ELECTRODERMAL CORRELATES OF CONDITIONED SUPPRESSION

CHANGES IN SKIN CONDUCTANCE, SKIN POTENTIAL AND THE SUPPRESSION OF OPERANT BEHAVIOUR DURING CER CONDITIONING IN THE RESTRAINED RAT

by

REBECCA YOUNG, B.A.

A thesis

Submitted to the Faculty of Graduate Studies

in Partial Fulfilment of the Requirements

for the Degree

Master of Arts

McMaster University

(October, 1968)

MASTER OF ARTS (1968)
(Psychology)

McMASTER UNIVERSITY
Hamilton, Ontario

TITLE: Changes in skin conductance, skin potential and the suppression of operant behaviour during CER conditioning in the restrained rat.

AUTHOR: Rebecca Young, B.A. (D'Youville College)

SUPERVISOR: Dr. L.E. Roberts

NUMBER OF PAGES: iv, 45

SCOPE AND CONTENTS:

Skin conductance (SC), skin potential (SP), and lever pressing for a food reward were examined throughout discriminative CER training in restrained rats. Restraint was accomplished through use of a fixed electrode technique in which the animal was immobilized on a platform and electrodermal recording electrodes attached directly to the footpads, thus eliminating the artifacts inherent in grid techniques of recording electrodermal phenomena. Conditioned suppression of lever pressing was accompanied by an increase in SC amplitude and an increase in SP negativity. These results were attributed to peripheral and central response mechanisms regulating electrodermal activity.

ACKNOWLEDGEMENTS

I would like to thank Dr. L.E. Roberts for his continued support and encouragement throughout the preparation of this thesis.

I would also like to thank Mr. Mike Brolih for his aid in constructing the apparatus and Mrs. Marian Crow for typing the manuscript.

TABLE OF CONTENTS

Changes in skin conductance, skin potential and the suppression of operant behaviour during CER conditioning in the restrained rat

Rebecca Young

McMaster University

Introduction and Background

The electrodermal phenomena of the skin have traditionally been used as indices of emotional reactivity. Two electrodermal variables that have been widely studied by physiologists and psychologists are skin resistance (SR) and skin potential (SP). SR is defined as resistance of the skin to the passage of a small direct current between two liquid or metal electrodes (Grings, 1953); whereas SP is a potential between the electrodes that appears to be generated by negatively charged membranes near the surface of the skin and lining the sweat gland tubules (Martin and Venables, 1966). SR data are usually converted into skin conductance (SC) measures since it is believed that SC, the reciprocal of SR, is more linearly related to effector activity (Darrow, 1964; Montagu and Coles, 1966).

SC and SP are determined by (a) central brain mechanisms which regulate electrodermal activity, (b) peripheral effector mechanisms, and (c) characteristics of the recording system. Neurophysiological studies of the central control of electrodermal activity have shown that many areas of the brain regulate the SP response. For example, several excitatory and inhibitory centres for electrodermal activity have been identified by examining the effects of chronic lesions, stimulation, or

local anesthetization of various brain structures upon the amplitude of spontaneous and evoked SP responses (Wang, 1964). The major excitatory areas which have been discovered are located in the sensorimotor cortex, the limbic and infralimbic areas, the anterior hypothalamus, the lateral portion of the midbrain reticular formation, and the dorsal thalamus (Wang, 1964). Inhibitory areas discovered include the ventromedial reticular formation of the hindbrain, the frontal cortex, the anterior cerebellar lobe, the caudate nucleus, and the hippocampus (Wang, 1964). In addition to these excitatory and inhibitory centres, Wang (1964) has provided evidence for the existence of a regulatory sweat centre located in the striopallidum. He believes that this regulatory centre has connections with excitatory and inhibitory centres and functions as a complicated servo-loop mechanism, accelerating excitatory centre discharges while at the same time decelerating inhibitory centre discharges, or vice versa. Most of the work on the neurophysiological control of electrodermal activity has been done on cats having chronic lesions or implanted electrodes and only SP has been extensively studied in these experiments.

The major component of the peripheral effector for SC, the sweat glands, is innervated by motoneurons from the sympathetic division of the autonomic nervous system. It is believed that the sweat glands determine SC through two interacting processes, (a) duct-filling and subsequent hydration of the epidermis (Darrow, 1964; Lloyd, 1962) and (b) depolarization of sweat gland membranes, permitting freer movement of ions through membrane pores (Lykken, Miller, and Strahan, 1966; Wilcott, 1967; Darrow, 1964). There is also evidence for a non-sudorific contribution to SC (Edelberg, 1966; Wilcott, 1967). At the present, the most plausible hypothesis (Edelberg, 1966; Roberts, 1967c) is that the stratum

lucidum, an epidermal structure known to control long-term water diffusion across the skin, affects SC by regulating the diffusion of ions through the skin according to existing concentration gradients. While no firm conclusions regarding the non-sudorific structures involