanged, or increase in magnitude, with stimulus repetition. Graham further suggests that biphasic responses are actually a composite of the OR and DR, with the DR (acceleration) overlapping the initial portion of the OR (deceleration). Schoenfeld (1976) has recently questioned the conceptualization of the cardiac rate response as consisting of different responses. In addition to this theoretical critique, there are also data that call into question the empirical basis for establishing two separate response categories.

To begin with, there are studies concerning the relationship between stimulus intensity and the temporal form of the heart-rate response contradicting findings from the laboratories cited previously. Several investigators, for example, have reported cardiac decelerations to tone intensities in the 60-120 db range (Davis, Buchwald, and Frankmann, 1955; Meyers, 1969; Meyers and Gullickson, 1967; Rudolf, 1965). Additionally, other investigators have reported cardiac acceleration to relatively "low"

intensity stimuli (20-60 db) (Germana and Klein, 1968; Roessler, Alexander, and Greenfield, 1963; Roessler et al., 1969). These findings suggest that variables other than stimulus intensity determine the temporal form of the cardiac rate response, and may account for the OR/DR dichotomy without reference to underlying processes.

Pre-stimulus heart-rate level, and its relationship to response magnitude, i.e., the so-called "law of initial values" (LIV), may account for some of the discrepancies in the literature concerning the form of the cardiac rate response. Originally defined by Wilder (1957; 1962),LIV specifies the following relationship: given a standard stimulus intensity, and a standard measurement period, the response magnitude will be systematically related to pre-stimulus levels. Several investigators have demonstrated that the magnitude, as well as the direction, of the cardiac rate response is a function of pre-stimulus level (Church et al., 1966, Ginsberg and Thysell, 1966; Ramsay, 1970; Roessler et al., 1969, Shearn, 1967).

There have also been conflicting data concerning the stimulus intensity-habituation function. To begin with, the relationship between habituation and stimulus intensity depends on the measure of habituation selected. For cardiac accelerations, when response magnitude to final stimulus presentations is the index of habituation, there appears to be an inverse relationship between habituation and intensity (Berg and Graham, 1970; Germana and Klein, 1968; Jackson and Graham, 1973; Raskin, et al., 1969). However, since the magnitude of cardiac acceleration is positively related to stimulus intensity (Church et al., 1966;

Jackson and Graham, 1973; Raskin et al., 1969; Uno and Grings, 1965), final response magnitudes may simply reflect the relationship between stimulus intensity and response magnitude during final stimulus presentations (cf., Roessler et al., 1969). If the response does not reach a "no-response" or operant level by the final stimulus presentations, there is no way of determining changes in response magnitude over trials since final response magnitude is not compared to response magnitude to initial stimulus presentations. Indeed, when the amount of change in cardiac acceleration is compared between initial and final trials, the magnitude of cardiac acceleration generally remains either unchanged or increases within a wide range of tone intensities (60-120 db) (Berg and Graham, 1970; Hart, 1974; Roessler et al., 1969; Uno and Grings, 1965).

When final response magnitudes have been used to measure the habituation of cardiac deceleration, the typical finding has been that they habituate to the same "no-response" or operant level over a wide range of tone intensities (20-60 db) (Berg and Graham, 1970; Hart, 1974; Jackson, 1974; Raskin et al., 1969). Thus, employing the criterion of final response level, there appears to be no systematic effect of stimulus intensity on the habituation of cardiac decelerations (cf., Graham, 1973; Jackson, 1974). When the amount of change in cardiac deceleration is compared over trials (i.e., the difference between initial and final response magnitudes), there appears to be a positive relationship between the degree of response decrement and intensity (Berg and Graham, 1970; Hart, 1974; Jackson, 1974; Jackson and Graham, 1973). However, this positive relationship

between habituation and intensity may simply reflect the fact that higher stimulus intensities elicit larger magnitude decelerations to initial presentations of the stimulus (Davis et al., 1955; Jackson and Graham, 1973; Meyers, 1969; Rudolf, 1965). Given that decelerations commonly reach the same "no-response" criterion for habituation differences in the amount of response decrement would be, in part, determined by initial response magnitudes (cf., Davis and Wagner, 1968; Graham, 1973; Jackson, 1974). Graham (1973) has suggested that a percentage measure of habituation would equate for differences in initial response magnitude at different levels of intensity.

Other laboratories report findings calling into question the differential effects of stimulus repetition on cardiac acceleration and deceleration. While the majority of the studies in the literature demonstrate that the magnitude of cardiac accelerations remain unchanged, or increase with stimulus repetition, and cardiac decelerations habituate rapidly, there are a few studies that have provided contradictory data. Several investigators, for example, have reported decreases in the magnitude of cardiac acceleration to single stimulus repetition (Bagshaw and Benzies, 1968; Dykman et al., 1959; Germana and Klein, 1968; Meyers, 1969; Raskin et al., 1969; Schneiderman et al., 1969). Additionally, researchers have reported failures to find decreases in the magnitude of cardiac deceleration to repeated stimulus presentations (Bagshaw and Benzies, 1968; Germana and Klein, 1968; Meyers, 1969; Roessler et al., 1969). Finally, there are reports that neither cardiac acceleration nor deceleration changed with stimulus repetition (Bloch-Rojas,

Toro, and Pinto-Hamuy, 1964; Galbrecht, Dykman, Reese, and Suzuki, 1965; Holdstock and Schwartzbaum, 1965).

The reconciliation of some of these discrepant findings concerning the effect of stimulus repetitions might be found in considering variables other than the intensity of the eliciting stimulus. Of particular significance are findings that components of the cardiac response occurring close to stimulus onset tend not to habituate, while responses later in the interstimulus interval (ISI) often undergo rapid habituation (Berg and Graham, 1970; Bloch, 1970; Germana and Klein, 1968; Graham and Jackson, 1973; Meyers, 1969; Smith and Strawbridge, 1968, 1969; Raskin et al., 1969). These findings suggest that the relationship between ISI and response latency is an important determinant of the effects of stimulus repetition on a particular phase of the cardiac response (cf., Lockhart, 1966).

Gatchel and Lang (1974)and Gatchel (1975), for example, found an inverse relationship between the amount of habituation of cardiac deceleration and ISI; a similar relationship has been found for other autonomic nervous system responses as well (Coombs, 1938; Germana, 1968; Schaub, 1965; Winokur, Stewart, Stern, and Pfeifer, 1962). Price and Geer (1972) specifically studied the relationship between ISI and response duration using the galvanic skin response (GSR), and found that responses early in the ISI did not decrease with stimulus repetition, while responses later in the interval did. Depending on ISI length successive presentations of the stimulus could occur at different temporal locations in the cardiac rate response, if the response

does not completely recover to baseline after a given presentation of the stimulus. The point in the heart-rate response at which the stimulus occurs may partly determine the effects of stimulus repetition (cf., Winokur et al., 1962).

The variable of ISI may also provide the basis for an account of the finding that in some cases cardiac accelerations show no habituation even after a large number of trials or sessions (Fehr and Stern, 1965; Galbrecht et al., 1965), while in other instances rapid habituation occurs, even after a small number of trials (Bagshaw and Benzies, 1968; Germana and Klein, 1968; Raskin et al., 1969). For example, both Davis (1970), studying the startle-response in rats, and Gatchel (1975), studying the heart-rate and skin conductance responses in humans, reported that short ISIs facilitated intrasession habituation, while long ISIs facilitated intersession habituation.

Finally, LIV has also complicated the determination of the effects of stimulus repetition on the phases of the cardiac response. Ginsberg and Thysell (1966) reported that pre-shock heart-rate level decreased within a session of repeated stimulus presentations (cf., Shearn and Clifford, 1964). Other investigators have reported decreases in basal heart-rate over sessions of stimulus presentations as well (Cohen and MacDonald, 1971; Galbrecht, et al., 1965; Ramsay, 1970). While these investigators have reported systematic shifts in baseline heart-rate over trials and sessions, it is not clear how these shifts are related to the effects of repeated stimulus presentations on the magnitude of the cardiac rate response. Given that the typical magnitude measure of the heart-rate response has been the

difference between pre- and post-stimulus levels, decreases in baseline heart-rate over trials or sessions would tend to accentuate the magnitude of cardiac accelerations and to attenuate cardiac decelerations (cf., Germana, 1968). Such shifts in pre-stimulus (baseline) heart-rate could lead to different interpretations of the effects of stimulus repetition.

Part of the present experiment was designed to clarify some of the effects of single stimulus repetition on the cardiac rate response. While shock has been typically employed as a UCS in conditioning experiments, little is known about the effects of repetitive presentations of shock-alone, particularly in the rhesus monkey. Three shock intensities were employed to determine the effect of intensity and repetition on the magnitude, latency, and temporal form of the cardiac rate response.

The majority of the studies reporting that the acceleratory phase of the cardiac rate response shows little or no habituation have generally employed a small number of trials (approximately 10-30 stimulus presentations). However, if cardiac accelerations habituate slowly, then prolonged stimulus exposure might be required before the effect of stimulus repetition becomes apparent. Therefore, an extended series of shock repetition was presented over a number of sessions.

Given that most studies of cardiac rate response habituation have not considered the effects of LIV (the "law of initial values"), it may be that different conclusions

have been drawn in the literature as to the effects of stimulus repetition and intensity since both these variables may affect pre-stimulus (baseline) levels as well as response magnitude. Therefore, the relationship between pre-stimulus heart-rate at each shock intensity and changes in the magnitude of the heart-rate response to stimulus repetition was analyzed.

The suggestion that biphasic cardiac responses are a composite of two distinct responses rests on the finding of a differential effect of stimulus repetition on the two response phases. However, this differential responsivness may be due, in part, to the relationship of ISI and response latency. ISI was manipulated to determine its relationship to the effects of stimulus repetition and intensity on the cardiac rate response. ISI was also explored as a means of accounting for differences reported in the literature between intersession and intrasession habituation.

There are also conflicting reports in the literature as to the form of the stimulus intensity-habituation function. These discrepancies may be due to differences in cross laboratory measures of habituation. Therefore, the relationship between shock intensity and habituation was determined, employing several different measures of habituation.

Paired Stimulus Presentations

A more complex form of stimulus presentation schedule can be derived from the basic schedule of single stimulus presentations by "intruding" a second stimulus, S2, which differs in one or more physical characteristics from the

original stimulus, S1 (Schoenfeld, 1972). The physical parameters of either S1 or S2 may be varied, as well as their temporal relationship, and it is possible to determine the effects of both stimuli on the response over successive repititions of S1 and S2. Given the intrusion of S2, the paradigm of Pavlovian conditioning can be readily derived. To achieve this end two conditions must be fulfilled: a) S1 (CS) must be a stimulus that either initially elicits no response, or a response that habituates within a few presentations of CS-alone, while S2 (UCS) elicits a relatively large magnitude response that shows relatively little habituation with repetition of the stimulus, and b) the temporal relationship between the stimuli is such that CS precedes UCS (Keller and Schoenfeld, 1950; Pavlov, 1927). It can readily be recognized, however, that such an arrangement represents only part of a continuum of possible stimulus relationships that can be derived by manipulating the temporal location of both CS and UCS (Catania, 1971; Schoenfeld, 1972; Turkkan, 1977). Historically, the main interest has been the response to CS, primarily because the acquisition of the CR was seen as representing a form of "learning" (Schoenfeld, 1972, 1976). Less interest was generated toward the response to UCS, since it did not appear that the UCR was readily modifiable, and was presumed necessary only insofar as it provided the basis for the CR (Dykman, 1967; Kimmel, 1973).

However, in many early classical conditioning studies it was observed that with extended presentations of the CS-UCS pair the UCR decreased in magnitude (e. g., Hilgard, 1933).

However, this phenomenon of UCR diminution did not receive specific consideration until the work of Dufort, Guttman and Kimble (1958) on the eyelid response. The phenomenon of UCR diminution during conditioning subsequently received confirmation in a number of laboratories employing a variety of response measures (galvanic skin response (GSR), Badia and Defran, 1970; GSR, Baxter, 1966; eyelid response, Fishbein and Levy, 1968; heart-rate response (HPR), Fitzgerald, 1966; GSR and volume-pulse change response (VPC), Furedy and Klajner, 1972; GSR, Grings and Schell, 1969a, b, 1971; eyelid response, Kimble and Ost, 1961; GSR, Kimmel and Pennypacker, 1962; GSR, Lykken et al., 1972; skin resistance response (SRR), Martin, Levy, and Slubicka, 1975; GSR, Morrow, 1966; GSR, Peeke and Grings, 1968; GSR, Schell and Grings, 1971). These findings demonstrate that the UCR is, in fact, modified during conditioning.

Theorists attempting to account for UCR diminution have primarily considered the effect of CS on UCR, each theorist ascribing to CS a specific "function" as the determinant of the decrement in UCR. For example, several theorist suggest the CS becomes a "conditioned inhibitor" during conditioning and the decrease in the magnitude of UCR is a result of the inhibitory effects of CS (Kimble and Ost, 1961; Kimmel, 1966; Rescorla, 1969). Others suggest that CS acts as a "warning signal" or "perceptual cue" for the oncoming UCS (Epstein, 1973; Grings, 1960, 1965; Lykken, 1962; Lykken and Tellegen, 1974).

The view that CS acquires control over the UCR is based on several lines of evidence. The finding that the removal

of CS after a number of conditioning trials (during which UCR decreases in magnitude) results in an increase in the magnitude of UCR constitutes one line of evidence (Baxter, 1966; Duffort and Kimble, 1958; Fitzgerald, 1966; Grings and Schell, 1969a, b, 1971; Kimble and Ost, 1961; Kimmel and Pennypacker, 1962; Morrow, 1966; Peeke and Grings, 1968). Another is based on manipulating some parameter of CS, such as CS intensity (Fishbein and Levy, 1968; Grings and Schell, 1969a), or some aspect of the temporal relationship between CS and UCS, such as the length or variability of the CS-UCS interval (Dufort and Kimble, 1958; Grings and Schell, 1969a, 1971; Kimble and Ost, 1961; Peeke and Grings, 1968). These studies demonstrated that changes in the physical properties of CS or its temporal relationship to UCS are functionally related to changes in the magnitude of UCR (e.g., the decrease in UCR magnitude is positively related to CS intensity).

The finding of a particular relationship between a parameter of CS and UCR diminution does not preclude the possibility that the decrement in UCR is determined by the repetition of the CS-UCS pair, and not the result of some "functional property" of CS (e.g., "inhibitory" or "warning" stimulus). If UCR diminution were a result of the repetition of the CS-UCS pair, then the decrease in the magnitude of the response to UCS during conditioning may be comparable to the decrement in the reponse to UCS-alone presentations. Thus, stimulus parameters that determine the effects of a single stimulus on UCR may also determine the effects of CS-UCS repetition. For example, the magnitude of the decrement in the UCR

over trials is inversely proportional to the ISI in the single stimulus case (Gatchel, 1975), and the length of the CS-UCS interval in the paired stimulus case (Grings and Schell ,1969a). Such findings suggest that "UCR habituation" (single stimulus) and "UCR diminution" (paired stimuli) are similar phenomena.

The contention that UCR diminution is a result of repetition of the CS-UCS pair is supported by the finding of Kimmel and Pennypacker (1962) that the magnitude of the decrement is positively related to the number of presentations of the CS-UCS pair. Additionally, the temporal relationship between CS and UCS determines the effects of repetition. Groups receiving either unpaired CS and UCS presentations, or UCS-CS pairings ("backward" conditioning), generally show smaller decreases in UCR magnitude than groups receiving CS-UCS presentations (Baxter, 1966; Fitzgerald, 1966; Grings and Schell, 1969a, b, 1971; Morrow, 1966; Peeke and Grings, 1968; Schell and Grings, 1971).

It appears that repetitions of forward pairings of CS and UCS (CS-UCS) result in a larger decrement in UCR when compared to CS and UCS presentations in other temporal relationships. However, comparing a group that receives CS-UCS pairings with another group that also receives CS and UCS but in a different temporal relationship does not allow for comparing the effects of conditioning and UCS-alone presentations (cf., Fitzgerald, 1966). There have only been a few studies in the literature that have made this latter comparison and the results have been inconsistent. Furedy (1970) and Lykken et al. (1972), both studying GSR, reported greater UCR decrements with CS-UCS pairings than with UCS-alone presentations.

Furedy and Klajner (1972) found a greater decrement in shock elicited GSR when preceded by CS, but no differences between CS-UCS pairings and UCS-alone for VPC (volume-pulse change response). Church et al. (1966) reported no differences between CS-UCS pairings and UCS-alone on the cardiac rate response to shock (UCS). Finally, Hupka, Kwaterski, and Moore (1970) and Grevert and Moore (1970) studying the nictitating membrane response in rabbits found UCR diminution to UCS-alone presentations, but not to the CS-UCS pair.

The conditions under which repetition of the CS-UCS pair result in a smaller or larger decrease in the magnitude of UCR than UCS-alone presentations remain unclear. The resolution of this question has important implications for an account of the phenomenon of UCR diminution. For example, a smaller decrease in UCR magnitude during conditioning than to UCS-alone presentations would indicate that UCR diminution was not a result of introducing CS, as many theorists suggest (Epstein, 1973; Kimmel, 1966; Lykken, 1962; Lykken and Tellegen, 1974; Rescorla, 1969). Such a finding might indicate that CS actually attenuates the habituation of UCR to UCS presentations incorporated into the conditioning paradigm. The possibility that UCR diminution is a function of UCS repetitions during conditioning has received little attention in the literature. Kimble and Ost (1961) attempted to ascertain the effects of UCS repetition on UCR diminution. When UCR magnitude on the last trial of conditioning was compared to UCS-alone presentations after conditioning UCR magnitude increased. However, when UCR magnitudes to UCS-alone presentations before and after

conditioning were compared they found a decrease in UCR magnitude. They concluded that part of the UCR diminution was atrributable to habituation to UCS during conditioning (cf., Dufort and Kimble, 1958; Fitzgerald, 1966; Furedy and Klajner, 1972). However, the finding of the latter comparison does not compel the conclusion that this is an effect of habituation to UCS. The post-conditioning diminution in UCR magnitude, in comparison to pre-conditioning UCR magnitude, may simply indicate that when CS is removed the recovery of the UCR to pre-conditioning levels is incomplete, i.e., there is a lasting effect of the repetition of the CS-UCS pair on UCR.

Data provided by Fitzgerald (1966) and Grevert and Moore (1970) demonstrate that the number of UCS repetitions during conditioning is directly related to the amount of UCR diminution. Fitzgerald (1966) found that when the number of CS presentations was held constant, reducing the number of UCS presentations resulted in a smaller UCR decrement. Grevert and Moore (1970) found that when UCS-alone presentations were interpolated between CS-UCS trials, the degree of UCR diminution was greater than to just CS-UCS presentations (cf., Hupka et al., 1970).

Aside from the studies on the number of UCS presentations, other parameters of UCS and their relationship to UCR diminution have received less attention. Furedy (1970) found no significant effects of UCS intensity on the amount of decrease in UCR magnitude during conditioning. Furedy and Klajner (1972) reported a significant interaction between CS-UCS pairings and UCS-alone presentations, at two different levels of UCS

intensity. However, they did not report the specific differences between the CS-UCS and UCS-alone groups, at each level of intensity. In conclusion, it would appear that before an adequate account of the phenomenon of UCR diminution can be developed a clearer understanding of the effects of a) UCS repetition during conditioning, and b) the parameters of UCS, and their relationship to the effects of CS intrusion, is necessary.

Another possible factor determining UCR diminution may be the effect of CR on the response to UCS. Several theorists have proposed such an explanation in terms of "response interference," suggesting that the CR "blends into" or "interferes with" the development of the UCR preventing its full blown occurrence (Badia and Defran, 1970; Grings and Schell, 1969a; Hilgard, 1933; Martin et al., 1975). One problem with this interpretation is that the process of interference is inferred from the behavior and is not measured independently of the data.

Wilder's (1957, 1962) conceptualization of the "law of initial values" (LIV) may provide an alternative to the response interference hypothesis as a means of understanding the relationship between CR and UCR magnitude. When the CS is in a fixed temporal relationship to UCS, the larger the magnitude of CR the higher the pre-UCS baseline will be (since the final portion of CR is, in essence, the pre-UCS level). If LIV occurs UCR magnitude will depend, in part, on CR magnitude. It would therefore be predicted that CR magnitude is inveresely related to UCR magnitude, over trials or sessions. Both Grings and Schell (1969a) and Martin et al. (1975) reported an inverse relationship between CR and UCR magnitude. However, these

authors concluded that this correlation supported the response interference hypothesis, since larger CRs would be expected to "interfere" more with UCR. Fitzgerald (1966) addressing the possibility of a relationship between LIV and UCR diminution also correlated CR and UCR magnitudes. While not significant the direction of the correlation was opposite to that predicted by LIV. Thus, the relationship between LIV and the phenomenon of UCR diminution needs further clarification.

LIV may present difficulties for between groups comparisons as well. For example, when comparing groups that receive either CS-UCS or UCS-alone presentations, if UCS-alone presentations have the effect of lowering pre-UCS levels (as was suggested earlier), while the CR in the CS-UCS group elevates the pre-UCS levels, the magnitude of the UCR in the UCS-alone group would appear to increase over trials, while the UCR in the CS-UCS group would appear to decrease over trials. An LIV effect of this type might account for the findings in some studies that conditioning results in a larger decrease in UCR magnitude than UCS-alone (Furedy, 1970; Furedy and Klajner, 1972; Lykken et al., 1972), although there is no evidence currently available that this is the case.

An important theoretical implication of the finding that UCR may decrease in magnitude with successive repetitions of the UCS before, as well as during, conditioning would be in terms of providing a basis for understanding the classical observation that the CR and UCR differ in such properties as magnitude, latency, and form (Schoenfeld, private communication). The fact that CR and UCR do differ has provided the foundation

for the assumption in behavior theory that they reflect two distinct response classes or states (Dykman, 1967; Gantt, 1968; Kimmel, 1973). However, differences between the CR and UCR can be accounted for without such an assumption (cf., Catania, 1970; Lockhart, 1966; Schoenfeld, 1972, 1976). One explanation may lie in the relationship between the rate of CR acquisition and the concomitant diminution of UCR. For example, if UCR diminution occurs at a slower rate than CR acquisition, a large number of conditioning trials might be necessary before the CR and UCR would converge toward equal magnitudes. Similarities between CR and UCR may have gone undetected in the literature when either relatively small numbers of conditioning trials were presented, or when dissimilarities between the parameters of CS and UCS were sufficient to produce greatly differing rates of CR acquisition and UCR diminution.

The present experiment was designed to answer some of the questions raised concerning the relationship of the effects of conditioning and UCS-alone presentations on the UCR. If the UCR shows an increase in magnitude over conditioning sessions after its habituation to prior UCS exposures, then this effect might be well considered part of the class of response events termed "dishabituation". However, should it be the case that UCR continues to decrease during conditioning, then the class of response events termed "habituation" may have to be expanded to include more than just the repetition of a single stimulus.

If the UCR magnitude decreases to both UCS-alone presentations and conditioning, then the possibility that the UCS presentations incorporated into the conditioning paradigm

are a determinant of the UCR diminution during conditioning should be considered. On the other hand, the introduction of conditioning may result in a decrement beyond that attributable to UCS repetitions. A comparison pertinent to this question would be between UCS-alone presentations and conditioning for the two groups receiving the same intensity UCS. Such a comparison would yield information as to the relative effects of UCS-alone presentations and conditioning on the UCR. Additionally, UCS presentations prior to conditioning provide an alternative means of manipulating the total number of UCS presentations, without varying the number of presentations during the period of conditioning. If shock repetition is an important determinant of UCR diminution, then manipulating the number of shocks prior to conditioning may have effects similar to varying the number of UCS presentations during conditioning.

UCS-alone presentations subsequent to conditioning also provide information concerning the effects of conditioning on UCR. If conditioning affects the UCR then removal of CS might result in a change in UCR magnitude. For example, given that repetitions of the CS-UCS pair result in a decrement in UCR, then UCR magnitude might increase upon removing CS.

The variables of UCS intensity and intertrial interval (ITI) length and their relationship to UCR diminution have received little attention in the literature. Therefore, the effect of these variables on changes in the UCR during conditioning, as well as the amount of UCR recovery to UCS-alone presentations after conditioning, was assessed.

LIV (the "law of initial values") presents difficulties

in ascertaining the effects of conditioning on the UCR. The statistical techniques used in the data analysis provided a means of determining the extent to which changes in UCR magnitude over conditioning sessions were dependent on shifts in pre-UCS (baseline) heart-rate levels due to CR acquisition.

The presentations of shock prior to conditioning provide an opportunity for the UCR to habituate before conditioning is instituted. Should it be true that the response that finally becomes "CR" is the same magnitude or temporal form as that which the original UCR devolves into at the end of its habituation, this finding would support the idea that differences typically noted between CR and UCR are partly due to differences in the extent of CR acquisition and UCR diminution. Bearing on this possibility would be observations as to whether the eventual CR differs from either the UCR at the end of UCS-alone presentations or conditioning.

Another issue addressed was the possibility that extinction to CS may generalize to the UCR, and drive the UCR to lower levels, thus elucidating yet another procedure that results in a decrease in UCR magnitude. The final experimental stage consisted of reconditioning in which the shock intensities for the high and low intensity groups were reversed. If CR and UCR magnitude are initially found to be related to UCS intensity during conditioning, then a shift in UCS intensity might reverse the effect on CR and UCR.

Method

Subjects

Twelve experimentally naive male rhesus monkeys (Macaca mulatta), acquired from Primate Imports, Inc., served as subjects. Their estimated age was between 2-4 years, and weights ranged between 8 and 12 pounds. They were regularly examined 2-3 months prior to the experiment to insure freedom from disease (including periodic tests for tuberculosis). Throughout the experiment they were fed three times daily (approx. 450 gm of Wayne Monkey Diet biscuits per day). One quarter apple was added as a diet supplement every other day.

Apparatus

For the entire duration of the experiment, the monkeys were restrained in BRS-Foringer Rhesus Test Chairs (No. PC-002, with thoracic plate). During the experimental sessions, the chairs were placed in BRS-Foringer Isolation Booths (No. PCH-002), lined with acoustic tile, and equipped with an exhaust fan, and 7.5 w blue overhead lamp, for ambient illumination. A Grason-Stadler Noise Generator (No. 901B), set at 10 db below 1.5 v, provided white masking noise to an overhead speaker (Quam, Inc.). The white masking noise and overhead lamp were on throughout each session.

Two white indicator lamps (Dialco, Inc., No. 327 bulbs) served as the visual simulus (CS) and were mounted approximately 23 cm in front of the animal, at eye level.

The second stimulus, electric shock (UCS), was delivered through brass electrodes (3.8 cm by 1.9 cm), attached to Velcro

strips and secured to a shaved portion of the monkey's tail. The tail was first cleansed with rubbing alcohol to remove surface dirt and oil, and was then secured in place by taping it to a cross-bar on the restraint chair. Electrode jelly (Cambridge, Inc.) was placed on the electrodes to improve contact with the tail. Constant current AC shock was delivered to the electrodes by a 650 v transformer whose output was first passed through a pair of matched capacitors. Shock intensity was manipulated by changing capacitance values. Switching transients were eliminated by a switching circuit designed by Ramsay, Knapp, and Zeiss (1970). Additional transients were eliminated by delaying shock onset and offset so that each occurred at zero volts in the AC voltage cycle. This was accomplished by means of a synchronous switch built from digital logic modules (BRS-Foringer, Inc.). Shock duration was set at 300 $\pm$ 4 msec.

Heart-rate was recorded through subcutaneous electrodes (Siemens, Subtrodes, No. 211140). Electrodes were generally implanted one over the sternum, and the other in the upper left portion of the abdomen. In cases where these sites did not produce noise free, large amplitude ECGs, other sites were selected to maximize the amplitude of the ECG signal. Implantations were conducted under a mild general anesthetic (Parke-Davis, Vetelar, ketamine hydrochloride). Soft leather vests were placed on the animals after electrode implantation, to minimize the possibility of their pulling the electrodes out (Ramsay, Pomerleau, and Snapper, 1968). The ECG signal was amplified by a Beckman Type-R Dynograph, whose output was

digitized by a Schmitt trigger circuit to provide an input suitable for computer processing. The amplified ECG signal, as well as the output of the digitizer circuit, was recorded on the writing unit of the polygraph, and the record provided a means to monitor the reliability of the ECG signal. An on-line PDP-8 computer (Digital Equipment) recorded the digitized ECG signal: successive interbeat times were measured to the nearest .01 sec, and were stored on paper tape for later analysis offline. The same computer also controlled the sequencing of the experimental stimuli employing a program specifically designed for behavioral experiments (Snapper and Kadden, 1973).

Procedure

All the animals were placed in restraint chairs 9-10 days prior to the beginning of the experiment. Approximately 7-8 days prior to the experiment the subjects were placed in the isolation booths one hour a day, for adaptation to the chambers. Three or four days prior to the experiment tails were shaved, and the shock electrodes were attached for the period the animals were in the chamber for adaptation. Implantations of the subcutaneous electrodes were conducted at this time. Following implantations the animals were not placed in the experiment for approximately 24 hrs to allow the Vetelar to pass from the animals' system. This procedure was followed whenever an implantation became necessary during the course of the experiment, as when the ECG signal became noisy.

The experiment was divided into nine successive "stages". Within any given stage all parameter values were kept constant

over sessions. For Stages 1-6 the mean inter-trial interval was 5.00 min and each session consisted of 16 trials, across all groups and stages.

For the 0.5, 4.0, and 13.5 mA groups, Stage 1 consisted of 10 sessions of UCS-alone presentations. The numbers that designate the different groups indicate the intensity of UCS each group received. The NP-4.0 mA group did not receive UCS in Stage 1, but the animals were strapped with electrodes and placed in the isolation chambers (with over-head lamp and white noise on), and they remained there for the duration of the experimental session. In subsequent stages, when there were shock presentations, the NP-4.0 mA group also received 4.0 mA shocks. The NP designation denotes that this group received no UCS presentations during the first stage.

During Stage 2, which also consisted of 10 sessions, the CS was introduced 10 sec before UCS onset and was terminated at UCS onset.

Stage 3 consisted of one session of UCS-alone presentations.

Stage 4 consisted of 10 sessions of CS-alone presentations.

Stage 5 was identical to Stage 1: 10 sessions of UCS alone presentations.

Stage 6 involved 5 sessions of CS-UCS pairings,with the shock intensities reversed for the 0.5 and 13.5 mA groups. The shock intensity for the 4.0 and NP-4.0 mA groups remained the same.

For the remaining stages (7-9), only the 4.0 and NP-4.0 mA groups were run, with two parameter changes: the mean ITI was reduced to 1.5 min, and the number of trials per session was

increased to 50. Shock intensity remained at 4.0 mA. Stage 7 consisted of 2 sessions of UCS-alone presentations, Stage 8 consisted of one session of CS-UCS pairings, and Stage 9 was identical to Stage 7.

Each group, as defined by the value of the intensity of shock, consisted of three subjects. Due to equipment limitations, the experiment was conducted in two passes: the 13.5 and 0.5 mA groups were run first, followed by the 4.0 and NP-4.0 mA groups.

Data were recorded as time between successive cardiac R-waves, i.e., the inter-beat interval (IBI), with a resolution of 10 msec. Data were recorded 12 sec before shock (2 sec before light onset, and 10 sec during light), and 30 sec following shock (except 0.3 sec during shock and 0.7 after, as the noise introduced into the signal by the shock circuit made the signal unanalyzable).

Results

Data Analysis. The following sequence of successive interbeat intervals (IBI) was recorded on each trial: 2-sec prior to CS onset, 10-sec during CS, and 30-sec post-UCS. For purposes of data treatment the mean interbeat interval (IBI) of successive two-second periods (or "bins") was determined. During conditions when CS was not present only the mean IBI for the 2-sec bin preceding shock and the fifteen 2-sec bins post-UCS were determined. Data are reported as IBIs to avoid the complications of using the non-linear transformation of IBI to rate (cf., Khachaturian, Kerr, Kruger, and Schachter, 1972).

Previous studies from this laboratory (e.g., Ramsay, 1970; Snapper, Pomerleau, and Schoenfeld, 1969) and inspection of the present data demonstrated that the characteristic form of the conditioned (CR) and unconditioned (UCR) cardiac rate response in Macaca mulatta to a wide variety of stimuli is biphasic. The first phase is an acceleration in heart-rate; the second is a deceleration in heart-rate. For purposes of analysis both phases were treated separately.

For the analysis of response magnitude three measures were employed: a) pre-stimulus (baseline) level, which was the mean IBI for the bin immediately preceding stimulus onset; b) the maximum level of the acceleratory phase of the response, which was the smallest mean IBI after stimulus onset; and c) minimum level of the subsequent deceleratory phase, which was the largest mean IBI following the peak of the acceleratory phase.

Hereafter, these measure will be called pre-stimulus level, magnitude of the acceleratory phase, and magnitude of the deceleratory phase respectively. The mean of each measure was determined for each session. Response magnitude refers to absolute post-stimulus levels and not to the difference between pre- and post-stimulus levels. Response latency was measured as the ordinal number of the bin in which the minimum mean IBI post-stimulus occurred. Finally, the temporal form of the response was determined by plotting the mean IBI per bin for an entire session (i.e., the overall session average of the mean IBI of each bin) as a function of successive bins.

As noted earlier, the "law of initial values" (LIV) is a major problem in analyzing response magnitude data from autonomic nervous system responses. In measuring the magnitude of the response to stimulation two measures of a given function are available: pre-stimulus level (X) and the level reached after the stimulus is applied (Y). The problem of LIV arises when the level of the response reached after the stimulus is applied is systematically related to the pre-stimulus levels, i.e., if there is a high correlation between pre- and post-stimulus levels (cf., Wilder, 1957, 1962). There is some question in the literature as to how LIV should be quantified. Either the correlation between pre-stimulus levels and response magnitude as measured by the difference between pre- and post-stimulus levels, or the absolute post-stimulus levels, has been most often used to express LIV (cf., Lacey, 1956; Oken and Heath, 1963).

In the present experiment the correlation between the

pre-stimulus level (X) and the absolute post-stimulus response level (Y) was used to assess the extent of the LIV relationship in each session. Pearson product-moment correlation coefficients (r_{xy}) were calculated between the pre-stimulus level and the magnitude of the acceleratory phase (smallest mean IBI post-stimulus), as well as between the pre-stimulus level and the magnitude of the deceleratory phase (largest mean IBI subsequent to the peak of the acceleration), for each session. The results for the acceleratory and deceleratory phases for both the UCR and CR are shown in Tables 1A and 1B respectively. The data in Table 1 show that in the majority of sessions there was a significant positive correlation between the mean pre-stimulus IBI level and a) the magnitude of the acceleratory phase and b) the magnitude of the deceleratory phase of both the CR and UCR. These data demonstrate a strong LIV relationship in a majority of sessions.

Given the occurrence of LIV various statistical techniques have been proposed to determine whether changes in response magnitude over trials, sessions, or groups are due to the effects of variation in pre-stimulus level, or to the effects of the experimental manipulation (Benjamin, 1963, 1967; Johnson and Fay, 1950; Lacey, 1956; Myers and Honig, 1969; Oken and Heath, 1963; Pigache, Graham and Freedman, 1976). Benjamin (1963, 1967) has reviewed a number of these techniques and concluded that the analysis of covariance (ANCOVAR) model was the best available technique. Furthermore, using the ANCOVAR model produces identical results when either absolute or difference scores are used as the measure of response magnitude (cf., Benjamin, 1963; Boismier, 1974). The regression analysis employed in the present

Table 1A

Correlations (r_{XY}) Between Pre-Stimulus Levels (X) and Post-Stimulus Levels (Y)

Response to Shock: Acceleratory Phase

Session	Subjects D-50	D-90	C-36	E-22	E-16	E-08	D-20	D-94	D-92	E-20	E-10	E-04
I-1	.81*	.52*	.02	.77*	.87*	.36*	.42*	.61*	.71*			
I-10	.51*	.24	.74*	.15	.02	.84*	.87*	.88*	.27			
II-1	.07	.47*	.82*	.12	.75*	.63*	.28	.89*	.69*	.83*	.97*	.12
II-10	.41*	.63*	.86*	.37*	.37*	.13	.31	.85*	.70*	.94*	.98*	.40*
III	.61*	.05	.80*	.10	.35*	.78*	.78*	.80*	.71*	.45*	.93*	.95*
V-1	-.11	.89*	.86*	.89*	.39*	.93*	.53*	.53*	-.50*	.50*	.99*	.36*
V-10	.45*	-.05	.75*	.70*	.88*	.64*	.76*	.88*	.65*	.45*	.96*	.23
VI-5	.40*	.48*		-.06	.73*	.97*	.33	.52*	.70*	.80*	.98*	.88*
VII-1					.58*	.95*	.84*	.93*		.93*	.92*	.20
VII-2					.71*	.98*	.97*	.69*		.69*	.93*	.94*
VIII					.54*	.91*	.70*	.79*		.79*	.16*	.07
IX							.95*	.60*		.60*	.96*	.53*

* $p < .05$ Note: Blank spaces indicate conditions not used in the data analysis

Table 1A

Correlations (r_{XY}) Between Pre-Stimulus Levels (X) and Post Stimulus Levels (Y)

Response to Shock: Deceleratory Phase

Stage & Session	D-50	D-90	C-36	E-22	E-16	E-08	D-20	D-94	D-92	E-20	E-10	E-04
I-1	.41*	.05	.01	.37*	.67*	.77*	.07	.37*	.58*			
I-10	.78*	.56*	.61*	.36*	.04	.35*	.74*	.56*	-.28			
II-1				.27	.79*	.69*				.94*	.90*	.07
II-10	.48*	.51*	.81*	.61*	.81*	.61*	.59*	.82*	.43*	.95*	.95*	.52*
III	.62*	.23	.31	.01	.39*	.60*	.73*	.75*	.46*	.20	.90*	.80*
V-1	.74*	.74*	.90*	.77*	.41*	.70*	.62*	.30	-.51*	.21	.96*	.35*
V-10	.55*	-.04	.82*	.01	.89*	.93*	.59*	.88*	.55*	.39*	.93*	.20
VI-5	.43*	.35*	.78*	.27	.70*	.39*	.25	-.13	.07	.84*	.97*	.73*
VII-1				.50*	.91*	.97*				.69*	.84*	.13
VII-2				.40*	.87*					.46*	.94*	.80*
VIII				.51*	.99*					.66*	.10	-.11
IX				.45*	.85*					.63*	.96*	.43

* $p < .05$ Note: Blank spaces indicate conditions not in the data analysis

Table 1B

Correlations (r_{XY}) Between Pre-Stimulus Levels (X) and Post-Stimulus Levels (Y)

Responses to Light: Acceleratory Phase

Stage & Session	D-50	D-90	C-36	E-22	E-16	E-08	D-20	D-94	D-92	E-20	E-10	E-04
II-1				.81*	.63*	.83*				.98*	.96*	.85*
II-10	.76*	.28*	.96*	.86*	.86*	.87*	.85*	.98*	.99*	.94*	.99*	.64*
IV-10	.79*	.68*	.44*	.94*	.92*	.90*	.83*	.46*	.98*	.91*	.85*	.57*
VI-5	.94*	.93*	.62*	.77*	.82*	.98*	.71*	.36*	.36*	.99*	1.00	.98*

* p .05 Note: Blank spaces indicate conditions not used in the data analysis

Response to Light: Deceleratory Phase

Stage & Session	D-50	D-90	C-36	E-22	E-16	E-08	D-20	D-94	D-92	E-20	E-10	E-04
II-1				.55*	.56*	.57*				.90*	.95*	.67*
II-10	.35*	-,20	.90*	.52*	.80*	.74*	.69*	.90*	.96*	.90*	.99*	.38*
IV-10	.29	.35*	.38*	.89*	.80*	.79*	.62*	.41*	.87*	.77*	.52*	.28
VI-5	.62*	.62*	.62*	.36*	.75*	.98*	.66*	-.06	-.58*	.90*	.98*	.96*

* p <.05 Note: Blank spaces indicate conditions not used in the data analysis

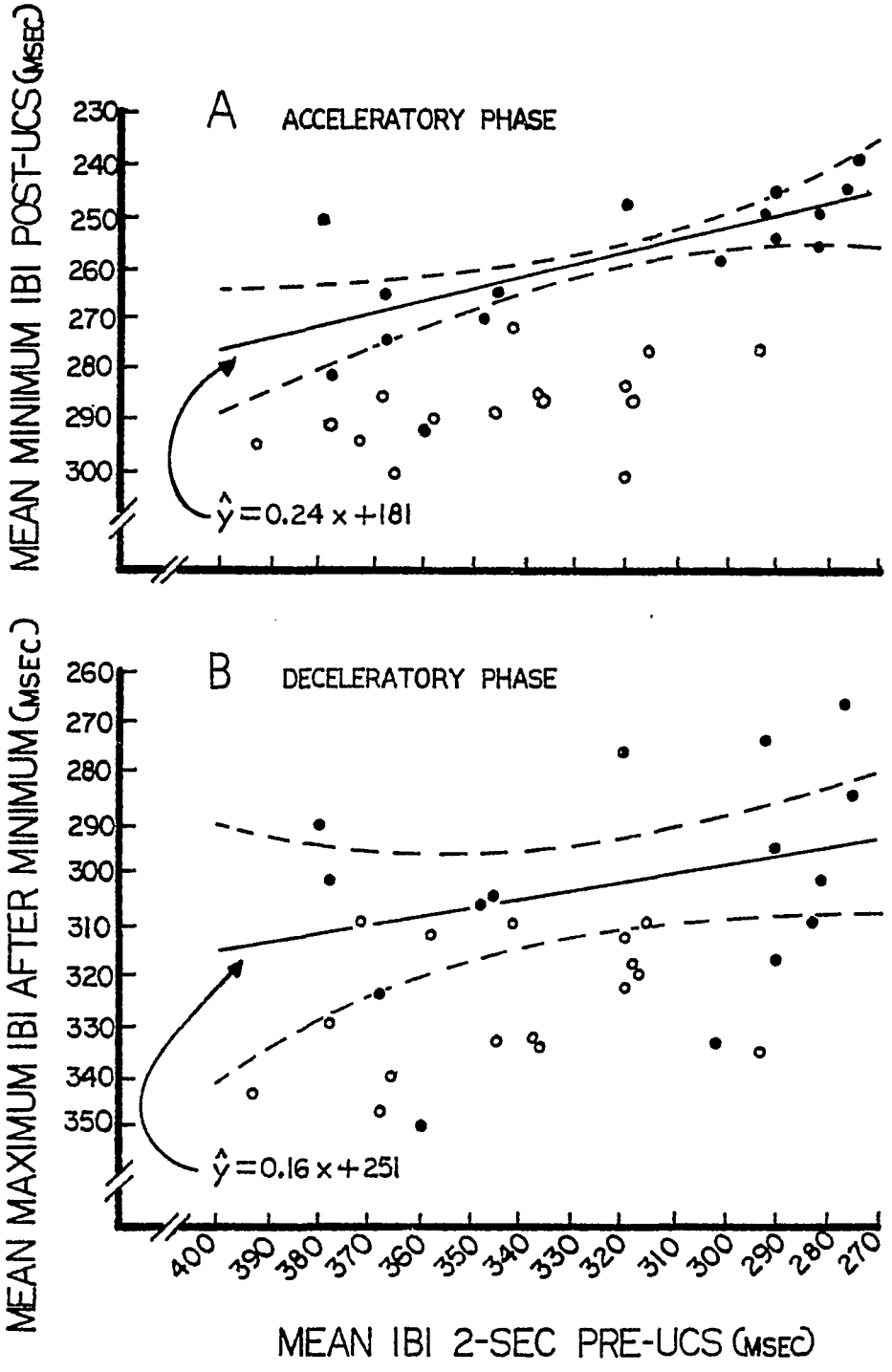
experiment to determine changes in the response magnitude over sessions for individual subjects is based on the covariance model (Snedecor and Cochran, 1967) and is similar to the technique described by Pigache et al. (1976).

The regression analysis applied to autonomic nervous system functions was proposed by Myers and Honig (1969) as a means of separating the effects of the experimental manipulation on response magnitude from the indirect effects on response magnitude resulting from the action of the experimental manipulation on pre-stimulus level. This analysis achieves two purposes. First of all, it allows for the prediction of the effects of systematic changes in pre-stimulus level over sessions on response magnitude. Second, it gives the probability that the response magnitude after a particular manipulation was different from the response magnitude before the manipulation.

The data from subject E-22 for sessions 1 and 10 of Stage 1 (shock-alone presentations) illustrate the implementation of the regression technique. Figure 1A shows the scatter-diagram of mean minimum IBI during UCS as a function of mean IBI pre-UCS, and Figure 1B shows the mean maximum IBI after the peak of the acceleratory phase as a function of mean IBI pre-UCS. Each data point represents the UCR magnitude on a particular trial (ordinate value) and the pre-UCS level for that trial (abscissa value). There are 15 data points for session 1 and 16 points for the data in session 10. A linear function describing the regression of the post-stimulus values (Y) on the pre-stimulus values (X) was determined for the data in session 1 and the functions for both the acceleratory and deceleratory phases of

Figure 1. Regression functions between the phases of the UCR (response to shock) and pre-shock levels (mean IBI 2-sec pre-shock), for the acceleratory phase of the response (mean minimum IBI post-shock; panel A), and for deceleratory phase of the response (mean maximum IBI after minimum; panel B), in subject E-22. Closed circles represent the pre-shock (UCS) level, and the corresponding post-shock level, for trials in session 1; open circles represent the pre-shock level, and corresponding post-shock level, for trials in session 10. The straight line in each panel represents the least-squares best fit for the data in session 1, and the curved lines around this line are the 95% confidence intervals for the regression line.

Fig. 1


A
ACCELERATORY PHASE
MEAN MINIMUM IBI POST-UCS (MSEC)
230
240
250
260
270
280
290
300
ŷ = 0.24 x + 181
B
DECELERATORY PHASE
MEAN MAXIMUM IBI AFTER MINIMUM (MSEC)
260
270
280
290
300
310
320
330
340
350
ŷ = 0.16 x + 251
400
390
380
370
360
350
340
330
320
310
300
290
280
270
MEAN IBI 2-SEC PRE-UCS (MSEC)

the response are drawn in Figures 1A and 1B respectively. The form of this function is given by:

$$Y' = b_{y.x}X + a$$

where $b_{y.x}$ is the slope of the function (coefficient of regression) and a is the Y-intercept. From this regression function predictions may be made of the UCR magnitude (Y') that would be expected if the only consequence of the experimental intervention was its effect on pre-stimulus level (X).

Myers and Honig (1969) describe a method for making this type of prediction and for testing whether the empirically determined effects of the experimental manipulation differ significantly from the prediction. Once the regression function is derived for a given session, a confidence interval for the function can be established based on the standard error terms for estimating the Y-intercept and the slope of the population regression function from the empirically determined regression function, and the desired level of confidence. The upper and lower limits of the confidence interval for the regression functions of subject E-22, within which the population regression equation lies with 95% certainty ($t = 2.16$, $df = 13$), is shown for the data of session 1 in Figures 1A (acceleratory phase) and 1B (deceleratory phase).

Given the limits of such a confidence interval it is possible to state, with 95% certainty, whether a set of data was drawn from the same population as the original sample. In this manner the effect of the experimental manipulation on response magnitude can be distinguished from the indirect effect

on response magnitude resulting from the effect of the manipulation on pre-stimulus (baseline) levels. In the present experiment the interest was in comparing the mean and variability of a given session after a particular manipulation to the confidence interval of the regression line of a session prior to the manipulation. In the example from the data of E-22, given the mean pre-stimulus level of session 10 the question was asked whether the mean empirically determined UCR magnitude of session 10 fell within the confidence limits of the regression line of session 1. If the mean magnitude of the response of session 10 fell within the confidence limits then changes in the UCR magnitude could be attributed simply to the difference in pre-UCS level between sessions1 and 10; if the mean UCR magnitude fell outside the confidence limits then any changes in UCR magnitude by session 10 could be attributed to the effects of the experimental manipulation, with 95% confidence. Mathematically, this was accomplished by using the regression equation of session 1 to predict a UCR magnitude value (Y_1') at the mean pre-stimulus level of session 10 ($\bar{X}_{10}$). That is to say, the mean pre-UCS level of session 10 was used as the predictor variable in the regression equation of session 1. The difference between the empirically obtained mean UCR magnitude in session 10 and the predicted response magnitude ($Y_1' - \bar{Y}_{10}$) is divided by the pooled error estimate, which is comprised of the standard error terms for the post-stimulus response levels in sessions 1 and 10, and the variability of the slope of the regression equation of session 1. The equation for the statistical test is:

$$t = \frac{Y'_1 - \bar{Y}_{10}}{\sqrt{\frac{s^2_{Y_1 \cdot X_1}}{N_1} + \frac{s^2_{Y_{10} \cdot X_{10}}}{N_{10}} + \frac{(s^2_{Y_1 \cdot X_1}) \times (\bar{X}_1 - \bar{X}_{10})^2}{\Sigma(X_1 - \bar{X}_1)^2}}}$$

and is tested against the Student's t-distribution with df = $N_1 + N_{10} - 3$. Snedecor and Cochran (1967, ch. 6) provide the derivation for the general form of this statistic. This t-test indicates, at a given level of confidence, whether the responses of a particular subject after some experimental manipulation were sampled from the same population as the responses in the session prior to the manipulation. By equating the pre-stimulus levels of the sessions at the beginning and end of the experimental manipulation, the regression technique allows for a comparison of the response magnitudes of these two sessions independent of any effect of pre-stimulus level on the response. It can be thus determined whether the experimental intervention significantly modified the response above any effect the manipulation had on pre-stimulus (baseline) levels.

Application of this t-statistic to the data in Figures 1A and 1B shows that the reduction in the magnitude of the acceleratory phase of the UCR (25 msec decrease), and the increase in the magnitude of the deceleratory phase (21 msec increase), by session 10 was significantly different ($p < .05$) from the UCR magnitude predicted from the mean pre-UCS level of session 10. Thus, it may be concluded for subject E-22 that the effects of shock-alone presentations over sessions reduced the magnitude of the acceleratory phase of the response to shock and increased the magnitude of the deceleratory phase,

beyond changes in UCR magnitude attributable to the effects of shock repetition on pre-UCS levels.

The comparison of the predicted and obtained response magnitudes outlined for subject E-22 was conducted in a similar fashion for all subjects. For the results of any comparison of two sessions the convention has been adopted to report the first session of the experimental manipulation from which the predicted response magnitude (Y') was obtained, and the last session of the manipulation from which the empirically obtained score ($\overline{Y}$) was determined. The first session provides a "baseline" to assess the effects of the experimental intervention after a specified number sessions. In all cases the predicted response magnitude was determined at the pre-stimulus level of the last session of the experimental intervention. In the tables reporting the results of the t-statistic for the acceleratory phase of the response only the differences between the predicted and obtained scores ($Y' - \overline{Y}$) are given ($p < .05$); for the deceleratory phase the differences between the obtained and predicted scores ($\overline{Y} - Y'$) are given ($p < .05$). This difference in reporting the results for changes in the magnitude of the acceleratory or deceleratory phase of the response was adopted so that negative difference scores would always indicate a decrease in magnitude over sessions and positive difference scores would indicate an increase in response magnitude. This difference score indicates the amount of change in response magnitude attributable to the experimental manipulation for a given number of sessions, independent of indirect effects of pre-stimulus level on magnitude.

The statistic described above was only employed in comparing response magnitudes over sessions in individual animals. Since this statistic could not be applied for the analysis of response magnitudes among groups a standard one-way analysis of covariance was used to make among groups comparisons (Winer, 1962, ch. 11). For the acceleratory phase of the response the post-stimulus level measure for the covariance analysis was the average of the session means of the magnitude of the acceleratory phase of the response (mean minimum IBI post-stimulus), for the three animals in each group; similarly, for the deceleratory phase of the response the post-stimulus measure was the average of the session means of the magnitude of the deceleratory phase of the response (mean minimum IBI post-stimulus) for the three animals in each group. For both phases of the response the covariate measure was the average of the session means for 2-sec pre-stimulus for the three animals in each group. Comparisons among means were accomplished by a standard multiple comparisons among means test (Winer, 1962, ch. 11).

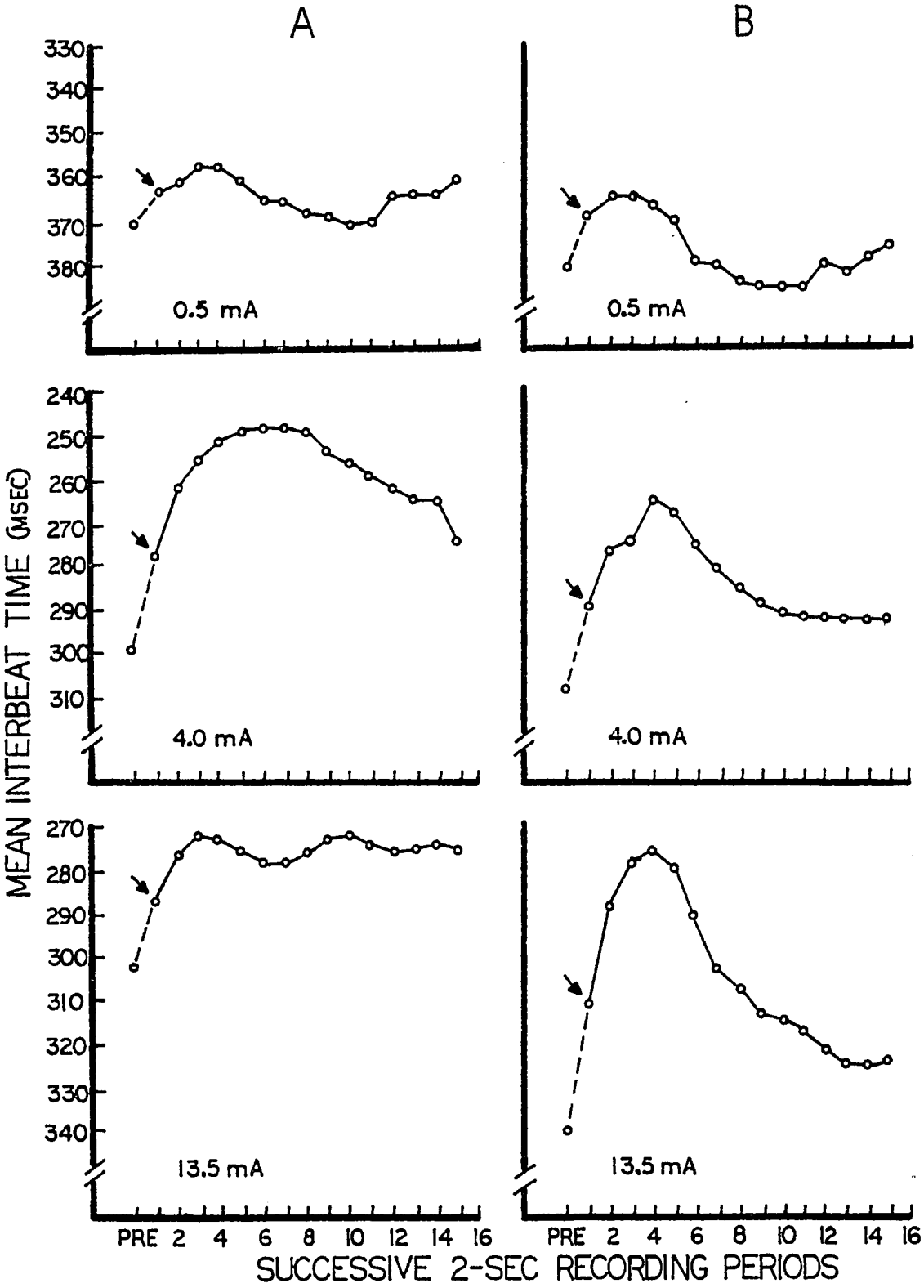
Stage 1

Stage 1 consisted of 10 sessions of shock (UCS)-alone presentations. The analysis within this stage centered on whether the response form, magnitude, or latency changed with repeated presentations of shock, within and across sessions.

Form of the Response. Figure 2 presents the group curves of the mean IBI per bin of sessions 1 and 10 of Stage 1, for the 0.5, 4.0, and 13.5 mA groups for the UCR. Each data point represents the mean of three animals. The form of the response

Figure 2. Mean IBI in successive 2-sec bins for the 13.5 mA, 4.0 mA, and 0.5 groups, in session 1 (panel A) and session 10 (panel B) of Stage 1, for the response to shock (UCS). Each data point represents the session mean IBI per bin, averaged over the three animals in each group. The first data point in each curve represents the mean IBI 2-sec pre-UCS. Arrows indicate UCS onset.

Fig. 2


A
B
330
340
350
360
370
380
0.5 mA
0.5 mA
240
250
260
270
280
290
300
310
4.0 mA
4.0 mA
270
280
290
300
310
320
330
340
13.5 mA
13.5 mA
MEAN INTERBEAT TIME (MSEC)
PRE 2 4 6 8 10 12 14 16
PRE 2 4 6 8 10 12 14 16
SUCCESSIVE 2-SEC RECORDING PERIODS

in session 1 was biphasic: the first phase of the response consisted of a large magnitude, rapid acceleration in heart-rate which reached a peak after 4-6 sec; the second phase was a small magnitude, slow deceleration.

Generally, the deceleratory phase went below pre-UCS levels. While in Figure 2 it appears that the deceleration did not return to pre-UCS levels for the 4.0 and 13.5 mA groups, this was a consequence of the 30-sec post-UCS recording period. To assess the full extent heart-rate returned to basal levels after each shock the pre-UCS level of each subsequent stimulus was included as an index of the final level of the deceleratory phase of the preceding stimulus on trials when heart-rate did not return to pre-UCS levels within the 30-sec post-UCS recording period. The last trial was excluded from this analysis since data after the 30-sec post-UCS were not available. The deceleratory phase went below pre-UCS levels on 68%, 51%, and 64% of the trials for the 0.5, 4.0, and 13.5 mA groups respectively. Additionally, classifying trials according to whether heart-rate remained above or went below pre-UCS levels revealed a relationship between pre-UCS levels and the extent heart-rate returned to basal levels. Table 2 shows the results of the *t*-test (Student's *t*) comparing the mean pre-UCS levels for trials in which the deceleration remained above pre-UCS levels with trials in which it went below pre-UCS levels for each group. When the mean pre-shock level was high the deceleratory phase went below pre-UCS levels; when the pre-shock level was low the deceleratory phase remained above pre-UCS levels.

Table 2

t-Tests for the Difference Between Mean Pre-Shock Levels When the the Deceleratory Phase Went Below, Or Stayed Above, Pre-Shock Levels

Intensity (mA)	13.5	4.0	0.5
Deceleratory Phase Above Pre-Shock Levels: Mean IBI Pre-Shock (msec)	333	321	397
Deceleratory Phase Below Pre-Shock Levels: Mean IBI Pre-Shock (msec)	287	288	364
Difference: Above-Below (msec)	46*	33*	33

* $p < .05$

Figure 2 also shows that in session 10 all groups displayed a biphasic response to shock (acceleration followed by deceleration). The most prominent difference in the form of the reponse in comparison to session 1 was a larger magnitude deceleratory phase. The group curves in Figure 2 are representative of the individual animals, and there were no systematic changes in the form of the response over the trials of session 10. The proportion of trials in which the phase of deceleration went below pre-shock levels was also approximately the same as in session 1.

Magnitude of the Response. Figure 2 illustrates the difficulty in assessing the changes in response magnitude across sessions without accounting for changes in pre-UCS level. In comparing the left panel (session 1) with the right panel (session 10) it can readily be seen that while the response magnitude changed across sessions the pre-UCS level changed as well. This is most apparent in the 13.5 mA group where UCR magnitude increased while pre-UCS level decreased. For the other two groups the shifts in baseline over sessions were not as large. Figure 2 shows that by session 10 for the 4.0 mA group the magnitude of the acceleration decreased while the deceleration remained unchanged; for the 0.5 mA group both phases of the response increased in magnitude. To assess changes in response magnitude over sessions independent of the shifts in pre-UCS level the *t*-test for individual comparisons was employed.

Between session changes in the magnitude of the response were based on a comparison of sessions 1 and 10. Table 3 presents

Table 3

t-Tests For The Difference Between the Predicted Magnitudes of Stage 1, Session 1 ($Y'_{1,1}$) and the Obtained Magnitudes of Stage 1 Session 10 ($\bar{Y}_{1,10}$); and t-Tests for the Difference Between the Mean Pre-UCS of Stage 1, Sessions 1 & 10 ($\bar{X}_1 - \bar{X}_{10}$)

Response to Shock (UCR): Acceleratory Phase.

Intensity (mA)	Subject	$\bar{X}_1 - \bar{X}_{10}$ (msec)**	$Y'_{1,1} - \bar{Y}_{1,10}$ (msec)
	D-50	-67*	- 8
13.5	D-90	+23	+29*
	C-36	-73*	-40*
	E-22	-23*	-25*
4.0	E-16	- 6	-30*
	E-8	+15	-10*
	D-20	-38*	- 9
0.5	D-94	-12	- 4
	D-92	+11	+18

Response to Shock (UCR): Deceleratory Phase

Intensity (mA)	Subject	$\bar{Y}_{1,10} - Y'_{1,1}$ (msec)
	D-50	+57*
13.5	D-90	-19
	C-36	+109*
	E-22	+21*
4.0	E-16	+85*
	E-8	+ 4
	D-20	+43*
0.5	D-94	+ 6
	D-92	+ 3

* $p < .05$

** Note: Reported values only for acceleratory phase since pre-UCS levels were identical for deceleratory phase.

the results of the t-tests comparing the difference between the predicted response magnitudes of session 1 and the obtained response magnitudes of session 10, for both the acceleratory and deceleratory phase of the response to shock. Also shown in Table 3 are the results of the t-tests (Student's t) for the difference between the pre-shock levels of sessions 1 and 10.

Seven of the nine animals showed a statistically significant change in the magnitude of either one or both phases of the UCR by session 10. The two animals that showed no significant change in the magnitude of either phase of the response (D-92 and D-94) were both in the 0.5 mA group. Seven animals showed a decrease in the magnitude of the acceleratory phase of the response, and an increase in the magnitude of the deceleratory phase. Six of these animals had a lower pre-UCS level in session 10 than in session 1. The extent to which the magnitude of the deceleratory phase increased over sessions was related to the degree to which the magnitude of the acceleratory phase decreased: the larger the decrease in the magnitude of the acceleratory phase the smaller the increase in the deceleratory phase. This relationship was found to be significant as shown by a Pearson product-moment correlation ($r = -.75$, $df = 12$, $p < .05$). Two animals showed an increase in the magnitude of the acceleratory phase of the UCR; one of these animals showed a decrease in the magnitude of the deceleratory phase of the response, while the other showed a slight increase in magnitude. The pre-shock level increased for both animals by session 10.

Comparing the magnitude of the UCR among groups presents

the same difficulty as with individual animals. As seen in Figure 2 the pre-UCS levels vary across groups and make direct comparisons of UCR magnitude difficult. Therefore, a one-way analysis of covariance for among groups comparisons was conducted. For the acceleratory phase of the response the F-ratios for both session 1 ($F=1.81$, $df = 2,5$) and session 10 ($F = 2.22$, $df = 2,5$) were non-significant. Similarly, for the deceleratory phase of the response, the F-ratios for both session 1 ($F = .67$, $df = 2,5$) and session 10 ($F = .12$, $df = 2,5$) were non-significant. While these F-ratios indicate that when pre-shock levels are adjusted to the same level there are no statistically significant differences among groups, in the magnitude of either phase of the response, Figure 2 shows the following relationship in session 10: the higher the intensity of the shock the larger the magnitude of the acceleratory phase of the response.

Statistical analysis of within session changes in the magnitude of the response was not possible due to the small number of trials for each subject. However, inspection of trial-by-trial data did not reveal any systematic change in either phase of the response to shock.

These results may be summarized as follows: The main effect of repeated shock presentation over sessions was to reduce the magnitude of the acceleratory phase of the response and to increase the magnitude of the deceleratory phase. There was also a concurrent reduction in pre-shock levels over sessions. Furthermore, the larger the decrease in the magnitude of the acceleratory phase of the response over sessions, the

smaller the increase in the deceleratory phase. Finally, there was a positive relationship between response magnitude and shock intensity in session 10.

Response Latency. The mean response latency, defined as the ordinal number of the bin in which the peak of the acceleration occurred, was determined for all groups (N=3) for both sessions 1 and 10. For session 1 the mean response latencies were as follows: 13.5 mA: bin 8; 4.0 mA: bin 7; 0.5 mA: bin 7. For session 10 the mean response latencies were as follows: 13.5 mA: bin 4; 4.0 mA: bin 6; 0.5 mA: bin 7. There were no significant differences among groups in either session for mean response latency. For all three groups mean response latency decreased over sessions, but this decrease was not statistically significant. However, there was a significant inverse relationship between the latency of the acceleratory phase and the magnitude of the deceleratory phase (measured as the difference between the minimum mean IBI of the acceleratory phase and the maximum mean IBI after the minimum) in session 10. The mean latency and mean magnitude of the deceleratory phase were derived for each animal, and a product-moment correlation coefficient was determined for these values. The correlation was negative and significant ($r = -.95$, $df = 16$, $p < .01$) and indicates that the shorter the latency of the acceleratory phase the larger the magnitude of the deceleratory phase.

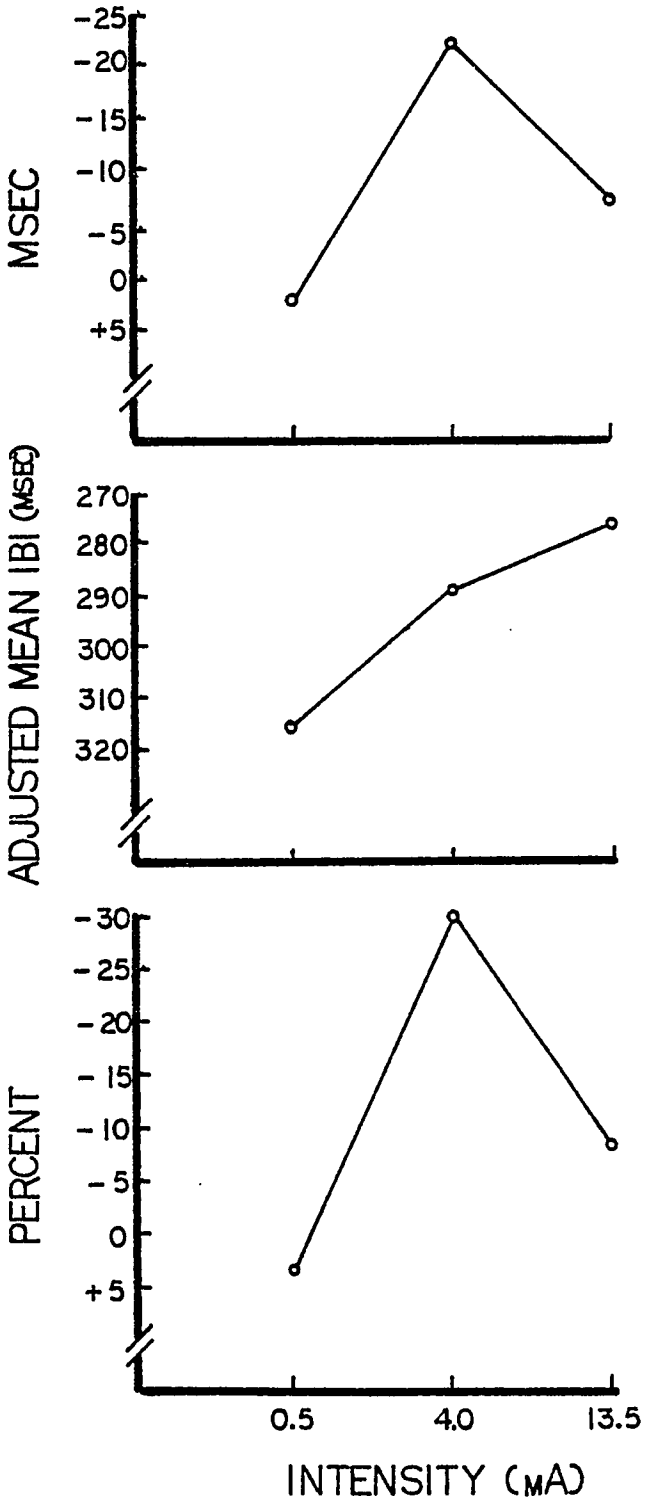
Shock Intensity-Habituation Function. To assess the habituation-intensity function three different measures of habituation were employed. For this analysis only the

acceleratory phase of the response was considered since only this phase showed a decrease in magnitude over sessions. The first measure was the absolute amount of change and is identical to the difference between the predicted and obtained scores ($Y_1'-\bar{Y}_{10}$) shown in Table 3. The group mean(N=3) for the absolute amount of change over sessions was determined. The second measure was the final response level in session 10 and consisted of the adjusted mean response magnitudes derived from the multiple comparisons among means test for session 10 (Note: While the F-ratio was non-significant, the adjusted magnitudes provided an index of final UCR magnitude adjusted for differences in pre-UCS levels among the groups). The third measure was the percentage change in response magnitude over sessions. This measure was assessed as the percent of change over sessions from the initial UCR magnitude in Session 1. For each subject the mean response magnitude in session 1 was determined as the difference between mean pre-stimulus level and the minimum mean IBI post-UCS ($\bar{X}_1-\bar{Y}_1$) and group means (N=3) were calculated. The percentage change across sessions was the ratio of the absolute amount of change over sessions to the magnitude of the response in session 1 ($Y_1'-\bar{Y}_{10}/\bar{X}_1 - \bar{Y}_1$).

Figure 3 presents the group curves for each of the measures of habituation as a function of shock intensity. Figures 3a and 3c show the relationship between the absolute amount of change and percentage change as a function of shock intensity respectively. Both these measures show essentially the same function: the low shock intensity group shows no change in the magnitude of the UCR, while the moderate shock

Figure 3. The relationship between shock intensity and habituation of the acceleratory phase of the response to shock, over sessions of Stage 1, as measured by: 1) absolute amount of change (panel A; ordinate values are the difference between the predicted magnitudes of session 1 and the obtained magnitudes of session 10 ($Y'_1 - \bar{Y}_{10}$), 2) final response levels (panel B; ordinate values are adjusted mean post-shock (UCS) IBI levels in session 10, 3) percentage change (panel C; ordinate values are percentages). Each data point represents the session means in each group.

Fig. 3


-25
-20
-15
-10
-5
0
+5
MSEC
A ABSOLUTE AMOUNT OF CHANGE $(\hat{y}_1 - \bar{y}_{10})$
270
280
290
300
310
320
ADJUSTED MEAN IBI (MSEC)
B FINAL RESPONSE LEVEL
-30
-25
-20
-15
-10
-5
0
+5
PERCENT
C PERCENTAGE CHANGE
0.5
4.0
13.5
INTENSITY (MA)

intensity group shows the greatest decrease in UCR magnitude. Figure 3b presents the curve for the adjusted mean response magnitudes in session 10 as a function of shock intensity and shows the magnitude of the response is positively related to intensity.

Pre-UCS level. To asses specifically the direction in which LIV affected the magnitude of the UCR two measures of change in UCR magnitude unadjusted for shifts in pre-UCS level were compared to changes in UCR magnitudes after adjustment for shifts in pre-shock level. The first measure was based on the difference between pre- and post-shock levels, a measure typically employed in the literature. For each subject the difference between mean IBI pre-shock ($\bar{X}$) and minimum IBI post-shock ($\bar{Y}$) was determined for sessions 1 and 10. This measure of response magnitude will be denoted as the difference score ($\bar{D}$). To determine the amount of change over sessions the magnitude of the response in session 10 was subtracted from the magnitude of the response in session 1 ($\bar{D}_1-\bar{D}_{10}$). The second measure, denoted as the absolute score ($\bar{A}$),was simply the difference between the obtained response magnitudes (mean minimum IBI post-shock) of sessions 1 and 10 ($\bar{A}_1-\bar{A}_{10}$). The statistical significance of these differences was tested by a Student's t-test, and are presented in Table 4. To facilitate comparisons with changes in response magnitude across sessions when pre-shock levels are equated by means of the regression analysis, the data in Table 3 are reproduced in Table 4.

Table 4 shows that for the difference score, when pre-UCS level decreased the amount of diminution in the acceleratory

Table 4

t-Tests For The Amount of Change in the Magnitude of the Acceleratory Phase of the Response to Shock Between Sessions 1 and 10 of Stage 1, as Measured by (1) Difference Scores ($\bar{D}_1 - \bar{D}_{10}$), (2) Absolute Scores ($\bar{Y}_1 - \bar{Y}_{10}$), and (3) Adjusted Scores ($Y'_1 - \bar{Y}_{10}$)

Intensity (mA)	Subjects	$\bar{D}_1 - \bar{D}_{10}$	$\bar{Y}_1 - \bar{Y}_{10}$	$Y'_1 - \bar{Y}_{10}$	$\bar{X}_1 - \bar{X}_{10}$**
13.5	D-50	+25*	-42*	- 8	-67*
	D-90	-13	+36*	+29*	+23
	C-36	+30*	-43*	-40*	-73*
4.0	E-22	- 8	-31*	-25*	-23*
	E-16	-27*	-34*	-30*	- 6
	E-8	-17*	- 1	-10*	+15
0.5	D-20	+ 8	-30*	- 9	-38*
	D-94	- 1	-13*	- 4	-12
	D-92	+13	+24	+18	+11

*$p < .05$

** $\bar{X}_1 - \bar{X}_{10}$ = Difference in pre-shock levels between sessions 1 and 10 of Stage 1

phase of the response is smaller than after adjustment for shifts pre-shock levels. In fact, in a number of subjects the difference score shows an increase in response magnitude over sessions. Thus, difference scores unadjusted for shifts in pre-shock levels showed, in general, either a smaller decrease in the magnitude of the acceleratory phase or an increase in magnitude when compared to changes across sessions adjusted for shifts in pre-shock levels. Table 4 also shows that for the absolute score changes in the magnitude of the response across sessions were generally in the same direction as the changes in the magnitude of the response after adjustment for shifts in pre-shock levels. However, the amount of change reflected in the absolute scores was generally larger than the adjusted scores.

Stage 2

Stage 2 consisted of 10 sessions of conditioning (light-shock pairings). There were several objectives in the analysis of this stage. The first was to determine the effects of conditioning on the UCR. A second was to examine the manner in which conditioning affects basal (pre-stimulus) IBI levels and how this relates to changes in response magnitude. A third was to determine the extent to which changes in the response to shock (UCR) during shock-alone presentations (Stage 1), as well as over the course of conditioning sessions (Stage 2), could account for the classical observation in the literature that CR and UCR are of different magnitude, temporal form, and latency.

Response to CS (CR). Figure 4 presents the group curves of mean IBI per bin for session 10 of Stage 2 for both the response to CS and UCS. Each data point represents the mean of three animals. Analysis of individual subject response curves revealed that nine of the twelve animals demonstrated a clear CR by session 10. As can be seen in Figure 3, the 0.5 mA group showed the poorest conditioning of all the groups. Figure 4 also shows that the temporal form of the CR for all four groups was biphasic (acceleration followed by deceleration), and that CR magnitude was positively related to shock intensity. However, from the data presented in Figure 4 it is difficult to compare the CR of the NP-4.0 mA group to the other groups as the pre-CS mean IBI level is much higher in this group than the other groups.

A one-way analysis of covariance for among groups comparisons was conducted on the data of Figure 4 in order to determine if there were statistically significant differences in response magnitude among the groups when pre-CS IBI levels were equated. The F-ratio testing the difference in the magnitude of the acceleratory phase among groups was significant ($F = 11.5$, $df = 3, 7$, $p < .01$), while the F-ratio for the deceleratory phase was non-significant ($F = .19$, $df = 3,7$). A multiple comparisons among means test was performed on the magnitude of the acceleratory phase of the CR in session 10 and Table 5 presents the results of the pairwise comparisons among means. A negative difference score indicates that the adjusted mean magnitude in column A was larger than its corresponding value in row B. The results presented in this table confirm the

Figure 4. Mean IBI in successive 2-sec bins for the 13.5 mA, 4.0 mA, 0.5 mA, and NP-4.0 mA groups, in session 10 of Stage 2, for the response to light (CS) and shock (UCS). Each data point represents the session mean IBI per bin, averaged over the three animals in each group. The first data point in each curve represents the mean IBI 2-sec pre-CS; the sixth point represents the mean IBI 2-sec pre-UCS, and is also the last bin in CS. Arrows indicate CS onset (C) and UCS onset (U).

Fig. 4

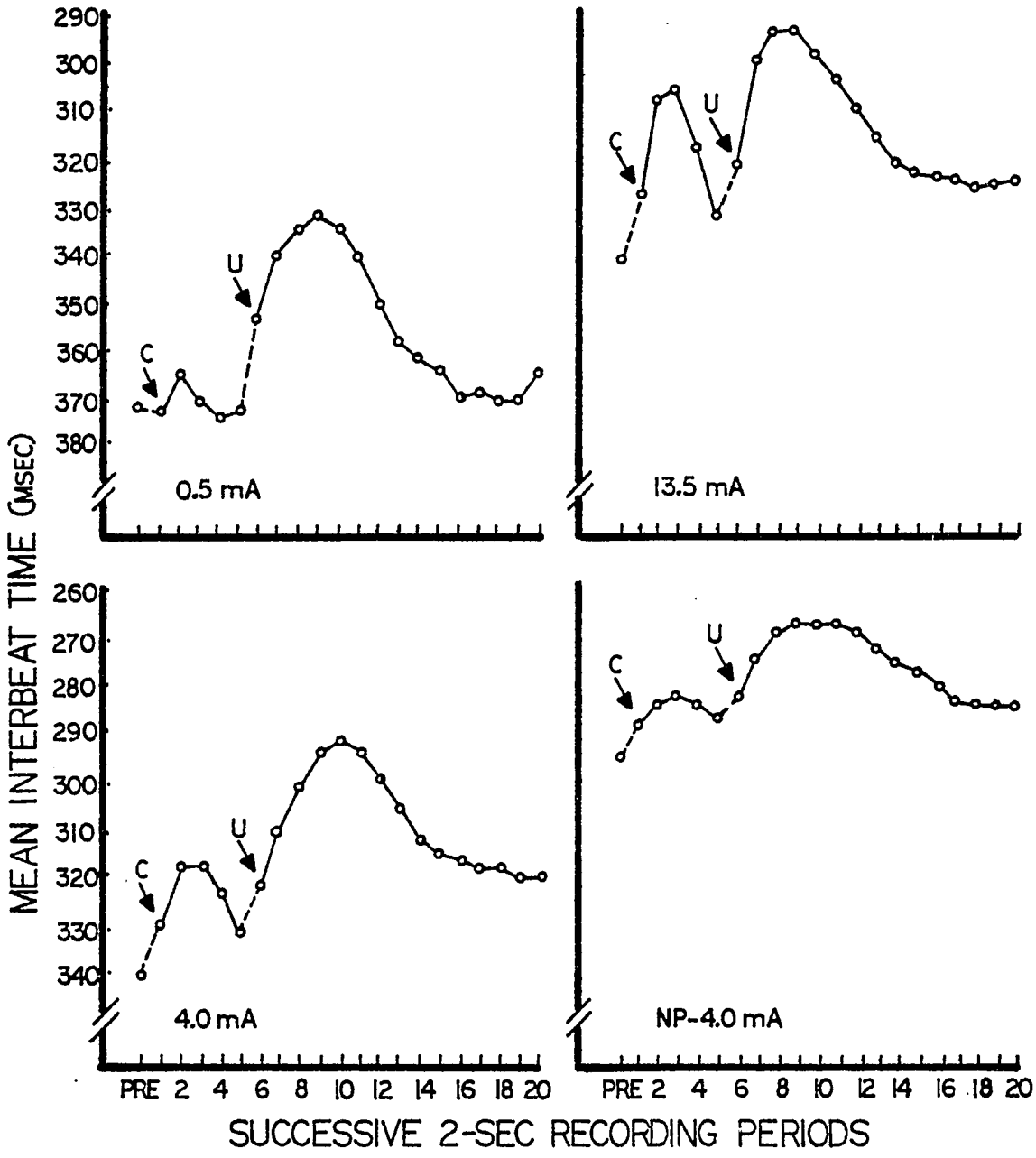

MEAN INTERBEAT TIME (MSEC)
290
300
310
320
330
340
350
360
370
380
C
U
0.5 mA
13.5 mA
260
270
280
290
300
310
320
330
340
4.0 mA
NP-4.0 mA
PRE 2 4 6 8 10 12 14 16 18 20
SUCCESSIVE 2-SEC RECORDING PERIODS

Table 5

Multiple Comparisons Among Means Tests For the Acceleratory Phase of the Response to Light (CR) in Stage 2, Session 10

Intensity (mA)			13.5	4.0	0.5	NP-4.0
		B	300	316	334	321
	A		d	d	d	d
13.5	300			-16*	-34*	
4.0	316				-18*	-05
0.5	334					
NP-4.0	321					

$F = +11.5$, $df = 3,7$, $p < .01$

* $p < .05$; Adjusted means (A,B) in msec; d = A-B

finding reported in Figure 4 that the magnitude of the acceleratory phase of the CR is positively related to UCS intensity. However, when differences in pre-CS IBI levels were equated among groups there was no difference between the 4.0 mA group(shock presentations prior to conditioning) and the NP-4.0 mA group (no prior shock presentations).

Response to Shock (UCR). Figure 4 shows that the temporal form of the UCR at the end of conditioning retained its biphasic form. Comparison of the UCR in Figures 2 and 4 shows that the magnitude of the acceleratory phase of the UCR at the end of Stage 2 in comparison to the end of Stage 1 was smaller for the 4.0 and 13.5 mA groups and larger for the 0.5 mA group.

Table 6 presents the results of the t-tests for individual subject comparisons comparing the predicted response magnitudes of session 10 of Stage 1 and the obtained response magnitudes of session 10 of Stage 2 for both phases of the UCR. The NP-4.0 mA group was excluded from this analysis since the animals in this group did not receive UCS-alone presentations in Stage 1. The majority of the animals in the 4.0 and 13.5 mA groups showed a reduction in the magnitude of the acceleratory phase of the UCR by the final session of conditioning. All the animals in the 0.5 mA group showed an increase in the magnitude of the acceleratory phase. The results for the deceleratory phase of the response parallel those of the acceleratory phase: the majority of animals in the 13.5 and 4.0 mA groups showed an increase in the magnitude of the deceleratory phase of the UCR, while all the animals in the 0.5 mA group showed a decrease in the magnitude of the deceleratory phase.

Table 6

t-Tests For the Difference Between the Predicted Magnitudes of Stage 1, Session 10 ($Y'_{1,10}$) and the Obtained Magnitudes of Stage 2, Session 10 ($\bar{Y}_{2,10}$)

Response to Shock (UCR): Acceleratory Phase

Intensity (mA)	Subject	$Y'_{1,10}-\bar{Y}_{2,10}$ (msec)
13.5	D-50	-22*
	D-90	-16*
	C-36	+ 6
4.0	E-22	-10*
	E-16	-22*
	E-8	-17*
0.5	D-20	+68*
	D-94	+10*
	D-92	+18*

Response to Shock (UCR): Deceleratory Phase

Intensity (mA)	Subject	$\bar{Y}_{2,10}-Y'_{1,10}$ (msec)
13.5	D-50	+20*
	D-90	+21*
	C-36	-39*
4.0	E-22	+15*
	E-16	+11
	E-8	+15
0.5	D-20	-25*
	D-94	-12
	D-92	- 4

* $p < .05$

The changes in the magnitude of both phases of the response found across the sessions of conditioning were predominantly in the same direction as those found across the sessions of Stage 1 (UCS-alone presentations), particularly for the 4.0 and 13.5 mA groups. Thus, both the procedures of UCS-alone presentations as well as conditioning have the effect of decreasing the magnitude of the acceleratory phase of the UCR and increasing the magnitude of the deceleratory phase.

Relationship Between the CR and UCR. It can be seen in Figure 4 that the temporal form of both the CR and UCR were approximately the same for all groups. It is also apparent from this figure that the latency (as measured by the ordinal number of the bin in which the peak of the acceleratory phase occurs) of both responses was approximately equal, although the latency of the UCR was generally slightly longer than that of the CR. Furthermore, Figure 4 shows that the higher the intensity of the shock the smaller the difference between the latency of the response to light and the response to shock. Additionally, the higher the intensity of the shock the smaller the difference between the magnitude of the CR (as measured by the difference between the mean IBI pre-CS and the smallest IBI post-CS) and the magnitude of the UCR (as measured by the difference between the mean of the last bin during the CS and the smallest mean IBI post-UCS). Finally, it can also be seen in Figure 4 that the larger the magnitude of the CR the smaller the magnitude of the UCR.

Another relationship found was as follows: the larger the decrease in the magnitude of the acceleratory phase

of the UCR during conditioning the smaller the difference between the magnitudes of the acceleratory phase of the CR and UCR. This relationship was determined by correlating the difference between the magnitudes of the acceleratory phase of the CR and UCR with the amount the acceleratory phase of the UCR to shock decreased during conditioning. The measure of the decrease in the acceleratory phase of UCR was the difference between the predicted values of session 10 of Stage 1 and the obtained values of session 10 of Stage 2 (see Table 6). The measure of the difference between the magnitude of the CR and the UCR was the difference between the peaks (mean minimum IBI post-stimulus) of the acceleratory phase of the two responses. This value was calculated for each animal in the 0.5, 4.0, and 13.5 mA groups, for session 10 of Stage 2. The correlation between these two measures was negative and significant ($r = -.87$, $df = 16$, $p < .05$). Thus, the larger the decrement in the magnitude of the acceleratory phase of the UCR during conditioning the smaller the difference between the magnitudes of the CR and UCR. A similar correlation was determined for the amount of change in the acceleratory phase of the UCR in Stage 1 and the difference between the magnitudes of the CR and UCR in session 10 of Stage 2. This correlation was negative, but non-significant ($r = -.27$, $df = 16$). Thus, the extent to which the magnitude of the CR and UCR differ at the end of conditioning depends, in part, on the degree to which the UCR magnitude is reduced during conditioning, but is not dependent on the degree to which the UCR decreases during presentations of shock prior to conditioning.

Between-Groups Differences (UCR). To assess the differences in response magnitude among the groups in sessions 1 and 10 of Stage 2 a one-way analysis of covariance was conducted. The F-ratio testing the differences in the magnitude of the acceleratory phase among groups for session 1 was significant ($F = 7.25$, $df = 3,7$, $p < .05$), but non-significant for session 10 ($F = 0.63$, $df = 3,7$). The F-ratios testing the differences in the magnitude of the deceleratory phase among groups were non-significant for both session 1 ($F = .82$, $df = 3,7$) and session 10 ($F = .04$, $df = 3,7$). The results of the multiple comparisons among means test for the adjusted mean magnitudes of the acceleratory phase of the UCR in sessions 1 and 10 are presented in Tables 7 and 8 respectively. The organization of these tables is similar to that of Table 5. The adjusted means are presented for session 10, although the F-ratio was non-significant, for purposes of comparison. The results show that at the beginning of conditioning (session 1) the magnitude of the acceleratory phase of the responsewas positively related to shock intensity. Furthermore, the magnitudes of the acceleratory phase of the UCR for the NP-4.0 mA and 4.0 mA groups were approximately equal at the beginning of conditioning. However, by the end of conditioning the magnitude of the acceleratory phase was approximately equal for all groups.

To compare the effects of shock repetition and conditioning several comparisons were made between the 4.0 mA group (prior shock presentations) and the NP-4.0 mA group (no prior shock presentations). The first determined whether or not there were differences in the amount of decrement in the UCR between these

Table 7

Multiple Comparisons Among Means Test For the Acceleratory Phase of the Response to Shock (UCR) in Stage 2, Session 1

Intensity (mA)		13.5	4.0	0.5	NP-4.0
	B	261	272	293	272
	A	d	d	d	d
13.5	261		-11	-32*	
4.0	272			-21*	0
0.5	293				
NP-4.0	272				

$F = +7.25$, $df = 3,7$, $p < .05$

* $p < .05$; Adjusted means (A,B) in msec; d = A-B

Table 8

Multiple Comparisons Among Means Tests For the Acceleratory Phase of the Response to Shock (UCR) in Stage 2, Session 10

Intensity (mA)		13.5	4.0	0.5	NP-4.0
	B	288	285	301	285
	A	d	d	d	d
13.5	288		+3	-13	
4.0	285			-16	0
0.5	301				
NP-4.0	285				

F = .63, df = 3,7

*
p <.05; Adjusted means (A,B) in msec; d = A-B

two groups during conditioning. To compare the effects of conditioning on the response to shock t-tests for individual subjects were conducted for the animals in the 4.0 mA and NP-4.0 mA groups. Table 9 presents the results of the t-tests testing the difference between the predicted magnitudes of session 1 of Stage 2, and the obtained magnitudes of session 10 of Stage 2. All the animals in both the 4.0 mA and NP-4.0 mA groups showed a reduction in the magnitude of the acceleratory phase of the response to shock and an increase in the magnitude of the deceleratory phase by the end of conditioning. It can also be seen in Table 9 that the amount of change over conditioning sessions was approximately equal for both groups.

The above results showed no differences between the 4.0 mA and NP-4.0 mA groups in the amount of UCR diminution during conditioning. However, there were substantial differences in the change in the magnitude of the UCR when the UCS-alone and conditioning sessions were combined for the 4.0 mA group, in comparison to the changes in UCR magnitude for the NP-4.0 mA group during conditioning. To determine the amount of change in the response to shock for the 4.0 mA group over the UCS-alone (Stage 1) and conditioning (Stage 2) sessions together, the difference between the predicted response magnitude of session 1 of Stage 1 and the obtained response magnitude of session 10 of Stage 2 were determined for each animal. Table 10 presents the results of the t-tests for this comparison for both phases of the UCR. It can be seen that the amount of decrement in the magnitude of the acceleratory phase of the UCR and increment in the deceleratory phase over the UCS-alone and conditioning sessions combined was substantially larger

Table 9

t-Tests For the Difference Between the Predicted Magnitudesof Stage 2, Session 1 ($Y'_{2,1}$) and the Obtained Magnitudes of Stage 2, Session 10 ($\bar{Y}_{2,10}$)

Response to Shock (UCR): Acceleratory Phase

Intensity (mA)	Subjects	$Y'_{2,1}-\bar{Y}_{2,10}$ (msec)
4.0	E-22	-13*
	E-16	-13*
	E-8	-37*
NP-4.0	E-20	-14*
	E-10	- 9*
	E-4	-13*

Response to Shock (UCR): Deceleratory Phase

Intensity (mA	Subjects	$\bar{Y}_{2,10}-Y'_{2,1}$ (msec)
4.0	E-22	+13
	E-16	+13
	E- 8	+58*
NP-4.0	E-20	+15*
	E-10	+16*
	E-4	+41*

* $p < .05$

Table 10

t-Tests For the Difference Between the Predicted Magnitudes of Stage 1, Session 1 ($Y'_{1,1}$) and the Obtained Magnitudes of Stage 2, Session 10 ($\overline{Y}_{2,10}$)

Response to Shock (UCR): Acceleratory Phase

Intensity (mA)	Subjects	$Y'_{1,1}-\overline{Y}_{2,10}$ (msec)
4.0	E-22	-36*
	E-16	-52*
	E-8	-20*

Response to Shock (UCR): Deceleratory Phase

Itensity (mA)	Subjects	$\overline{Y}_{2,10}-Y'_{1,1}$ (msec)
4.0	E-22	+35*
	E-16	+92*
	E-8	+27*

* $p < .05$

for the 4.0 mA group when compared to changes in UCR magnitude of the NP-4.0 mA group during conditioning (See Table 9).

It was also possible to compare the separate effects of conditioning and UCS-alone presentations on the UCR by comparing the data in Stage 1 (UCS-alone presentations) for the 4.0 mA group (Table 3) to the data in Stage 2 (conditioning) for the NP 4.0 mA group (Table 9), since the latter group had no prior history of shock; conditioning was the first exposure to any stimulus operation. The results of this comparison showed a larger decrease in the magnitude of the acceleratory phase to the UCS-alone presentations (mean decrease of 22 msec; N=3) than to conditioning (mean decrease of 12 msec; N=3). For the deceleratory phase of the response there was a larger increase in UCR magnitude to shock-alone presentations (mean increase of 37 msec; N=3) than to conditioning (mean increase of 24 msec; N=3).

Pre-stimulus (baseline) level. Figure 4 shows that the pre-UCS level for the 4.0 mA group was much lower than for the NP-4.0 mA group. To determine whether or not this difference was related to the difference in prior shock presentations a comparison was made between baseline IBI levels over conditioning sessions. Since no separate baseline sessions were conducted prior to the experiment for the 4.0 mA group the pre-CS level in trial 1 of session 1 for the first stage was used as a baseline measure for this group before any stimuli were introduced. Sessions in which baseline IBI levels were recorded before the introduction of stimuli were available for the NP-4.0 mA group. For consistency with the 4.0 mA group the mean IBI of the first bin in the first trial of the last

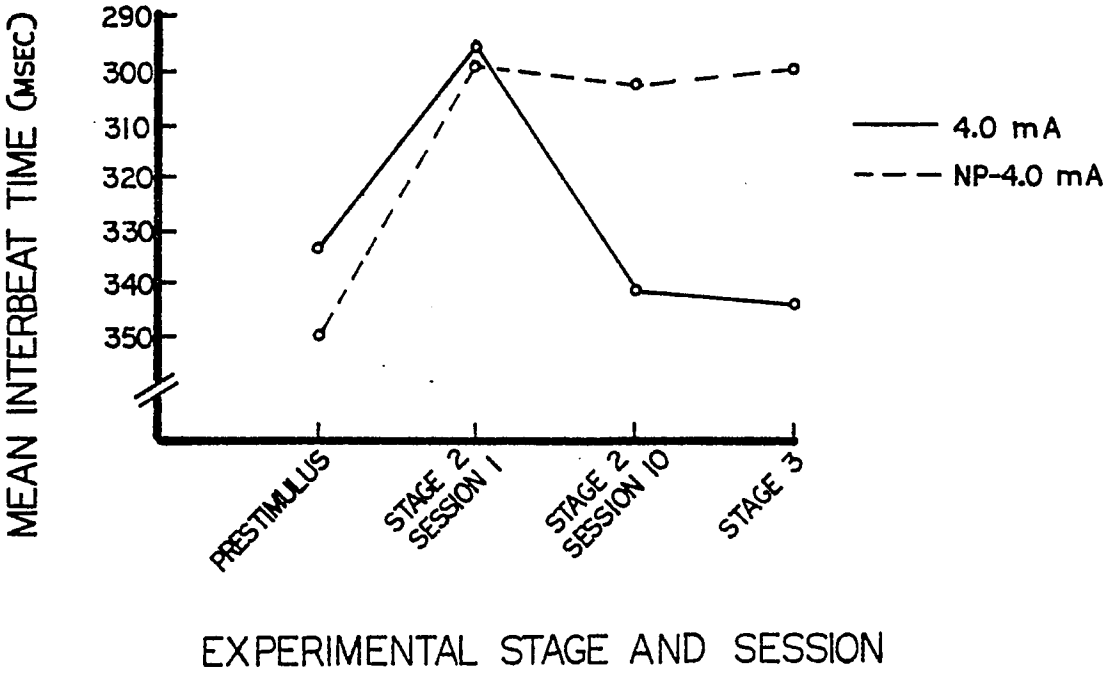
"baseline" session was used. The mean IBI baseline level for each group was determined and the difference between groups was tested by means of a Student's t-test and was non-significant ($t = .52$, $df = 4$). Thus, there was no statistically significant difference between the baseline levels of the 4.0 and NP-4.0 mA groups.

Figure 5 shows the change in baseline and pre-shock levels over the course of Stages 2 and 3. The first data point is the baseline level before any stimuli were introduced. It can be seen from this figure that baseline levels of the two groups were equivalent both before stimulation and during the first session of conditioning ($t = .07$, $df = 4$). However, by the final session of conditioning the pre-CS levels between the two groups differed greatly ($t = 3.21$, $df = 4$, $p < .05$) and this difference was maintained for the pre-UCS levels of Stage 3 (UCS-alone presentations). These results indicate that the pre-stimulus (baseline) IBI levels declined over the conditioning sessions for the 4.0 mA group, while they remained high and showed little change for the NP-4.0 mA group.

Relationship of pre-shock levels and the magnitude of the response to shock. Since in the majority of subjects there was a substantial CR by the end of conditioning, it was possible that part of the decrease in the magnitude of the acceleratory phase of the response over conditioning sessions could be attributed to the increase in pre-shock levels due to CR. To assess the relationship of the increase in pre-UCS levels over conditioning sessions to the decrease in the magnitude of the acceleratory phase of the UCR a comparison was made between the

Figure 5. Pre-stimulus levels during Stage 2 (conditioning) and Stage 3 (UCS-alone), for the 4.0 and NP-4.0 mA groups. Each data point represents the session means averaged over the three animals in each group for the mean IBI 2-sec pre-stimulus. The first data point represents the mean IBI for the first 2-sec in trial 1 of session 1 of Stage 1 (i.e., prior to stimulation) for the 4.0 mA group, and the mean IBI for the first 2-sec bin of the first trial of the last "baseline" (no stimulation) session, for the NP-4.0 mA group. For Stage 2, pre-stimulus levels are mean IBI pre-CS; for Stage 3, pre-stimulus levels are mean IBI pre-UCS.

Fig. 5


MEAN INTERBEAT TIME (MSEC)
290
300
310
320
330
340
350
PRESTIMULUS
STAGE 2 SESSION 1
STAGE 2 SESSION 10
STAGE 3
4.0 mA
NP-4.0 mA
EXPERIMENTAL STAGE AND SESSION

changes in the magnitude of the UCR before and after pre-UCS levels were equated between sessions 1 and 10 of Stage 2. To compare the amount of change in UCR magnitude before adjustment for shifts in pre-UCS levels the difference between the obtained magnitudes of the acceleratory phase of the UCR of session 10 of Stage 1 and session 10 of Stage 2 ($\bar{Y}_1 - \bar{Y}_{10}$) was computed and was tested by a Student's t-test. The results of this analysis are presented in Table 11. To facilitate comparison with the amount of change over conditioning sessions after adjustment for changes in pre-UCS levels the data reported in Table 6 ($Y_1' - \bar{Y}_{10}$) are reproduced in Table 11.

Table 12 shows that for the 4.0 and 13.5 mA groups, the amount of decrease in the acceleratory phase of the UCR over conditioning sessions was generally larger before adjustment for shifts in pre-UCS level than after. For the 0.5 mA group the increase in the magnitude of the acceleratory phase is generally larger before adjustment for pre-shock levels than after.

A similar analysis was conducted comparing the 4.0 and NP-4.0 mA groups. Since the NP-4.0 mA group did not receive shock-alone presentations the comparison between UCR magnitudes before and after adjustment was made between sessions 1 and 10 of Stage 2. Table 12 presents the results of the Student's t-test testing the difference between the obtained response magnitudes of sessions 1 and 10 of Stage 2. Again, to facilitate comparison with the changes in UCR magnitude after changes in pre-UCS levels were equated over sessions the data in Table 9 ($Y_1' - \bar{Y}_{10}$) are reproduced in Table 12. Generally,

Table 11

t-Tests For the Difference Between the Obtained Magnitudes of Stage 1, Session 10 ($\bar{Y}_{1,10}$) and Stage 2, Session 10 ($\bar{Y}_{2,10}$), and t-Tests For the Difference Between the Predicted Magnitudes of Stage 1, Session 10 ($Y'_{1,10}$) and the Obtained Magnitudes of Stage 2, Session 10 ($\bar{Y}_{2,10}$), For the Acceleratory Phase of the Response to Shock

Intensity (mA)	Subject	$\bar{Y}_{1,10}-\bar{Y}_{2,10}$	$Y'_{1,10}-\bar{Y}_{2,10}$
13.5	D-50	-40*	-22*
	D-90	-17*	-16*
	C-36	+16*	+ 6
4.0	E-22	-12*	-10*
	E-16	-22*	-22*
	E-8	-40*	-17*
0.5	D-20	+73*	+68*
	D-94	+18*	+10*
	D-92	+15*	+18*

* $p < .05$

Table 12

t-Tests For the Difference Between the Obtained Magnitudes of Stage 2, Session 1 ($\bar{Y}_{2,1}$) and Stage 2, Session 10($\bar{Y}_{2,10}$), and t-Tests For the Difference Between the Predicted Magnitudes of Stage 2, Session 1 ($Y'_{2,1}$) and the Obtained Magnitudes of Stage 2, Session 10($\bar{Y}_{2,10}$), For the Acceleratory Phase of the Response to Shock

Intensity (mA)	Subjects	$\bar{Y}_{2,1}-\bar{Y}_{2,10}$	$Y'_{2,1}-\bar{Y}_{2,10}$
4.0	E-22	-12*	-13*
	E-16	-27*	-13*
	E-8	-48*	-37*
NP-4.0	E-20	-19	-14*
	E-10	- 4	- 9*
	E-4	-16*	-13*

* $p < .05$

for the 4.0 mA group the decrease in the magnitude of the acceleratory phase of the UCR was larger before adjustment for changes in pre-UCS levels than after. The NP-4.0 mA group showed essentially no differences in the amount of change in UCR magnitude before and after pre-UCS adjustment.

These results show that the decrease in the magnitude of the acceleratory phase of the response for the 4.0 and 13.5 mA groups, and the increase in the magnitude of the acceleratory phase for the 0.5 mA group, was generally larger before shifts in pre-shock levels were equated by means of the regression analysis. For the NP-4.0 mA group, there was little difference in the amount of decrease in the magnitude of the acceleratory phase of the response to shock before and after adjustment of pre-shock level.

In addition to the heart-rate at the time at which the UCS occurs determining UCR magnitude, another factor may be the rate at which the heart is changing during the intrusion of the UCS. To determine the effect of this variable on the UCR the relationship between the slope of the deceleratory phase of the CR (i.e., the rate of deceleration), and the magnitude and latency of the UCR was determined. The slope of the deceleratory phase was approximated by fitting a straight line (by the method of least squares) to all the IBIs of the deceleratory phase of the CR, on a trial-by-trial basis, in session 10 of Stage 2. The slope of this line constituted the approximate rate of deceleration. For each animal the mean rate of deceleration for session 10 of Stage 2 was determined.

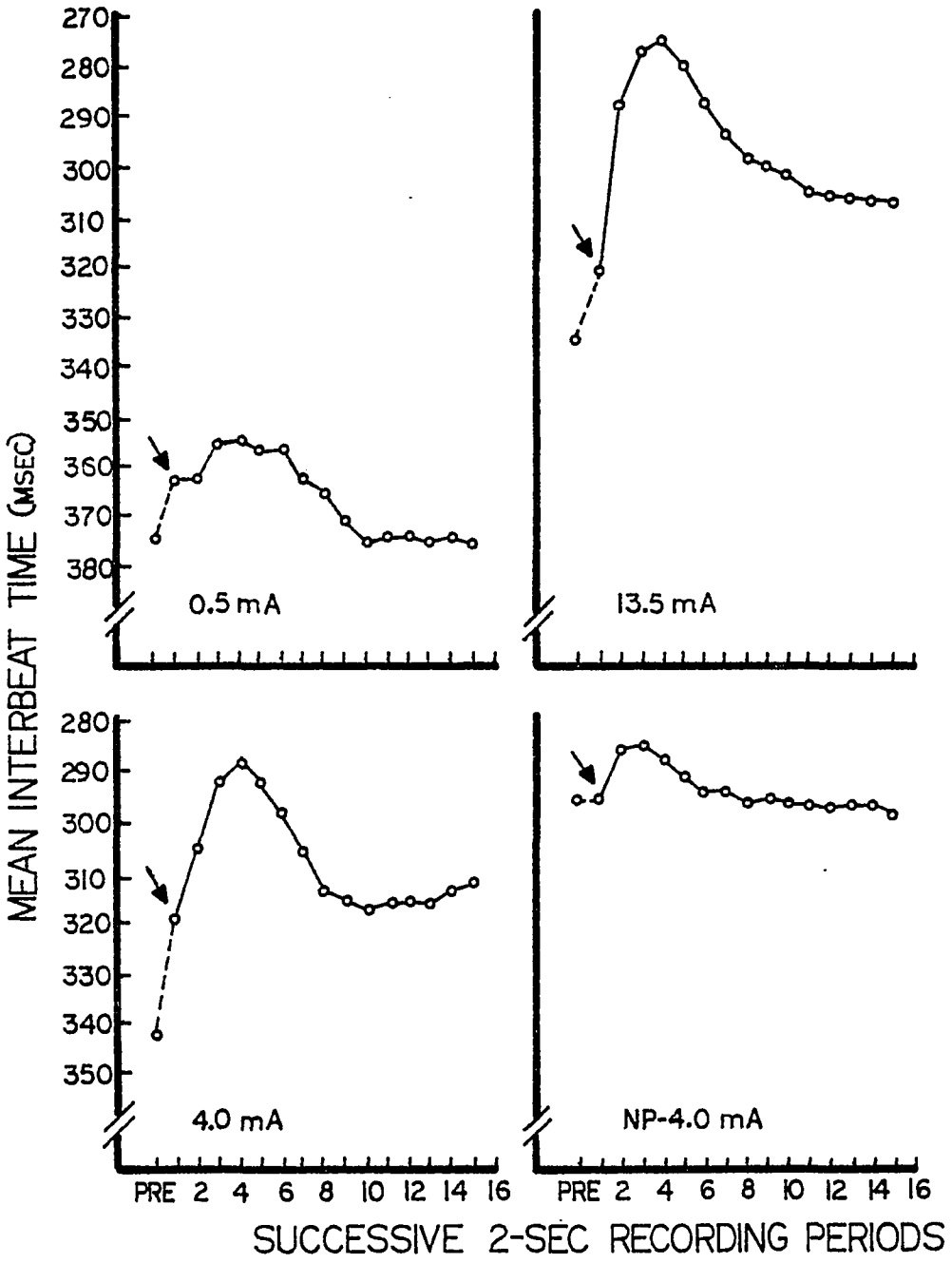
To assess the relationship between the rate of CR deceleration and UCR magnitude a Pearson product-moment correlation was computed for the slope of the deceleratory phase of the CR and the peak of the acceleratory phase (minimum mean IBI post-UCS) of the UCR for session 10 of Stage 2. The correlation was positive and significant ($r = +.48$. $df = 22$, $p < .05$). This direct relationship indicates that the faster the heart was decelerating the smaller the subsequent UCR. To determine the relationship between the rate of CR deceleration and UCR latency a Pearson product-moment correlation was computed for the slope of the deceleratory phase of the CR and the latency (mean ordinal number of the bin in which the peak of the acceleration occurred) of the UCR in session 10 of Stage 2. The correlation was negative and significant ($r = -.44$, $df = 22$, $p < .05$). This inverse relationship indicates that the faster the heart was decelerating the longer the latency of the peak of the UCR.

Stage 3

Stage 3 (one session of UCS-alone presentations) determined the effect of removing the CS on the magnitude and temporal form of the UCR. Figure 6 presents the group curves of mean IBI per bin for Stage 3. Each data point represents the mean of three animals. The UCR retained its characteristic biphasic form in Stage 3. However, there is a substantial change in the magnitude of the acceleratory phase of the UCR in Stage 3 when compared to the end of conditioning (Figure 4). For the 4.0 and 13.5 mA groups the magnitude of the acceleratory phase

Figure 6. Mean IBI in successive 2-sec bins for the 13.5 mA, 4.0 mA, 0.5 mA, and NP-4.0 mA groups, in Stage 3, for the response to shock (UCS). Each data point represents the session mean IBI per bin, averaged over the three animals in each group. The first data point in each curve represents the mean IBI 2-sec pre-UCS. Arrows indicate UCS onset.

Fig. 6


MEAN INTERBEAT TIME (MSEC)
270
280
290
300
310
320
330
340
350
360
370
380
0.5 mA
13.5 mA
280
290
300
310
320
330
340
350
4.0 mA
NP-4.0 mA
PRE 2 4 6 8 10 12 14 16
PRE 2 4 6 8 10 12 14 16
SUCCESSIVE 2-SEC RECORDING PERIODS

increased while it decreased for the 0.5 and NP-4.0 mA groups.

To assess whether the changes in response magnitude between session 10 of Stage 2 and Stage 3 were statistically reliable the t-test for individual subjects was employed. Table 13 presents the results of the t-tests for the difference between the predicted response magnitudes of session 10 of Stage 2 and the obtained response magnitudes of Stage 3 for both phases of the UCR. Five of the six animals in the 4.0 and 13.5 mA groups showed an increase in the magnitude of the acceleratory phase while one subject showed a decrease in magnitude. For the 0.5 mA group two animals showed a decrease in the magnitude of the acceleratory phase and one subject showed a small increase in magnitude. All the animals in the 4.0 and 13.5 mA groups showed a decrease in the magnitude of the deceleratory phase of the UCR. Two animals in the 0.5 mA group showed an increase in the magnitude of the deceleratory phase while the other animal showed a small increase. While all the groups that received shock presentations prior to conditioning showed a change in UCR magnitude in Stage 3 this was not the case with the NP-4.0 mA group (no prior shock). For the NP-4.0 mA group removal of the CS had little effect on the magnitude of either phase of the UCR.

In sum, for all but the NP-4.0 mA group the predominent effect of removing CS in Stage 3 was to reverse the direction of the changes in the magnitude of both phases of the UCR found during conditioning. That the changes in UCR magnitude in Stage 3 were opposite in direction to those in Stage 2 indicates that the UCR magnitude recovered to pre-conditioning levels when the CS was removed and UCS-alone was presented. To assess

Table 13

t-Tests For the Difference Between the Predicted Magnitudes of Stage 2, Session 10 ($Y'_{2,10}$) and the Obtained Magnitudes of Stage 3 ($\bar{Y}_3$)

Response to Shock (UCR): Acceleratory Phase

Intensity	Subjects	$Y'_{2,10}-\bar{Y}_3$ (msec)
13.5	D-50	+48*
	D-90	+23*
	C-36	+11*
4.0	E-22	-12
	E-16	+15*
	E-8	+17*
0.5	D-20	-54*
	D-94	- 3
	D-92	+ 2
NP-4.0	E-20	- 9
	E-10	+ 3
	E-4	- 6

Response to Shock (UCR): Deceleratory Phase

Intensity	Subjects	$\bar{Y}_3-Y'_{2,10}$ (msec)
13.5	D-50	-33*
	D-90	-43*
	C-36	-19*
4.0	E-22	- 5
	E-16	-12
	E-8	-18
0.5	D-20	+25*
	D-94	+ 5
	D-92	- 9
NP-4.0	E-20	- 1
	E-10	- 9
	E-4	- 3

* $p < .05$

the extent of recoverythe UCR magnitude in Stage 3 was compared to the first session of conditioning. Since the NP-4.0 mA group was not included in Stage 1, the first session of conditioning was used as the index of pre-conditioning levels instead of session 10 of Stage 1. However, the comparison between the last session of Stage 1 and Stage 3 was conducted for the groups included in Stage 1. The results were essentially the same as using the first session of conditioning and validates the use of the first session as the index of pre-conditioning levels. This analysis was conducted only on the acceleratory phase of the UCR.

Table 14 presents the results of the t-tests of the difference between the predicted response magnitudes of session 1 of Stage 2 with the obtained response magnitudes of Stage 3. For the 13.5 mA group the response magnitude either remained the same or increased slightly by Stage 3. Thus, when the CS was removed in the 13.5 mA group the magnitude of the UCR returned completely to pre-conditioning levels. The results for the 4.0 mA group showed that UCR magnitude decreased in Stage 3, indicating that UCR magnitude only partially recovered to preconditioning levels. For the 0.5 mA group there was either no change in UCR magnitude or a decrease. However, for this group intrusion of the CS in Stage 2 increased the magnitude of the acceleratory phase over conditioning sessions. Thus, for these animals UCR magnitude decreased toward pre-conditioning levels in Stage 3, while for the other two groups UCR magnitude increased toward pre-conditioning levels. Finally, the NP-4.0 mA group showed a decrease in UCR magnitude in Stage 3 when compared to pre-

Table 14

t-Tests For the Difference Between the Predicted Magnitudes of Stage 2, Session 1 ($Y'_{2,1}$) and the Obtained Magnitudes of Stage 3 ($\overline{Y}_3$), For the Acceleratory Phase of the Response to Shock

Intensity (mA)	Subjects	$Y'_{2,1}-\overline{Y}_3$ (msec)
13.5	D-50	+ 7*
	D-90	+ 4*
	C-36	+11*
4.0	E-22	-28*
	E-16	+ 5
	E-8	-23*
0.5	D-20	-42*
	D-94	- 1
	D-92	- 8
NP-4.0	E-20	-42*
	E-10	- 6*
	E-4	-19*

* $p < .05$

conditioning levels. However, removal of CS had virtually no effect on UCR magnitude in this group and the data of the present comparison reflect something other than recovery to preconditioning levels for the NP-4.0 mA group.

To compare differences in UCR magnitude among groups in Stage 3 a one-way analysis of covariance was conducted. The F-ratio for the magnitude of the acceleratory phase of the UCR was significant ($F = 4.55$, $df = 3.7$, $p < .05$), while non-significant for the magnitude of the deceleratory phase ($F = 1.30$, $df = 3,7,$). The results of the multiple comparisons among means test for the adjusted mean magnitudes of the acceleratory phase of the UCR are reported in Table 15. The organization of Table 15 is similar to that of Table 5. The results show that the magnitude of the acceleratory phase was positively related to shock intensity. Furthermore, the magnitude of the acceleratory phase of the 4.0 mA group was significantly larger than the NP-4.0 mA group.

Stage 4

Stage 4 consisted of 10 sessions of light-alone presentations and determined if the CR would extinguish after 160 extinction trials. Figure 7 presents the group curves of mean IBI per bin for session 10 of Stage 4 for all four groups. Each data point represents the mean of three animals. Comparing the data presented in Figure 7 with the response to light in Figure 4 (i.e., the last session of conditioning) shows that the magnitude of the acceleratory phase of the response to light was reduced at the end of the extinction trials for the 13.5, NP-

Table 15

Multiple Comparisons Among Means Tests For the Acceleratory Phase of the Response to Shock (UCR) in Stage 3

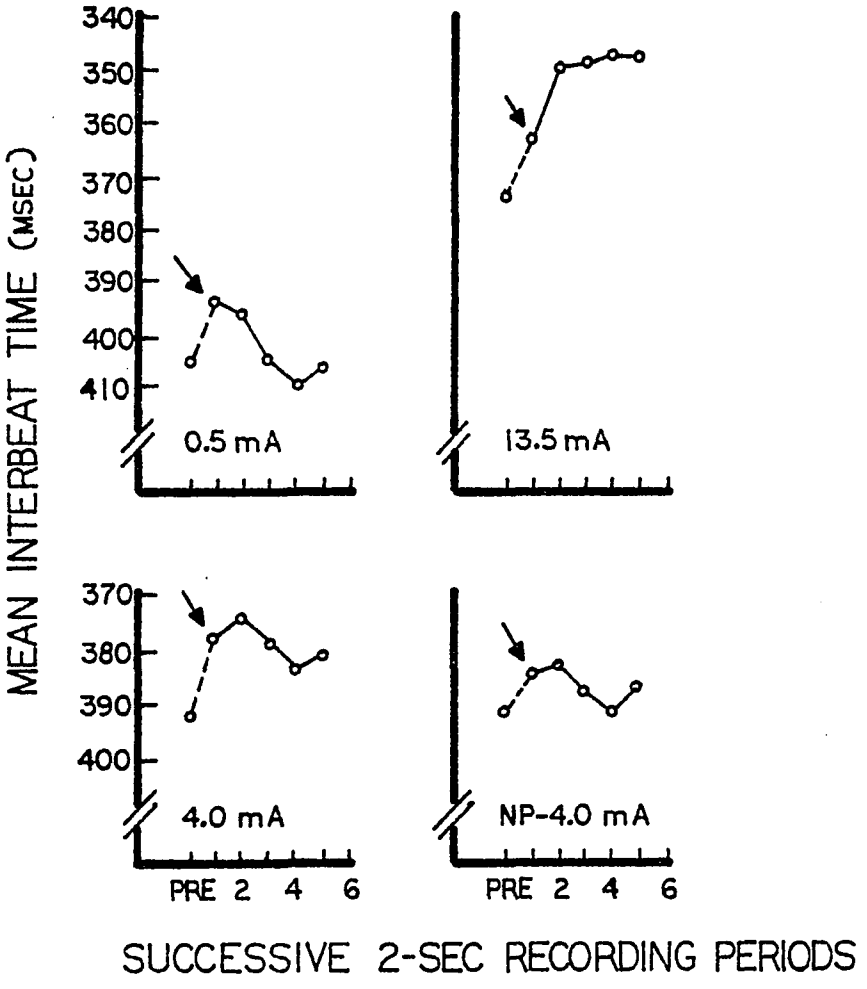
Intensity (mA)		13.5	4.0	0.5	NP-4.0
	B	272	279	312	308
	A	d	d	d	d
13.5	272		-07	-40*	
4.0	279			-33*	-29*
0.5	312				
NP-4.0	308				

F = +4.55, df = 3,7, $p < .05$

*$p < .05$; Adjusted means (A,B) in msec; d = A-B

Figure 7. Mean IBI in successive 2-sec bins for the 13.5 mA, 4.0 mA, 0.5 mA, and NP-4.0 mA groups, in session 10 of Stage 4, for the response to light (CS). Each data point represents the session mean IBI per bin, averaged over the three animals in each group. The first data point in each curve represents the mean IBI 2-sec pre-CS. Arrows indicate CS onset.

Fig. 7


MEAN INTERBEAT TIME (MSEC)
340
350
360
370
380
390
400
410
0.5 mA
13.5 mA
370
380
390
400
4.0 mA
NP-4.0 mA
PRE 2 4 6
PRE 2 4 6
SUCCESSIVE 2-SEC RECORDING PERIODS

4.0 and the 4.0 mA groups. The 0.5 mA group showed a slight increase in the magnitude of the acceleratory phase of the CR by the end of Stage 4. The changes in the deceleratory phase of the CR are less obvious in Figure 7. The 0.5, 4.0, and NP-4.0 mA groups showed either no change or a slight increase in the magnitude of the deceleratory phase, while the 13.5 mA group showed a substantial decrease in magnitude.

To assess the changes in magnitude during Stage 4 independent of shifts in pre-CS IBI levels the t-test for individual subject comparisons was used. Table 16 presents the results of the t-tests comparing the predicted response magnitudes of session 10 of Stage 2 (conditioning) with the obtained response magnitudes of session 10 of Stage 4 (extinction) for both phases of the CR. Seven of the nine animals in the 13.5, 4.0 and NP-4.0 mA groups showed a decrease in the magnitude of the acceleratory phase of the CR while the other two animals showed an increase in magnitude. All the animals in the 0.5 mA group showed an increase in the magnitude of the acceleratory phase of the CR. Nine of the twelve animals in all four groups showed an increase in the magnitude of the deceleratory phase of the CR while the other three animals showed a decrease in magnitude. These results confirm the findings reported for the CR in the comparison of Figures 4 and 7. In general, repeated presentations of CS-alone after conditioning reduced the magnitude of the acceleratory phase of the CR and increased the magnitude of the deceleratory phase.

Table 16

t-Tests For the Difference Between the Predicted Magnitudes of Stage 2, Session 10 ($Y'_{2,10}$) and the Obtained Magnitudes of Stage 4, Session 10 ($\bar{Y}_{4,10}$)

Response to Light (CR): Acceleratory Phase

Intensity (mA)	Subjects	$Y'_{2,10}-\bar{Y}_{4,10}$ (msec)
13.5	D-50	-45*
	D-90	-48*
	C-36	+40*
4.0	E-22	-12*
	E-16	-47*
	E-8	+11*
0.5	D-20	+ 3
	D-94	+21*
	D-92	+ 7*
NP-4.0	E-20	-29*
	E-10	-23*
	E-4	-20*

Response to Light (CR): Deceleratory Phase

Intensity (mA)	Subjects	$\bar{Y}_{4,10}-Y'_{2,10}$ (msec)
13.5	D-50	+26*
	D-90	+62*
	C-36	-61*
4.0	E-22	+ 7
	E-16	+57*
	E-8	- 6
0.5	D-20	- 6
	D-94	+10
	D-92	+18*
NP-4.0	E-20	+18
	E-10	+65*
	E-4	+26

* $p < .05$

Stage 5

Stage 5 consisted of another 10 sessions of shock-alone presentations. The primary purpose of this stage was to determine if the extinction sessions of Stage 4 would affect UCR when the UCS-alone presentations were re-introduced. To determine this the UCR in Stage 3 (UCS-alone session before extinction) was compared to the response to shock in the first session of Stage 5 (UCS-alone session after extinction). Figure 8 presents the group curves of mean IBI per bin for session 1 of Stage 5 for all four groups. Each data point represents the mean of three animals. Comparison of the curves in Figure 8 with the curves presented in Figure 6 (Stage 3) showed there was little change in the UCR for the 0.5 and 13.5 mA groups after extinction. The 4.0 and NP-4.0 mA groups showed a substantial reduction in the magnitude of the acceleratory phase of the UCR. However, the pre-UCS levels in session 1 of Stage 5 were considerably higher than in Stage 3 and could account for the reduction in UCR magnitude (i.e., an LIV effect). Therefore, the *t*-tests for individual comparisons assessed changes in the magnitude of the UCR after extinction independent of shifts in pre-UCS levels.

Table 17 presents the results of the *t*-tests comparing the predicted response magnitudes of Stage 3 and the obtained response magnitudes of session 1 of Stage 5 for both phases of the UCR. Ten of the twelve animals showed an increase in the magnitude of the acceleratory phase of the UCR and two animals showed a small decrease in magnitude. Ten animals also showed a decrease in the magnitude of the deceleratory

Figure 8. Mean IBI in successive 2-sec bins for the 13.5 mA, 4.0 mA, 0.5 mA, and NP-4.0 mA groups, in session 10 of Stage 5, for the response to shock (UCS). Each data point represents the session mean IBI per bin, averaged over the three animals in each group. The first data point in each curve represents the mean IBI 2-sec pre-UCS. Arrows indicate UCS onset.

Fig. 8

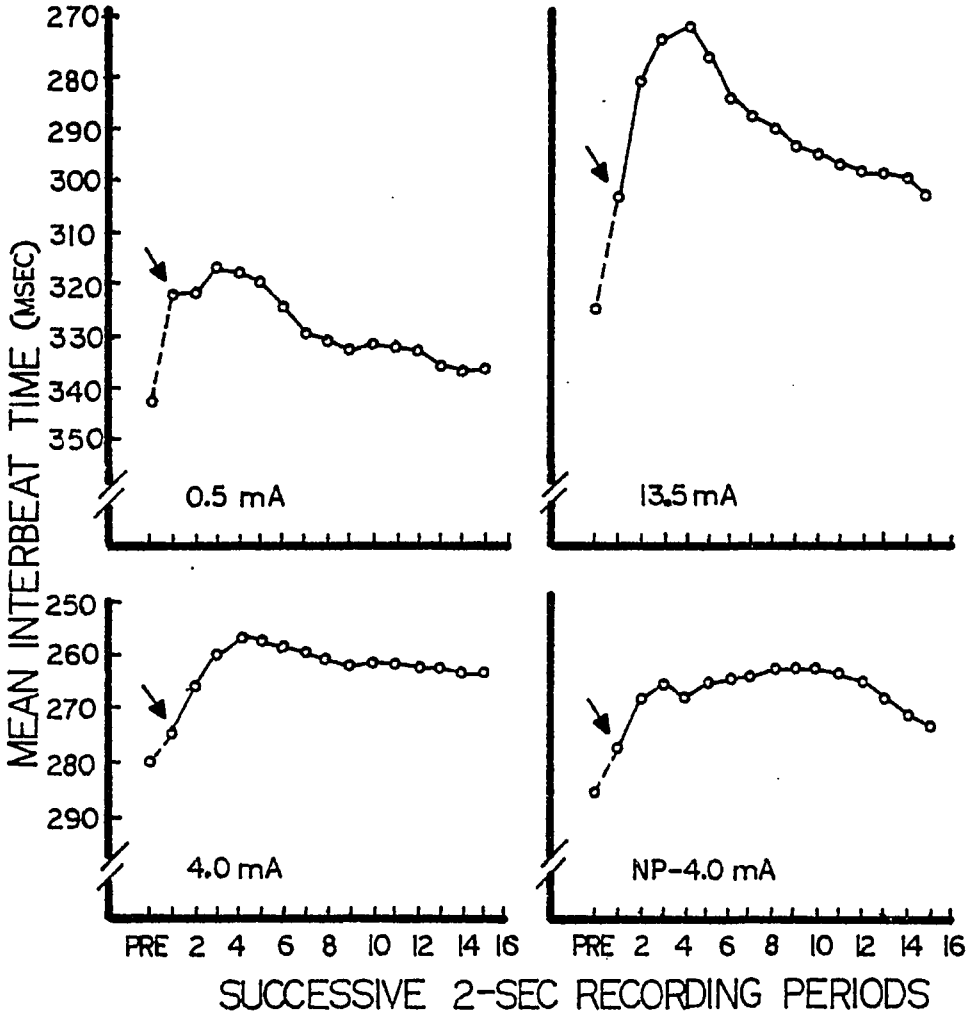

MEAN INTERBEAT TIME (MSEC)
270
280
290
300
310
320
330
340
350
0.5 mA
13.5 mA
250
260
270
280
290
4.0 mA
NP-4.0 mA
PRE 2 4 6 8 10 12 14 16
PRE 2 4 6 8 10 12 14 16
SUCCESSIVE 2-SEC RECORDING PERIODS

Table 17

t-Tests For the Difference Between the Predicted Magnitudes of Stage 3 (Y'_3) and the Obtained Magnitudes of Stage 5, Session 1 ($Y_{5,1}$)

Response to Shock (UCR): Acceleratory Phase

Intensity (mA)	Subjects	$Y'_3 - \bar{Y}_{5,1}$ (msec)
13.5	D-50	- 1
	D-90	- 8
	C-36	+ 8*
4.0	E-22	+35*
	E-16	+ 8
	E-8	+ 4
0.5	D-20	+20*
	D-94	+ 3
	D-92	+21*
NP-4.0	E-20	+39*
	E-10	+ 1
	E-4	+28*

Response to Shock (UCR): Deceleratory Phase

Intensity (mA)	Subjects	$\bar{Y}_{5,1} - Y'_3$ (msec)
13.5	D-50	- 6
	D-90	+28*
	C-36	-19
4.0	E-22	-62*
	E-16	-31
	E-8	-10
0.5	D-20	-57*
	D-94	+ 3
	D-92	-28
NP-4.0	E-20	-42*
	E-10	- 4
	E-4	-31*

* $p < .05$

phase of the UCR and the other two showed an increase in magnitude. In sum, comparing UCR magnitudes in the UCS-alone sessions before and after extinction showed an increase in the magnitude of the acceleratory phase and a decrease in the magnitude of the deceleratory phase.

A comparison was also made between Stage 3 and the last session of Stage 5 to determine if additional repetitions of shock would induce further changes in the magnitude of the UCR. Table 18 presents the results of the t-tests for the difference between the predicted magnitudes of the UCR in Stage 3 and the obtained UCR magnitudes of session 10 of Stage 5. Only the 4.0 mA group showed a further decrement in the magnitude of the acceleratory phase of the UCR as well as an increase in the magnitude of the deceleratory phase. The other three groups either showed no substantial change in UCR magnitude, as in the case of the 0.5 mA group, or an increase in the magnitude of the acceleratory phase and a decrease in the magnitude of the deceleratory phase, as in the case of the 13.5 and NP-4.0 mA groups.

Stage 6

Stage 6 consisted of 5 sessions of re-conditioning, i.e., further presentations of light-shock pairings. The intensity of the shock was reversed for the 0.5 and 13.5 mA groups: the original 0.5 mA group was re-conditioned at 13.5 mA; the original 13.5 mA group was re-conditioned at 0.5 mA. The notation R (for reversal) denotes the group when its shock intensity was reversed. Thus, R-0.5 mA denotes the group that originally received 0.5 mA and was switched

Table 18

t-Tests For the Difference Between the Predicted Magnitudes of Stage 3 (Y'_3) and the Obtained Magnitudes of Stage 5, Session 10 ($Y_{5,10}$)

Response to Shock (UCR): Acceleratory Phase

Intensity (mA)	Subjects	$Y'_3 - \bar{Y}_{5,10}$ (msec)
13.5	D-50	+ 7*
	D-90	-10
	C-36	+10*
4.0	E-22	- 4
	E-16	-47*
	E-8	-39*
0.5	D-20	- 2
	D-94	+ 1
	D-92	+ 3
NP-4.0	E-20	- 5
	E-10	+17*
	E-4	+47*

Response to Shock (UCR): Deceleratory Phase

Intensity (mA)	Subjects	$\bar{Y}_{5,10} - Y'_3$ (msec)
13.5	D-50	-33*
	D-90	+46*
	C-36	-27
4.0	E-22	+20*
	E-16	+68*
	E-8	+36
0.5	D-20	-22
	D-94	+11
	D-92	-12
NP-4.0	E-20	- 8
	E-10	+12
	E-4	-62*

* $p < .05$

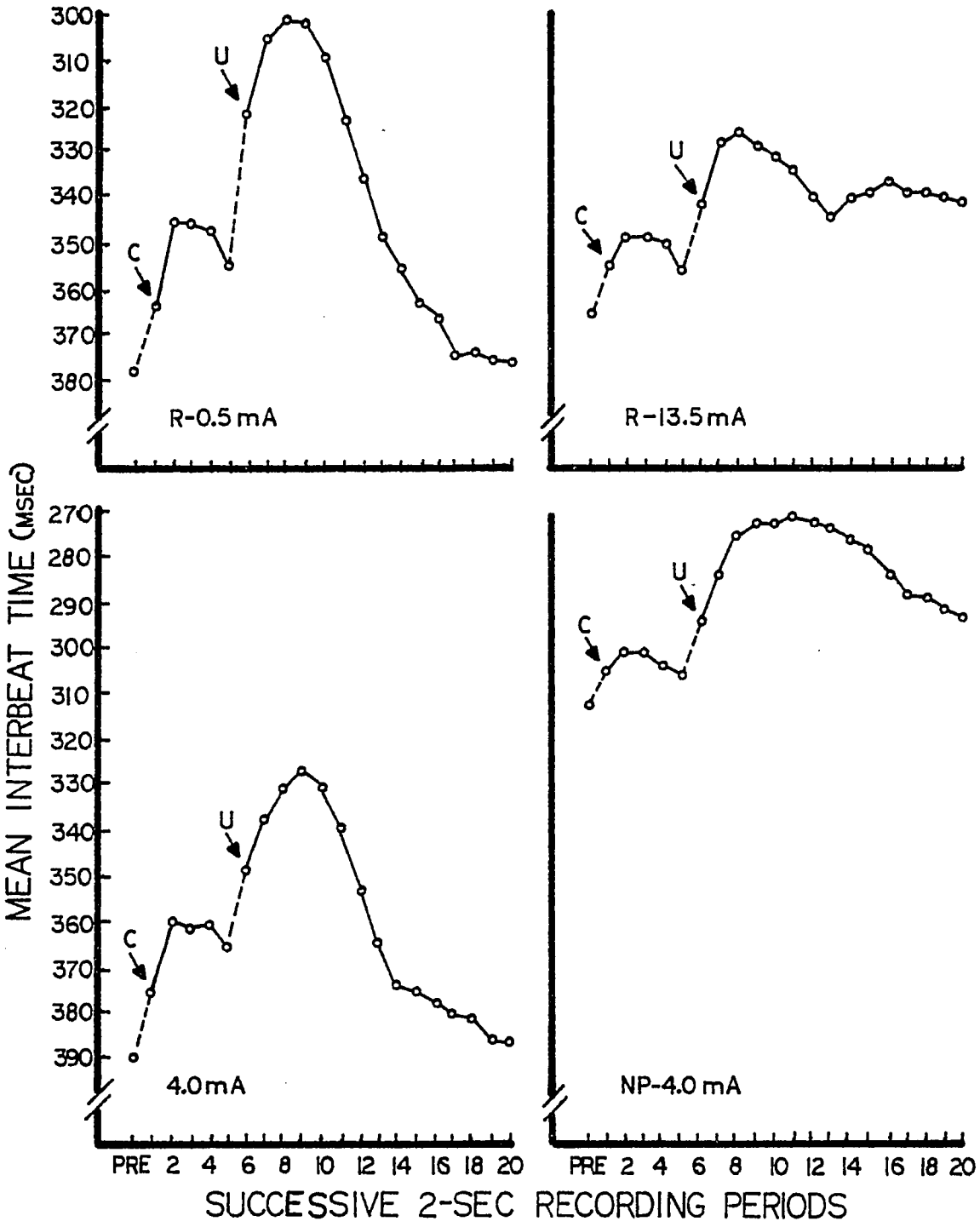
to 13.5 mA in Stage 6; R-13.5 mA denotes the group that originally received 13.5 mA and was switched to 0.5 mA in Stage 6.

Response to Light (CR). The analysis of the data in Stage 2 (conditioning)demonstrated that the magnitude of the response to light (CR) was positively related to the intensity of the shock. Since the magnitude of the response to light was a function of the intensity of shock during the initial conditioning sessions reversing the intensity of the shock might affect the magnitude of the CR. A comparison of the groups that had their shock intensities reversed with their initial conditioning sessions would indicate whether a shift in the intensity of shock affected the magnitude of the CR.

Figure 9 presents the group curves of mean IBI per bin for both the CR and UCR for session 5 of Stage 6. Each data point represents the mean of three animals. Comparisons of the CR in Figure 9 to the CR in Figure 4 (last session of conditioning) showed for the two groups in which the shock intensity was reversed substantial changes in CR magnitude. The R-0.5 mA group showed a large increase in the magnitude of both phases of the CR while the R-13.5 mA group showed a decrease in the magnitude of both phases. The CR for the 4.0 and NP-4.0 mA groups remained relatively unchanged.

These results established that when the shock intensity employed during acquisition was reversed there was a corresponding change in the magnitude of the response to light: when shock intensity was increased there was a corresponding

Figure 9. Mean IBI in successive 2-sec bins for the R-13.5 mA, 4.0 mA, R-0.5 mA, and NP-4.0 mA groups, in session 5 of Stage 6, for the response to light (CS) and the response to shock (UCS). Each data point represents the session mean IBI per bin, averaged over the three animals in each group. The first data point in each curve represents the mean IBI 2-sec pre-CS; the sixth data point represents the mean IBI 2-sec pre-UCS, and is also the last bin of CS. Arrows indicate CS-onset (C) and UCS onset (U).


MEAN INTERBEAT TIME (MSEC)
300
310
320
330
340
350
360
370
380
C
U
R-0.5 mA
R-13.5 mA
270
280
290
300
310
320
330
340
350
360
370
380
390
4.0 mA
NP-4.0 mA
PRE 2 4 6 8 10 12 14 16 18 20
SUCCESSIVE 2-SEC RECORDING PERIODS

increase in CR magnitude; when the shock intensity was decreased there was a corresponding decrease in CR magnitude. The results of the t-tests for comparing individual animals across sessions confirm this finding. Comparisons were made between the predicted CR magnitudes of session 10 of Stage 2 (conditioning) and the obtained CR magnitudes of session 5 of Stage 6 (reconditioning) for both phases of the CR. Table 19 presents the results of the t-tests. All the animals in the R-13.5 mA group showed a decrease in the magnitude of the acceleratory phase of the CR while all the animals in the R-0.5 mA group showed an increase in the magnitude of the acceleratory phase. None of the animals in the 4.0 mA group showed a substantial change in the magnitude of the acceleratory phase. However, the animals in the NP-4.0 mA group showed a decrease in the magnitude of the acceleratory phase.

Two animals in the R-13.5 mA group showed an increase in the magnitude of the deceleratory phase of the CR while the other animal showed a decrease in magnitude. All the animals in the R-0.5 mA group showed a decrease in the magnitude of the deceleratory phase. Again, all the animals in the 4.0 mA group showed small changes in the magnitude of the deceleratory phase, while all the animals in the NP-4.0 mA group showed a decrease in the magnitude of the deceleratory phase of the CR.

Table 19

t-Tests For the Difference Between the Predicted Magnitudes of Stage 2, Session 10 ($Y'_{2,10}$) and the Obtained Magnitudes of Stage 6, Session 5 ($\overline{Y}_{6,5}$)

Response to Light (CR): Acceleratory Phase

Intensity (mA)	Subjects	$Y'_{2,10}-\overline{Y}_{6,5}$ (msec)
R-13.5	D-50	-29*
	D-90	-52*
	C-36	-27*
4.0	E-22	+ 1
	E-16	- 2
	E-8	+10
R-0.5	D-20	+21*
	D-94	+32*
	D-92	+59*
NP-4.0	E-20	-11
	E-10	- 2
	E-4	-14*

Response to Light (CR): Deceleratory Phase

Intensity (mA)	Subjects	$\overline{Y}_{6,5}-Y'_{2,10}$ (msec)
R-13.5	D-50	+16
	D-90	+76*
	C-36	-47*
4.0	E-22	+ 6
	E-16	+ 4
	E-8	+ 1
R-0.5	D-20	-18*
	D-94	-31*
	D-92	-33*
NP-4.0	E-20	+15
	E-10	+ 7*
	E-4	+19*

* $p < .05$

Response to shock (UCR). Comparison of Figures 4 and 9 shows that for the R-13.5 mA group the magnitude of both phases of the UCR decreased, while for the R-0.5 mA group both phases of the UCR increased. The magnitude of both phases of the UCR also appears to have increased in the 4.0 and NP-4.0 mA groups. The results of the t-tests for individual subjects for the difference between the predicted UCR magnitudes in session 10 of Stage 2 (conditioning) and the obtained UCR magnitudes of session 5 of Stage 6 (re-conditioning) are presented in Table 20 for both phases of the UCR.

Table 20 shows that two animals in the R-13.5 mA group showed a decrease in the magnitude of the acceleratory phase of the UCR and a decrease in the magnitude of the deceleratory phase; the other animal showed a decrease in the acceleratory phase and an increase in the magnitude of the deceleratory phase. In the R-0.5 mA group two animals showed an increase in the magnitude of the acceleratory phase and the third showed a decrease in magnitude. Two animals showed a decrease in the magnitude of the deceleratory phase, while the third showed an increase in magnitude.

All the animals in the 4.0 mA group showed a decrease in the magnitude of the acceleratory phase of the UCR, and an increase in the magnitude of the deceleratory phase. The effects for the NP-4.0 mA group were in the opposite direction. All three animals in the NP-4.0 mA group showed a decrease in the magnitude of the acceleratory phase. Two animals showed

Table 20

t-Tests For the Difference Between the Predicted Magnitudes of Stage 2, Session 10 ($Y'_{2,10}$) and the Obtained Magnitudes of Stage 6, Session 5 ($\overline{Y}_{6,5}$)

Response to Shock (UCR): Acceleratory Phase

Intensity (mA)	Subjects	$Y'_{2,10}-\overline{Y}_{6,5}$ (msec)
R-13.5	D-50	-85*
	D-90	+30
	C-36	-21*
4.0	E-22	- 5
	E-16	-24*
	E-8	-80*
R-0.5	D-20	+40*
	D-94	- 8
	D-92	+ 7
NP-4.0	E-20	+13*
	E-10	+ 7*
	E-4	+10

Response to Shock (UCR): Deceleratory Phase

Intensity (mA)	Subjects	$\overline{Y}_{6,5}-Y'_{2,10}$ (msec)
R-13.5	D-50	+ 1
	D-90	- 6
	C-36	+28*
4.0	E-22	+34*
	E-16	+61*
	E-8	+59*
R-0.5	D-20	-18*
	D-94	- 2
	D-92	+31
NP-4.0	E-20	- 6
	E-10	-15*
	E-4	-19*

* $p < .05$

a small increase in the magnitude of the deceleratory phase and the third animal showed a slight increase in magnitude.

Stage 7

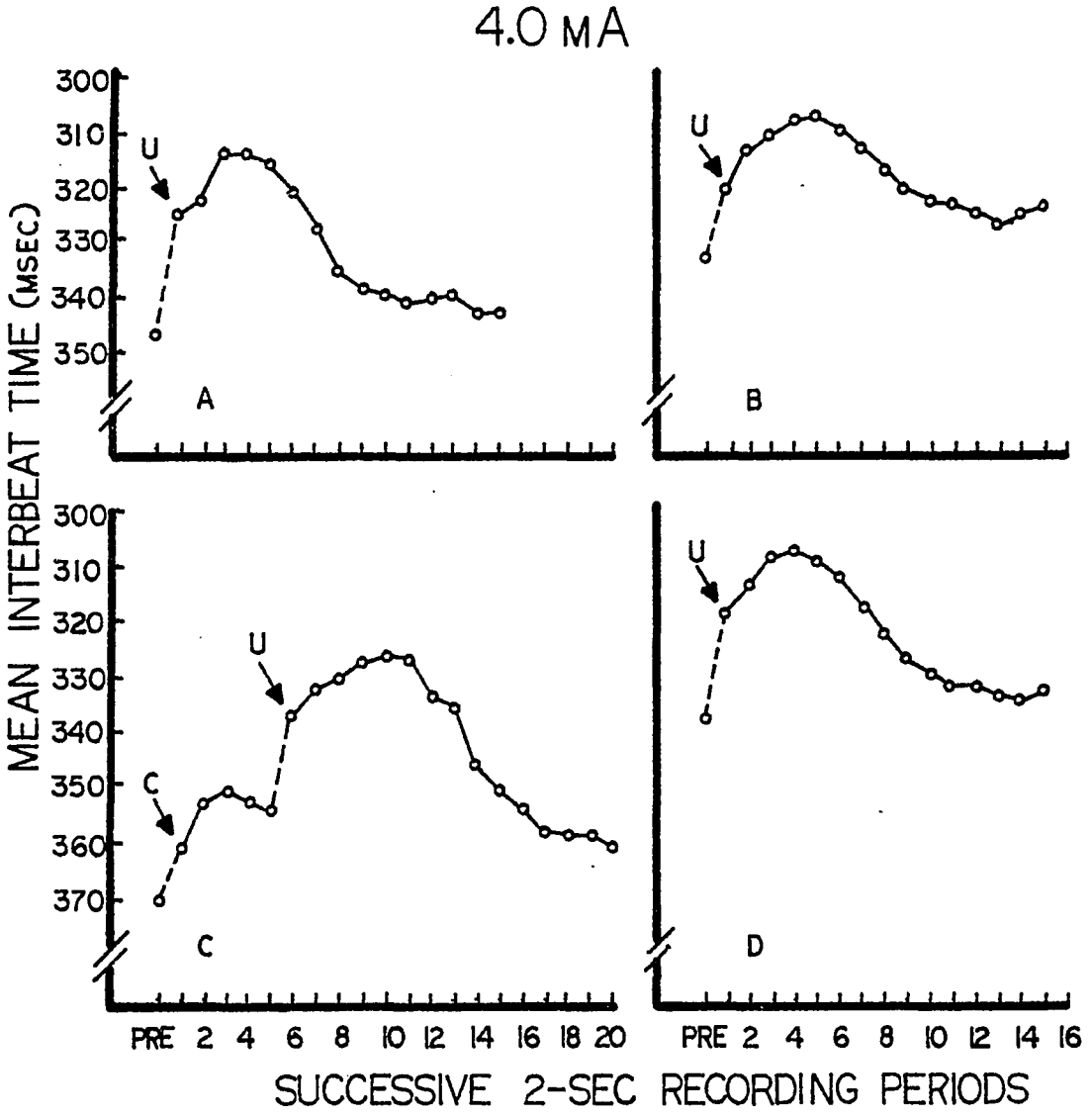
Many studies in the literature have employed smaller intertrial intervals (ITI) than that of the present study. To determine the effects of a smaller ITI on the UCR the 5.00 min mean ITI of the previous stages was reduced to 1.5 min and the first three stages were partially replicated using only the 4.0 and NP-4.0 mA groups.

Stage 7 consisted of two sessions (50 trials per session) of UCS-alone presentations. Figures 10A and 10B, and Figures 11A and 11B present the group curves of mean IBI per bin for sessions 1 and 2 for the 4.0 and NP-4.0 mA groups respectively. A comparison of the curves for session 1 (A) with session 2 (B) shows that by session 2 the magnitude of both phases of the UCR was reduced. However, direct comparison of the raw data was difficult, since the pre-UCS IBI levels were higher in session 2 for both groups. Therefore, the *t*-tests for individual subject comparisons were used to determine if there were changes in the magnitude of the UCR between the sessions.

Table 21 presents the results of the *t*-tests for the difference between the predicted UCR magnitudes of session 1 and the obtained response magnitudes of session 2 for both phases of the UCR. All six animals showed an increase in the magnitude of the acceleratory phase of the UCR. However, the increase was smaller for the 4.0 mA group then for the NP-4.0 mA group. In sum, when the ITI was reduced in Stage 7 the direction of the change in the magnitude of the UCR was

Figures 10-11. Mean IBI in successive 2-sec bins for the 4.0 mA (Figure 10) and NP-4.0 mA (Figure 11), in sessions 1 (panel A) and 2 (panel B) of Stage 7, of Stage 8 (panel C), and of Stage 9 (panel D), for the response to shock (UCS), and light (CS; only for Stage 8, panel C). Each data point represents the session mean IBI per bin, averaged over the three animals in each group. The first data point in panels A, B, and D represents the mean IBI 2-sec pre-UCS. The first data point in panel C represents the mean IBI 2-sec pre-CS; the sixth data point represents the mean IBI 2-sec pre-UCS, and is also the last bin in CS. Arrows indicate CS onset (C) and UCS onset (U).

Fig. 10


4.0 MA
MEAN INTERBEAT TIME (MSEC)
300
310
320
330
340
350
360
370
U
C
A
B
C
D
PRE 2 4 6 8 10 12 14 16 18 20
PRE 2 4 6 8 10 12 14 16
SUCCESSIVE 2-SEC RECORDING PERIODS

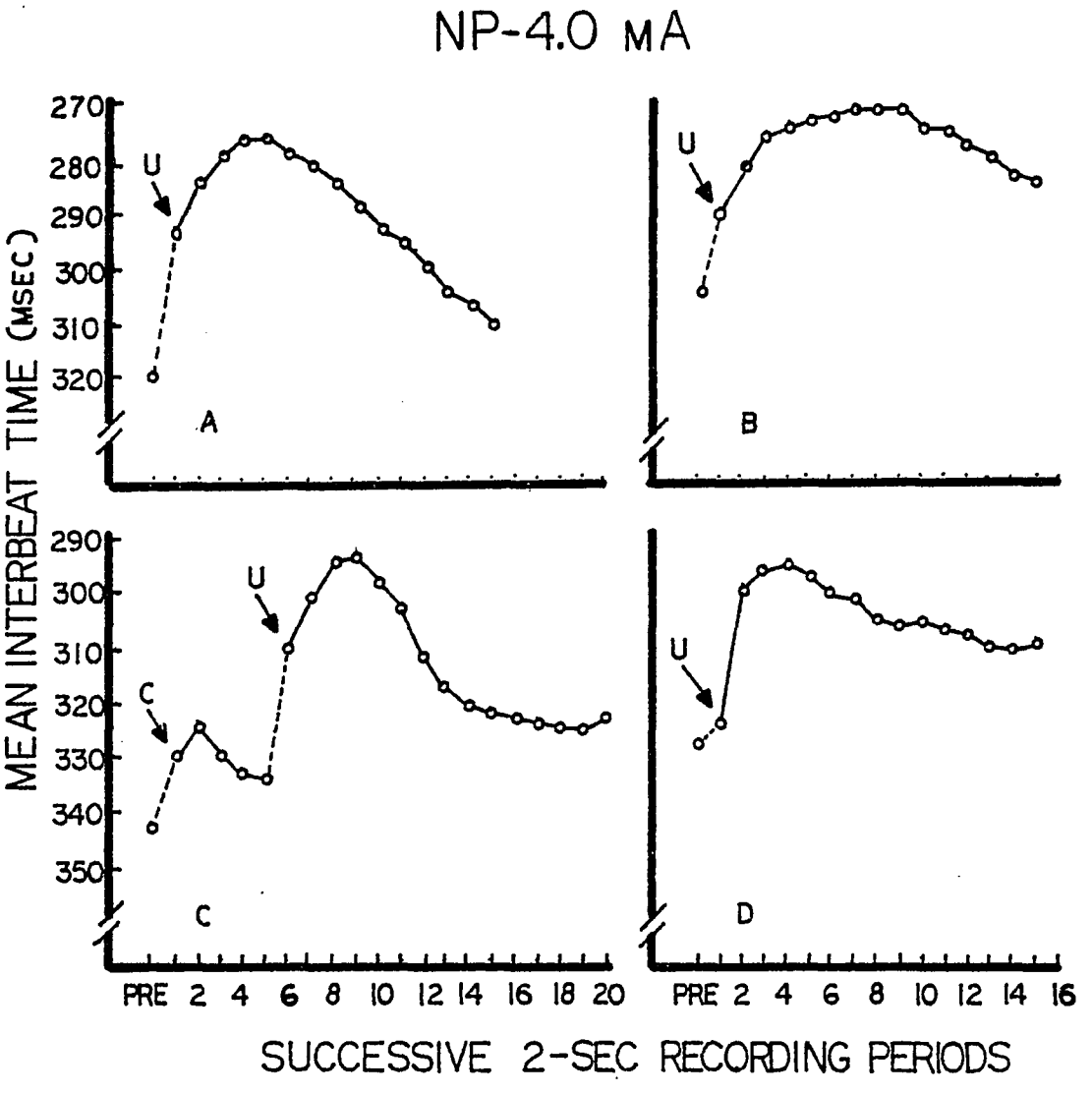

NP-4.0 mA
MEAN INTERBEAT TIME (MSEC)
270
280
290
300
310
320
290
300
310
320
330
340
350
U
C
A
B
C
D
PRE 2 4 6 8 10 12 14 16 18 20
PRE 2 4 6 8 10 12 14 16
SUCCESSIVE 2-SEC RECORDING PERIODS

Table 21

t-Tests For the Difference Between the Predicted Magnitudes of Stage 7, Session 1 ($Y'_{7,1}$) and the Obtained Magnitudes of Stage 7, Session 2 ($\bar{Y}_{7,2}$)

Response to Shock (UCR): Acceleratory Phase

Intensity (mA)	Subjects	$Y'_{7,1}-\bar{Y}_{7,2}$ (msec)
4.0	E-22	+ 7
	E-16	+ 1
	E-8	+ 3
NP-4.0	E-20	+ 8*
	E-10	+ 3*
	E-4	+24*

Response to Shock (UCR): Deceleratory Phase

Intensity (mA)	Subjects	$\bar{Y}_{7,2}-Y'_{7,1}$ (msec)
4.0	E-22	-25
	E-16	- 4
	E-8	- 1
NP-4.0	E-20	-11
	E-10	-13*
	E-4	-64*

* $p < .05$

opposite to that found in Stage 1. In Stage 7 the magnitude of the acceleratory phase increased, and the magnitude of the deceleratory phase decreased.

Stage 8

Stage 8 consisted of one session of conditioning (50 trials), with the ITI reduced to 1.5 min. Figures 10C and 11C present the group curves of mean IBI per bin for the 4.0 and NP-4.0 mA groups respectively. The data for both the CR and UCR are presented. Each data point represents the mean of three animals. As can be seen from these curves both groups show a well developed CR. Furthermore, the UCR appears larger than in session 2 of Stage 7.

The pre-UCS level was lower in Stage 8 than in Stage 7 thereby making direct comparison of the two curves in Figures 10 and 11 difficult. Table 22 presents the results of the *t*-tests comparing the difference between the predicted response magnitudes in session 2 of Stage 7 and the obtained response magnitudes of Stage 8 for both phases of the UCR. Five of the six animals showed a reduction in the magnitude of the acceleratory phase of the UCR and one animal showed a slight increase. Five animals also showed an increase in the magnitude of the deceleratory phase of the UCR and one animal showed a small decrease. The changes in the magnitude of the UCR found in Stage 8 were similar to those found in the initial stage of conditioning (Stage 2): in both Stages 2 and 8 there was a reduction in the magnitude of the acceleratory phase of the UCR, and an increase in the magnitude of the deceleratory phase. While in Stage 2 the direction of these changes was

Table 22

t-Tests For the Difference Between the Predicted Magnitudes of Stage 7, Session 2 ($Y'_{7,2}$) and the Obtained Magnitudes of Stage 8 ($\bar{Y}_8$)

Response to Shock (UCR): Acceleratory Phase

Intensity (mA)	Subjects	$Y'_{7,2}-\bar{Y}_8$ (msec)
4.0	E-22	- 8*
	E-16	- 5*
	E-8	+ 5
NP-4.0	E-20	-98*
	E-10	-37*
	E-4	- 6*

Response to Shock (UCR): Deceleratory Phase

Intensity (mA)	Subjects	$\bar{Y}_8-Y'_{7,2}$ (msec)
4.0	E-22	+19*
	E-16	+15*
	E-8	+ 1
NP-4.0	E-20	- 9
	E-10	+86*
	E-4	+ 4

* $p < .05$

similar to that of the UCS-alone presentations of Stage 1, in Stage 8 the direction of the changes in magnitude was opposite to that found in Stage 7 (UCS-alone).

Stage 9

Stage 9 consisted of one session of shock-alone presentation and is the procedural analogue of Stage 3. Figures 10D and 11D present the group curves of the mean IBI per bin for the response to shock in Stage 9 for the 4.0 and NP-4.0 mA groups respectively. Each data point represents the mean of three animals. Comparison of the UCR in Stages 8 (C) and 9 (D) showed no apparent change in UCR magnitude. The results of the statistical analysis confirmed this finding.

Table 23 presents the results of the t-tests comparing the difference between the predicted UCR magnitudes of Stage 8 and the obtained UCR magnitudes of Stage 9 for both phases of the UCR. Five of the six animals showed an increase in the magnitude of the acceleratory phase of the UCR, and a decrease in the magnitude of the deceleratory phase when UCS-alone was presented in Stage 9, although this effect was small; the other animal showed a decrease in the magnitude of the acceleratory phase of the UCR and an increase in the deceleratory phase. These findings were similar to those reported in Stage 3, when the CS was removed after the initial stage of conditioning: in both Stages 3 and 9 there was an increase in the magnitude of the acceleratory phase of the UCR and a decrease in the magnitude of the deceleratory phase.

Table 23

t-Tests For the Difference Between the Predicted Magnitudes of Stage 8 (Y'_8) and the Obtained Magnitudes of Stage 9 ($\overline{Y}_9$)

Response to Shock (UCR): Acceleratory Phase

Intensity (mA)	Subjects	$Y'_8-\overline{Y}_9$ (msec)
4.0	E-22	+63*
	E-16	+ 1
	E-8	+10
NP-4.0	E-20	+ 6
	E-10	+25
	E-4	-30*

Response to Shock (UCR): Deceleratory Phase

Intensity (mA	Subjects	$\overline{Y}_9-Y'_8$ (msec)
4.0	E-22	-19
	E-16	- 1
	E-8	- 1
NP-4.0	E-20	- 1
	E-10	-104*
	E-4	+ 63*

* $p < .05$

Discussion

Single Stimulus Presentations

In a sense, the first experimental stage represents the most elementary form of the "intruded stimulus paradigm" (Farmer and Schoenfeld, 1966; Kadden,Washton, McMillan and Schoenfeld, 1957; Schoenfeld, 1972) in that a single stimulus was "intruded" into the organism's "behavior stream," and the behavioral consequences of successive presentations of that stimulus were recorded. This stimulus schedule is the one most often used to study habituation. Contemporary theories of habituation have been greatly influenced by Sokolov's work on the orienting (OR) and defensive (DR) responses (Sokolov, 1963). Notably, Sokolov's schemata have been employed to account for changes in the cardiac rate response to successive stimulus presentations (Graham, 1973; Graham and Clifton, 1966; Jackson, 1974). Cardiac acceleration is considered a component of the DR, while cardiac deceleration a component of the OR.

The distinction between the OR and DR as response categories rests on the finding that various stimulus operations differentially affect the phases of the cardiac rate response. One major criterion for distinguishing between the OR and DR is that the former decreases with repetition of the stimulus, while the latter remains unchanged or increases in magnitude (Graham and Clifton, 1966). However, the results of Stage 1 do not provide experimental support for the distinction between the OR and DR.

Cardiac Acceleration. The results of Stage 1 showed

that the form of the UCR to shock was biphasic, consisting of an acceleration in heart-rate, followed by a deceleration. There appeared to be no changes in the magnitude of the acceleratory phase within either session 1 or 10 of Stage 1. However, a majority of animals showed a decrease in the magnitude of the acceleratory phase over sessions. This result is somewhat unusual in view of the findings from other laboratories. The magnitude of cardiac acceleration is generally reported to show either no change or increase with repeated stimulus presentations (e.g., Geer, 1964; Jackson and Graham, 1973; Keefe and Johnson, 1970; McDonald et al., 1964; Sokolov et al., 1961; Uno and Grings, 1965). On the basis of this finding, Graham has asserted that cardiac acceleration qualifies as a component of the DR (Graham, 1973; Graham and Clifton, 1966). In the present experiment the finding of a substantial decrement in the magnitude of the acceleratory phase over sessions demonstrates, however, that cardiac acceleration "habituates" with repeated stimulus presentations. Given "non-habituability" of the response as one main criterion for defining the DR (Sokolov, 1963), this finding does not support the conclusion that cardiac acceleration invariably represents a particular response class (i.e., the DR).

There were several variables that could account for the decrement in the acceleratory phase. For one thing, the magnitude of the acceleratory phase showed a decrease only across sessions, not within sessions. Lazarus and Opton (1966) similarly reported habituation of physiological

activity to frightening movies over days but not within sessions. Galbrecht et al., (1965), however, did not find habituation of cardiac acceleration within or across sessions. It is not clear whether differences in habituation within and across sessions are attributable to the delay between sessions, or to the total number of stimulus presentations. Studies on the habituation of the cardiac rate response are usually conducted within one session and incorporate no more than 20-30 stimulus presentations. The finding that the decrement in the acceleratory phase in Stage 1 only occurred between sessions may be due to the fact that there was a larger total number of stimulus presentations over sessions than in any single session. Unfortunately, there are no studies in the literature comparing stimulus presentations within a session with the same number of presentations apportioned over sessions. However, investigations of the effects of protracted stimulus presentations over trials or sessions report that cardiac accelerations do not habituate (Fehr and Stern, 1965; Galbrecht, et al., 1965; Roessler et al., 1969), in contradistinction to the present findings.

Another variable that could account for differences between within and across sessions habituation is the interstimulus interval (ISI). The mean ISI employed in Stage 1 (300 sec) was much longer than that of the typical habituation study (10-90 sec). When the mean ISI was reduced in Stage 7 (90 sec) the magnitude of the acceleratory phase did not change or increased over sessions, which is the finding generally reported in the literature (Graham, 1973). This result was

not entirely unexpected since the animals had an extensive history of shock by Stage 7 and the UCR might have already undergone the full course of its habituation. However, an additional decrement in the UCR was found in the subsequent Stage 8 demonstrating that UCR magnitude had not reached a lower limit in Stage 7. Nevertheless, since the animals had a complex experimental history by Stage 7 the following points regarding the effects of ISI are intended to be tentative. It is proposed that the longer ISI in Stage 1 may have facilitated the decrement in the magnitude of the acceleratory phase across sessions, while the shorter ISI attenuated the decrement in Stage 7. Gatchel (1975), for example, found that the heart-rate and skin conductance responses habituated more rapidly during training to a short ISI; after a delay habituation was greater for the group initially trained on the long ISI (cf., Davis, 1970). The comparatively long ISI in Stage 1 may account for the habituation of the acceleratory phase across but not within sessions.

While shortening the ISI attenuated intersession habituation in Stage 7 it did not facilitate intrasession habituation of the acceleratory phase of the UCR. The likelihood that the ISI in Stage 7 was sufficiently long to allow the response to recover to baseline levels may account for the failure to find intrasession decreases in UCR magnitude. There are studies demonstrating that a large part of the response decrement over single stimulus repetitions within sessions occurs when the ISI is shorter than response duration (Bloch, 1970; Coombs, 1938; Germana and Klein, 1968; Grings and

Schell, 1969a; Raskin et al., 1969). The few studies reporting within session decrements of cardiac accelerations have generally employed very short ISIs (10-15 sec), and frequently find that the response did not recover to baseline before the next stimulus presentation (Bloch, 1970; Dykman et al., 1959; Germana and Klein, 1968; Meyers, 1969; Raskin et al., 1969). If it is the case that when the ISI is larger than the duration of the response the decrement in response magnitude will be minimal, then the failure in the present experiment and other studies to find within session decreases in the magnitude of the acceleratory phase of the response may be partly attributable to ISIs longer than response duration (cf., Winokur et al., 1962).

The majority of animals in Stage 1 showed a decrease in pre-stimulus levels over sessions and confirms the results of several other studies (Cohen and MacDonald, 1971; Galbrecht et al., 1965; Ramsay, 1970). The results also demonstrate this change in baseline (pre-UCS) level indirectly affects UCR magnitude, i.e., LIV (Wilder, 1962). Without accounting for LIV changes in response magnitude with stimulus repetition may be exaggerated (cf., Germana, 1968). In the present experiment, LIV obscured changes in the response magnitude of the acceleratory phase due to the systematic effects of stimulus repetition on pre-shock levels. When difference scores were used as the measure of habituation subjects displaying a decrease in pre-UCS level, for example, often showed an increment in the magnitude of the acceleratory phase before pre-UCS levels were equated over sessions. However, equating pre-UCS levels

by the regression analysis revealed that the acceleratory phase decreased over sessions. Using absolute scores the amount of decrease in the acceleratory phase was often larger before adjustment for shifts in pre-shock levels than after. Thus, had a measure of response magnitude been used without adjustment for systematic changes in pre-shock level over sessions different conclusions would have been drawn as to the effect of stimulus repetition on response magnitude. Perhaps the lack of success of other investigators to detect habituation of cardiac acceleration is partly attributable to not accounting for LIV (Oken and Heath, 1963; Wilder, 1962).

There appeared to be no clear relationship between the intensity of UCS and the amount of decrement in the magnitude of the acceleratory phase of the cardiac rate response over sessions. Depending on the measure of response change, different relationships were obtained: For the absolute amount of change score (predicted minus obtained responses) the moderate and high stimulus intensity groups showed a larger decrement than the low intensity group, with the moderate intensity group showing the largest decrease in magnitude. However, the moderate and high intensity groups may have shown larger response decrements because their initial response magnitudes in session 1 were larger than the low intensity group. Since the amount of change score uses both initial and final responses magnitudes in its computation, differences in habituation among groups depend not only on the final level of the response but on the magnitude of the response to initial stimulus presentations as well. The form of the function relating shock intensity and percentage

change was similar to the absolute amount of change measure. Thus, it would appear that using the absolute amount of change and percentage measures of habituation, greater habituation occurs at higher intensities, although a somewhat larger decrement of UCR magnitude occured at the moderate intensity than the high intensity. However, the percentage measure encounters the same problem as the amount of change measures since it too incorporates initial response magnitudes into its calculation (cf., Graham, 1973). Another measure of habituation commonly employed is the final magnitude of the response at the end of a series of stimulus presentations. When the magnitude of the response in session 10 (smallest mean IBI post-shock) was used as the index of the effects of UCS presentations over sessions, it was found that the final UCR magnitude was positively related to stimulus intensity. On the basis of this measure one would conclude that habituation was greatest for the low intensity group, since it showed the smallest final response magnitude. However, there was a positive relationship between UCR magnitude in session 1 and stimulus intensity as well (cf., Berg and Graham, 1970; Jackson, 1974; Raskin et al., 1969; Roessler et al., 1969). Thus, the positive relationship between UCR magnitude and UCS intensity in session 10 does not indicate whether or not any change in UCR magnitude occurred over sessions, since using the final response level measure no comparison is made between initial and final response magnitudes(Graham, 1973; Jackson, 1974).

The problem of the relationship between initial response

magnitude and changes in response magnitude with successive stimulus repetitions is analogous to that of LIV, in the sense that the initial level of the response partly determines the amount of decrement. Therefore it is not surprising that authors employing different measures of habituation have reached conflicting conclusions as to the effects of stimulus intensity on habituation (e.g., Graham, 1973; Koepke and Pribram, 1966; Ratner, 1970; Thompson et al., 1973). It would appear that a method for equating initial response magnitudes among groups is required to assess the various measures of habituation before a definitive statement can be made concerning the habituation-intensity function.

In summary, the results of Stage 1 do not support the idea that cardiac acceleration represents a unique response class, i.e., the DR. One of the main criteria for identifying the DR is that it does not habituate with repeated presentations of the stimulus. However, this clearly was not the case as the acceleratory phase of the response decreased in magnitude over the sessions of Stage 1. Another major criterion for classifying the DR is its elicitation by high intensity stimuli. Again this criterion was not met since the low intensity shock group characteristically displayed a biphasic UCR, the first phase being cardiac acceleration.

Cardiac Deceleration. There was a period of deceleration occurring after the acceleratory phase of the UCR. The data showed that the magnitude of the deceleratory phase increased over sessions, indicating a more rapid return to baseline in session 10 than in session 1. This accounts for the fact

that the cardiac response appears more biphasic by session 10. The rate at which the deceleratory phase returned to baseline was partly determined by the magnitude and latency of the acceleratory phase: it was found that the smaller the magnitude of the acceleratory phase, and the shorter the latency, the more rapidly the deceleration recovered to baseline (pre-shock) levels.

These findings could account for the apparent effect of stimulus intensity on the rate of recovery to baseline. There was some indication within the 30-sec post-shock recording period that the higher the stimulus intensity the slower the rate of recovery to baseline. However, the magnitude of the acceleratory phase was also positively related to stimulus intensity and larger magnitude accelerations require a longer time to recover to baseline (c.f., Ginsberg and Thysell, 1966). Furthermore, shock intensity does not affect the final level attained by the deceleratory phase: on any given trial the UCR generally returned to or went below baseline (pre-UCS) levels for all groups.

Graham and Clifton (1966) have speculated that biphasic responses are actually a composite of the OR and DR. They contend that the DR (cardiac acceleration) is superimposed upon the OR (cardiac deceleration); decelerations that go below pre-stimulus levels supposedly reflect the final portion of the OR that would have been entirely present had the DR not been superimposed. However, the fact that the deceleratory phase goes below baseline is not sufficient reason to establish a separate response category. It was found that the degree

to which the deceleration goes below "baseline" is relative to pre-stimulus values: when the pre-stimulus level was high the response was more likely to go below pre-stimulus levels than when pre-stimulus level was low.

Graham and Clifton further argue that their view is supported by the finding that the deceleratory phase habituates rapidly (Berg and Graham, 1970; Jackson, 1974; Keefe and Johnson, 1970; Lang and Hnatiow, 1962; Uno and Grings, 1965), thus meeting one of the criteria for the OR (Sokolov, 1963). However, in the present experiment the deceleratory phase increased over sessions. Furthermore, if the acceleratory and deceleratory phases are different responses then changes in the latter should be relatively independent of changes in the former, which was not the case, as discussed earlier. Other theorists have advanced additional arguments questioning the necessity of establishing the decleratory phase as an independent entity in the biphasic response (Meyers, 1969; Roessler et al., 1969; Schoenfeld, 1976). The deceleratory phase of the cardiac rate response appears to simply reflect the heart's return to basal levels rather than constitute a separate response.

In conclusion, the classification of the OR and DR has as its basis a body of data suggesting that these two response are differentially affected by various stimulus operations. However, the findings of the present experiment and from other laboratories demonstrate that when a broader range of parametric stimulus operations is considered the criteria for the OR and DR are not substantiated (e.g., across sessions

decrements in the magnitude of the acceleratory phase). Furthermore, it was suggested that unless such factors as response duration, initial response magnitude, and systematic changes in basal heart-rate are considered different conclusions may be drawn concerning the effects of stimulus repetition and intensity on changes in the phases of the cardiac rate responses over trials and sessions.

Paired Stimulus Presentations

UCR Diminution. In addition to the decrease in response magnitude to single stimulus presentations, the literature has also considered the diminution of the UCR over successive conditioning trials. In accounting for UCR diminution during conditioning theorists have generally focused on CS as the determinant of the decrease in UCR magnitude (Badia and Defran, 1970; Grings and Schell, 1969a; Kimmel, 1966; Lykken and Tellegen, 1974; Martin et al., 1975). However, the extent to which this decrement may be habituation to UCS repetitions incorporated into the conditioning procedure itself has not been clearly established (cf., Fitzgerald, 1966; Kimble and Ost, 1961). The present experiment was designed to clarify this question, and to determine how several parameters of UCS affect the UCR decrement, thereby providing data which are lacking in the literature.

To assess the effects of CS intrusion on the UCR "baseline" established to UCS-alone presentations in Stage 1 a light (CS) was "intruded" before shock in Stage 2. For the 4.0, NP-4.0, and 13.5 mA groups the UCR decreased in magnitude over the course of successive conditioning sessions, a change in the same direction as that to repeated UCS-alone presentations. There are two possible logical explanations for this finding: a) the diminution of the UCR in Stage 2 was a function of continued UCS presentations, and independent of the pairing of CS and UCS, or b) the decrease in UCR magnitude was a function of the conditioning procedure itself. Several lines of evidence will be presented demonstrating that it was the

combined effect of repetitions of the CS and UCS that determined the UCR diminution during Stage 2, and that the effects of conditioning could be differentiated from the effects of UCS-alone presentations.

To begin with, in the 4.0 and 13.5 mA groups when the subjects were presented with an additional session of shock-alone (Stage 3) after conditioning the UCR magnitude increased. The removal of CS had the effect of reversing the direction of change in UCR magnitude found during conditioning, indicating that the decrease in UCR magnitude in Stage 2 was at least partly a result of CS-UCS pairings, and not simply an effect of repeated presentations of shock.

Additional evidence for the effect of CS-UCS pairings on UCR magnitude was provided by the finding that the positive relationship between UCS intensity and UCR magnitude in the first session of Stage 2 was no longer evident in the final session of conditioning (i.e., there were no among groups differences in UCR magnitude). Since UCR magnitude among the groups during conditioning converged to approximately equal values, while a positive relationship between UCR magnitude and UCS intensity was maintained over the UCS-alone presentations of Stage 1 even though UCR magnitude decreased, indicated that the UCR diminution during conditioning was not attributable only to further UCS repetitions.

The finding that UCR magnitudes among the groups at the end of conditioning were approximately equal also points to a positive relationship between UCS intensity and the amount of UCR diminution. The decreases in UCR magnitude in the moderate and high UCS intensity groups, in conjunction with the increase

in UCR magnitude in the low UCS intensity group, resulted in equal UCR magnitudes among the groups by the end of conditioning. The finding of a positive relationship between UCS intensity and UCR diminution stands in contrast to earlier findings of an inverse relationship between CS intensity and UCR diminution (Fishbein and Levy, 1966; Grings and Schell, 1969a).

The data provided by the low UCS intensity group also demonstrated that changes in the UCR during conditioning were not simply a result of UCS repetition. This group showed a decrease in UCR magnitude to UCS-alone presentations (Stage 1). If changes in the UCR magnitude during conditioning were solely due to further UCS repetitions it would be expected that UCR magnitude continue to diminish. However, contrary to expectation UCR magnitude increased over conditioning sessions. Thus, the intensity of UCS not only determined the amount of change in UCR magnitude during conditioning, but the direction of change as well: at low intensities repetition of the CS-UCS pair had a facilitative effect on UCR magnitude, while at higher intensities UCR magnitude was attenuated. Furthermore, it appears that CS-UCS pairings do not invariably result in a decrease in UCR magnitude; under certain circumstances UCR magnitude may increase during conditioning (c.f., Putnam, Ross and Graham, 1974), and in some instances not change at all (cf., Church et al., 1966; Grevert and Moore, 1970).

In sum, the data of the first three stages demonstrated that the degree of UCR diminution during conditioning was not simply a result of continued UCS repetitions. That is to say, UCR diminution was a function of both CS and UCS presentations.

However, the findings discussed do not necessarily support the view that the CS was the determinant of the decrease in UCR magnitude. In fact, there was additional data that showed that the amount of UCR diminution was smaller during conditioning than during UCS-alone presentations.

Comparison of the 4.0 mA and NP-4.0 mA groups provided a means to distinguish between the effects of UCS-alone presentations and CS-UCS pairings on the UCR. There were no differences between these two groups either in the magnitude of the UCR at the end of conditioning, or in the amount of decrement in UCR magnitude over conditioning sessions. However when the combined effects of UCS-alone and CS-UCS presentations on UCR magnitude for the 4.0 mA group were compared to the effect of conditioning-alone on UCR magnitude for the NP-4.0 mA group, the UCR showed a larger decrement to the combination than to just conditioning. Thus, the larger overall number of UCS repetitions received by the 4.0 mA group resulted in a greater decrease in UCR magnitude than the NP-4.0 mA group. The positive relationship between the number of UCS presentations before conditioning and the amount of UCR diminution extends earlier findings of a similar positive relationship when the total number of UCS presentations is manipulated during conditioning (Fitzgerald, 1966; Grevert and Moore, 1970).

To further assess the relative effects of repetition of the CS-UCS pair and UCS-alone on UCR magnitude the amount of change in the UCR for the 4.0 mA group during Stage 1 and for the NP-4.0 mA group during Stage 2 was compared. This comparison revealed that repetition of UCS-alone resulted in a larger UCR

decrement than did conditioning. This difference was not attributable to differences in the pre-UCS level (LIV) between the two groups.Several studies have reported findings which are opposite to those of the present experiment concerning the relative effects of conditioning and UCS-alone presentations (Furedy, 1970; Furedy and Klajner, 1972; Lykken et al.,1972). However, these studies did not take into account differences in pre-UCS level among groups which may account for discrepancies between the present data and these other studies (e.g., Lykken et al., 1972).

In sum, the data of the first two stages revealed that the UCR displayed a greater decrement to UCS-alone presentations than to repetitions of the CS-UCS pair. This would suggest that the modulation of UCS by CS was to attenuate the effects of UCS repetition during conditioning. In fact, in the case of the low intensity shock group UCR magnitude increased during conditioning, although it decreased to UCS-alone presentations.

Additional evidence for the differential effects of conditioning and UCS-alone presentations on UCR magnitude was provided by the data of the 4.0 mA groups in the final experimental stages. During Stage 7 (shock-alone presentations) UCR magnitude either remained unchanged or increased. Thus, even though shock-alone presentations no longer resulted in a diminution of UCR further CS-UCS pairings did produce an additional decrement. Therefore, the decrease in the magnitude of the UCR during Stage 8 cannot be simply attributed to the continuance of UCS presentations in conditioning and must have resulted from the repetition of the CS-UCS pair.

The results from the final stages also suggest that the intertrial interval (ITI) is an important determinant of the effects of conditioning on UCR magnitude. In the initial two stages (Stages 1 and 2) the UCR decreased during both UCS-alone and CS-UCS repetitions. However, when the ITI was reduced in the final stages UCR diminution occurred only during conditioning. While the prior history of shock for these animals make conclusions concerning ITI tentative, the duration of the ITI in the final stages may have attenuated the effects of shock-alone on UCR magnitude (see preceding section) but not the effects of CS-UCS repetitions.

The present experiment also provided information concerning some of the variables affecting the recovery of UCR magnitude to post-conditioning UCS-alone presentations. The re-emergence of the positive relationship between UCS intensity and UCR magnitude (which was no longer present at the end of conditioning) during Stage 3 (UCS-alone) indicated that the amount of UCR recovery was positively related to shock intensity. The high intensity group completely recovered to pre-conditioning levels, while the moderate intensity group only partially recovered. The results of the 0.5 mA group complicated this relationship. This group showed an increase in UCR magnitude during conditioning and a decrease in Stage 3 when CS was removed. However, the amount of recovery was smaller than the other two groups. That is to say, the relationship between UCS intensity and the amount of recovery maintained even though the direction of recovery differed for the low UCS intensity group and the other two groups. In sum,

whatever the direction of change in the UCR during conditioning once UCS-alone presentations were resumed the UCR recovered toward pre-conditioning levels, with the degree of recovery dependent, in part, on UCS intensity.

Comparison of the 4.0 mA and NP-4.0 mA groups revealed that the amount of UCR recovery is also a function of the total number of shock presentations. While there were no differences in UCR magnitude between these two groups during conditioning (Stage 2), differences emerged in Stage 3 (UCS-alone): the 4.0 mA group showed an increase in UCR magnitude when CS was removed while the NP-4.0 mA group showed relatively little change. Since the only difference between these two groups was their treatment in Stage 1 the differences between these two groups in Stage 3 was a function of their prior history of UCS-alone presentations. Thus, in addition to shock intensity, the total amount of shock also determines the extent of UCR recovery: the greater the overall number of shock presentations the greater the amount of recovery. While other studies have focused on the parameters of CS and their relationship to UCR recovery (Fishbein and Levy, 1968; Grings and Schell, 1969a;Kimmel, 1966), the data of the present experiment show that the parameters of UCS (e.g., UCS intensity, number of UCS presentations) also determine the extent and direction of UCR recovery.

The results of the present experiment show a clear differentiation between the effects of UCS-alone presentations and conditioning, and that the phenomenon of UCR diminution cannot be accounted for simply by the fact that the conditioning

procedure itself incorporates UCS presentations. However, neither the nature of the affect on UCR of intruding a CS before UCS, nor how this intrusion is related to UCS repetitions, is entirely clear. While the data of the early experimental stages indicated that CS attenuated the effects of UCS repetition, the later experimental stages provided data, albeit tentative, indicating CS facilitates UCR diminution. Furthermore, an account of the changes in UCR magnitude when CS is removed and UCS-alone presentations are reinstituted is necessary. Interpretations of UCR diminution have usually attributed the decrease in UCR magnitude to some "functional" property (e.g., "inhibitory," "preparatory") of CS (Badia and Defran, 1970; Furedy, 1968, 1970; Grings, 1965; Grings and Schell, 1969; Kimble and Ost, 1961; Kimmel, 1966; Lykken, 1962; Lykken and Tellegen, 1974; Rescorla, 1969).

The present experiment provides data particularly relevent to the "inhibition" hypothesis (Kimmel, 1966; Rescorla, 1969). Kimmel (1966) maintains that UCR diminution is determined by the "inhibitory" properties of CS (cf., Kimble and Ost., 1961; Rescorla, 1969). That the CS becomes an inhibitor, he argues, is not only indicated by the diminution of UCR itself but by "inhibition of delay", the tendency for CR to decrease in magnitude and increase in latency over extended exposures of the CS-UCS pair; the CS not only "inhibits" the UCR, but also the CR. However, there was no evidence of a decrease in CR magnitude in the present experiment; in fact, the CR increased in magnitude over conditioning sessions, while the UCR magnitude decreased (cf., Fishbein and Levy, 1968). In order to maintain

the notion of "inhibition" in this instance, it would be necessary to assume that the CS does not have an inhibitory effect on CR but only on UCR. However, if "inhibition of delay" is a necessary criterion for corroborating the "function" of CS as a "conditioned inhibitor," which Kimmel suggests it is, then the findings of the present experiment do not support this hypothesis (cf., Badia and Defran, 1970). Another difficulty for the "inhibition" hypothesis was the increase in UCR magnitude over conditioning sessions in the low shock intensity group. If the CS did acquire inhibitory properties with extended presentations of CS-UCS pairings, then it would have been expected that the UCR would decrease in magnitude in this group as well.

From the point of view of theory explanations of UCR diminution that impute a particular function to CS have proved to be inadequate. Schoenfeld et al. (1972) have pointed out that such "functions" of a stimulus are often inferred from the behavior itself and not measured directly, and therefore are tautological explanations. Furthermore they add:

> "Functions of a stimulus, whether 'discriminative' or 'reinforcing' or 'neutral' or whatever, may then be derived from schedule parameters. Together with the latter, the physical properties of a stimulus - its intensity, time course, mode or bodily locus of application, and so on - make up all the variables that underlie the control of behavior (Schoenfeld et al., 1972, p. 73)."

An alternative explanation of UCR diminution during conditioning may be provided by Schoenfeld's (1972) analysis of stimulus control. According to him stimulus control is defined by replicable changes in behavior (the response) with successive presentations of a stimulus. His conceptualization

proceeds from the understanding that behavior is continuous (the so-called "behavior stream") and is determined by continuous stimulus operations (defined by the physical properties of the stimulus and its temporal relationship to other stimuli). The extent to which a particular stimulus, S1, replaces some of the original determining conditions of the behavior stream (i.e., the stimulus conditions in which one initially finds the organism) determinesthe control of S1 over the response.

Furthermore, the extent to which the post-S1 response returns to its original state is determined by the physical parameters of S1 as well as the temporal relationship among successive repetitions of S1. Additionally, given the intrusion of a second stimulus, S2, the effects of that stimulus depend not only upon its physical properties and temporal relationship to S1, but on the current state of the "behavior stream" as well. As Schoenfeld (1972, p. 60) notes: "The less complete the return (of the response to its original state), the less the chance, after the next repetition of S2, of obtaining behavioral effects that are generically relatable (and therefore lawful)."

The implication of Schoenfeld's analysis for the phenomenon of UCR diminution lie in the relative control of CS and UCS on a particular response system, given that some of the properties of the post-CS response are generically relatable to the post-UCS response. Depending on the physical properties of CS, and its temporal relationship to UCS, the degree to which the CS controls the response in turn determines the degree of control by UCS. Schoenfeld (1972) suggests one way of manipulating the degree of control is to vary stimulus intensity. The finding of an inverse relationship between CS intensity and UCR diminution supports the

notion of a reciprocal relationship between the degree of stimulus control of CS and UCS (Fishbein and Levy, 1968; Grings and Schell, 1969a). In the current study, both the amount of UCR diminution and CR magnitude were positively related to UCS intensity, indicating that at higher UCS intensities stimulus control by CS increases and control by UCS decreases.

A second determinant of the modualation of UCS by CS, the rate of recovery of the CR to its original (baseline) state, is dependent, in part, on the temporal relationship between CS and UCS. If the rate of CR recovery is related to the degree of UCR diminution, it would be expected that the decrease in UCR be inversely related to the CS-UCS interval (Dufort and Kimble, 1958; Grings and Schell, 1969a; Kimble and Ost, 1961), since the longer the interval the more time allocated to the CR for recovery (cf., Block, 1970; Germana and Klein, 1968; Price and Geer, 1972; Raskin et al., 1969). The inverse relationship between the CS-UCS interval and UCR diminution may account for the increase in UCR magnitude in Stage 3 (UCS-alone). No CS may be tantamount to an ISI sufficiently long that CS no longer modulates UCS, i.e., removing CS may have similar effects to lengthening the CS-UCS interval. Turkkan (1977) provides data corroborating this suggestion (cf., Winokur et al., 1962).

The present experiment provides additional evidence that the amount and rate of CR recovery may determine the effect (control) of UCS, particularly if UCS occurs during the post-CS response perturbations. One factor isolated in the present experiment is the change in pre-UCS baseline level as a result

of CR acquisition, i.e., the effect of LIV on the UCR as a function of changes in pre-UCS level due to CR. The present data showed that there was an inverse relationship between the amount of CR acquisition and UCR diminution. Thus, the larger the magnitude of CR the higher the pre-UCS level and the smaller the UCR. This relationship was also a function of UCS intensity. The largest increases in pre-UCS levels (i.e., the largest CRs) as well as the greatest decreases in UCR magnitude were both positively related to UCS intensity. In accord with the foregoing considerations it was found that before shifts in pre-UCS level were taken into account the extent of the UCR diminution was larger than after pre-UCS levels were statistically equated over sessions. Thus, part of the UCR diminution was attributable to the indirect effect on UCR magnitude resulting from increases in pre-UCS level due to CR. The UCR diminution found after shifts in pre-UCS level were accounted for statistically could be attributed to stimulus repetition.

Another possibility for the effect of CR on the subsequent intrusion of UCS is the rate of change of the heart at the time UCS occurs. Generally, the question of LIV has been considered from the point of view of measuring the momentary rate of the heart for some small period of time prior to stimulation, and determining the functional relationship between these pre-stimulus values and the maximal post-stimulus change. However, the effect of a given stimulus may vary depending on whether the stimulus was intruded when the response system was relatively stable (i.e., steady state) or when the response

was undergoing a change (i.e., transition state). It was found, for example, that there was a positive relationship between the slope (rate of change) of the deceleratory phase of the CR and the magnitude of the UCR, and an inverse relationship between the slope and the latency of UCR, i.e., the faster the heart was decelerating at the time of UCS intrusion the smaller the magnitude of UCR and the longer the time necessary for it to reach peak amplitude. Support for this thesis is provided by electrocardiographic studies of the heart which indicate that "the electrical activity of the heart (and, indeed, its mechanical activity) takes some time to adjust to a change in frequency (Noble, 1975, p. 133)."

In conclusion, the results indicate that a differentiation can be made between the effects of UCS-alone repetitions and CS-UCS pairings on UCR magnitude. There appear to be two main sets of variables that can account for UCR diminution during conditioning. The first variable, stimulus repetition, appears to be primarily a result of the fact that the conditioning paradigm incorporates UCS presentations. This conclusion rests on three main findings. Firstly, acquisition of the CR was occurring with repetition of the CS-UCS pair, while only the UCR was decreasing in magnitude, i.e., only the response to UCS was "habituating", and not the response to CS. Secondly, the amount of UCR diminution is directly proportional to the total number of UCS presentations. Finally, UCS-alone presentations resulted in a larger decrease in UCR than CS-UCS repetitions. This indicates that the intrusion of CS before UCS attenuates the amount of diminution that occurs to UCS repetition. The second variable affecting UCR was the manner in which CS modified pre-UCS

response characteristics (e.g., pre-UCS mean IBI level; rate of the heart's deceleration pre-UCS) determiningthe effects of the subsequent intrusion of UCS, and thereby the amount of UCR diminution.

Relationship Between CR and UCR. Theoretical distinctions between the CR and UCR have been based on differences in temporal form and dynamic properties, such as magnitude and latency (vide, Schoenfeld, 1976). However, in the present study it was found that by the end of conditioning the temporal form of the CR and UCR was nearly identical in all animals. CR and UCR latencies were similar, as well. Of particular interest, though, was the finding in many animals that the CR magnitude was equal to, if not greater than, UCR magnitude. These findings were confirmed in Stage 6 (reconditioning) where the shock intensities for the high and low UCS intensity groups were reversed.

On the basis of the comparison of the CR and UCR it would appear that these two responses do not represent two different response classes (c.f., Dykman, 1967; Kimmel, 1973). The fact that the CR and UCR were similar at the end of conditioning (even though the CS and UCS were of different intensity and modality) was apparently due to the concurrent decrement of the UCR and acquisition of the CR. That other researchers have not found similarities in the UCR and CR may well be due to differences in the rates of CR acquisition and UCR diminution. For the animals that received prior presentations of shock the UCR partially habituated prior to conditioning, with further decreases in UCR magnitude occurring during conditioning. Thus, by the end of conditioning the UCR had sufficiently decreased in

magnitude for similarities between CR and UCR to emerge. In fact, it was found that the larger the decrease in the magnitude of the UCR over conditioning sessions the smaller the differences between the magnitudes of CR and UCR by the end of conditioning.

Extinction. The fourth experimental stage assessed the effects of CS-alone presentations (extinction) on the UCR to subsequent presentations of UCS-alone. Procedurally, "extinction" and "habituation" involve the same stimulus operations: repeated presentations of a single stimulus. They have been given different names despite their procedural similarities since they do not necessarily produce similar behavioral effects. Kimmel (1973) has argued, for example, that extinction of the CR represents a different process from habituation of the UCR (c.f., Galbrecht, et al., 1965).

If the behavioral effects of these two procedures are similar then the effects of repeated presentations of CS-alone might generalize to further presentations of shock and "drive" the UCR to lower levels. Schoenfeld (private communication) has suggested that such a demonstration would provide a basis for distinguishing between habituation and extinction as procedures insofar as it can be shown that a decrease in UCR magnitude can occur by means other than UCS repetition (i.e., prior presentations of CS). While a majority of the animals showed a decrease in CR magnitude to CS-alone presentations, UCR magnitude did not exhibit additional decrements when shock-alone presentations were resumed. In fact, most animals showed an increase in UCR magnitude (cf., Morrow, 1966).

There are two possible explanations for the failure to find a further decrement of UCR magnitude after extinction. Firstly,

the UCR diminution may have already reached a lower limit by Stage 3 thereby minimizing the possible effects of extinction on the UCR. There was some indication that this was the case: additional presentations of UCS over the sessions of Stage 5 generally did not produce further decrements in UCR magnitude. A second explanation may be in terms of generalization gradients. The extent to which a stimulus generalizes depends on its "similarity" to the training stimulus (c.f., Furedy,1968; Zimny and Miller, 1966; Bagshaw and Benzies, 1968; Weisbard and Graham, 1971). It may have been the case that the generalization gradient between the CS and UCS in the present experiment was to broad to detect possible intermodal generalization effects.

Summary

The present study explored changes in the cardiac rate response over repetitions of UCS-alone and CS-UCS pairings in the rhesus monkey at three different intensities of UCS (13.5, 4.0 and 0.5 mA) and two interstimulus intervals (300 and 90 sec).

Single stimulus presentations. The first experimental stage (UCS-alone presentations) elucidated several variables that determine the effects of UCS-alone repetitions on the cardiac rate response. A main finding was the decrease in the magnitude of acceleratory phase of the biphasic cardiac rate response over sessions. This finding stands in contrast to reports in the literature that cardiac accelerations do not habituate to single stimulus presentations. Several alternative explanations of this finding were provided. Firstly, an overall larger number of stimulus presentations was employed in comparison to other studies. Another explanation was indicated by the fact that the UCR decrement occurred only over sessions and not within sessions. It was suggested that the difference between within and across sessions habituation was a function of ISI: shorter ISIs attenuate intersession habituation, but facilitate intrasession habituation.

The decrease in UCR magnitude was also a function of LIV. Without considering LIV the magnitude of the acceleratory phase often appeared to increase over sessions. However, pre-UCS levels decreased over the sessions of Stage 1. When changes in pre-UCS levels were statistically equated over sessions

the magnitude of the acceleratory phase decreased with UCS repetitions.

The form of the habituation-intensity function depended on the measures of habituation. Absolute amount of change and percentage change scores showed a positive relationship between stimulus intensity and UCR habituation, while final response levels showed an inverse relationship. One difficulty noted in comparing habituation among groups receiving different stimulus intensities was the dependency of the amount of habituation on the magnitude of the response to initial stimulus presentations.

There was a deceleratory phase of the UCR subsequent to the acceleratory phase. The rate at which the deceleratory phase recovers to baseline levels was positively related to UCS intensity. This finding was interpreted as an indirect result of the effect of UCS intensity on the acceleratory phase: rate of recovery of the deceleratory phase was inversely related to the magnitude and latency of the acceleratory phase. However, there was no relationship between UCS intensity and the final level of the deceleratory phase: all groups showed recovery to or below baseline levels. Generally, the deceleratory phase went below baseline levels only when the pre-UCS level was high.

Paired stimulus presentations. Part of the present experiment was to determine whether or not UCR diminution found during conditioning was a function of the conditioning procedure itself, or a result of further UCS presentations during conditioning. Several lines of evidence showed that the conditioning procedure itself determined the UCR decrement. It was reported that the term habituation might be expanded to include the

effects of procedures other than single stimulus repetition. The literature on UCR diminution has generally focused on the effects of CS in producing the phenomenon, with little consideration for the effects of stimulus repetition or the parameters of UCS.

It was found that the positive relationship between UCR magnitude and UCS intensity evident at the beginning of conditioning was no longer present at the end of conditioning. This provided evidence that repetitions of the CS-UCS pair, and not simply the additional presentations of UCS, resulted in the UCR decrement during conditioning. Furthermore, the amount of UCR diminution over conditioning sessions was positively related to UCS intensity. UCS intensity also determined the direction of the change in the UCR during conditioning: at moderate and high UCS intensities UCR magnitude decreased, while at the low UCS intensity UCR magnitude increased. Since the low UCS intensity group showed a decrease in UCR to UCS-alone presentations, the fact that UCR magnitude increased during conditioning provided additional evidence for the differential effects of CS-UCS pairings and UCS-alone repetitions on UCR.

Comparison of the 4.0 and NP-4.0 mA groups also provided evidence for a distinction between UCS-alone and CS-UCS presentations on UCR. While there were no differences between these two groups during conditioning, when the combined effects of UCS-alone and conditioning for the 4.0 mA groups were compared to the effects of conditioning alone for the NP-4.0 mA group, the 4.0 mA group showed a larger overall decrease in UCR magnitude than the NP-4.0 mA group. This indicated that the

larger the overall number of UCS presentations the greater the decrement in UCR. It was also found that UCS-alone presentations produced a larger decrement in UCR magnitude than did CS-UCS repetitions; this difference could not be attributed to LIV.

Additional evidence for the differential effects of UCS-alone and CS-UCS was provided by the final experimental stages. In Stage 7 (UCS-alone; ITI shortened) UCR magnitude either remained unchanged or increased while in Stage 8 (conditioning) UCR magnitude decreased. If UCS repetitions alone produced the decrement in Stage 8, it would have been expected that UCR magnitude remain unchanged or increase as in Stage 7. While the prior stimulus history made conclusions concerning the ITI reduction in the final stages tentative, it was suggested that the shorter ITI attenuated the effects of UCS-alone but not the effects of CS-UCS presentations on UCR.

When the CS was removed in Stage 3, and UCS-alone was again presented, the magnitude of the UCR increased for the 4.0 and 13.5 mA groups while it decreased for the 0.5 mA group. This indicated that the changes in UCR magnitude during conditioning were not simply a function of UCS repetitions during conditioning. Furthermore, whatever the direction of change in UCR during conditioning after CS removal the UCR recovered to pre-conditioning levels. The degree of UCR recovery was positively related to UCS intensity. Additionally, the finding that the NP-4.0 mA group showed no change in UCR magnitude when CS was removed, while the 4.0 mA group showed

an increase in magnitude, demonstrated that the total amount of shock also determined the amount of UCR recovery, i.e., the greater the number of shock presentations, the greater the recovery.

UCR diminution was interpreted on the basis of Schoenfeld's (1972) analysis of stimulus control. It was suggested that the amound of UCR diminution was dependent on the relative control of CS and UCS on the cardiac response and the amount of recovery of CR to baseline at the time UCS was intruded. It was further suggested that LIV might provide one means to conceptualize how changes in pre-UCS level due to CR might affect responding to UCS. For example, the decrease in UCR magnitude during conditioning was greater before differences in pre-UCS level were equated over sessions by means of the regression analysis than after adjustment. Thus, UCR diminution was partly attributable to increasesin pre-UCS level due to the CR.

It was concluded that there were two main variables that could account for UCR diminution during conditioning. The first variable was UCS repetitions incorporated into the conditioning paradigm. This conclusion was based on three points: a) only the UCR decreased in magnitude during conditioning; the CR magnitude increased, b) the amount of UCR diminution was directly proportional to the total number of shock presentations, and c) CS-UCS pairings produced a smaller decrement in UCR than UCS-alone presentations. The second variable was the change in pre-UCS level attributable to the magnitude and the rate of change of the CR.

The present analysis provided an explanation of the classic finding that CR and UCR are of different temporal form, magnitude and latency. The form of the CR and UCR were almost identical, and the magnitude and latency were approximately equivalent, by the end of conditioning (Stage 2) and reconditioning (Stage 6, where UCS intensities were reversed in the high and low shock intensity groups). Furthermore, CR magnitude more closely approximated that of UCR as the latter decreased over conditioning sessions. This suggested that differences between CR and UCR may be attributable, in part, to differences in the rate of CR acquisition and UCR diminution. When CR is compared to the UCR at the end of its habituation, it is more likely the two responses will approximate each other in form, magnitude and latency.

The fourth (CS extinction) and fifth (UCS-alone) stages determined whether or not CS-alone presentations would effect a further decrement in UCR when UCS-alone presentations were resumed. Further decrements in UCR magnitude after exposure to CS-alone would elucidate another procedure resulting in a decrement in UCR, and would provide a basis for differentiating between extinction and habituation. The findings did not support this idea: UCR magnitude did not decrease after extinction. Two explanations were provided to account for this result: a) the UCR magnitude may have reached a lower limit by Stage 5, thus not allowing for the detection of the effect of extinction, and b) the generalization gradient between CS and UCS may have been too broad to show any effect of inter-

modal stimulus generalization.

In conclusion, while a number of different stimulus operations effecting a diminution in the UCR were considered, it was shown that these decrements could be accounted for by the functional relationships between the experimental manipulations and the response decrements without invoking hypothetical underlying states to account for the phenomenon. The general theoretical orientation was to consider where these phenomena lie in a broad range of parametric stimulus operations, rather than to consider the phenomena as isolated cases produced by unique "stimulus functions."

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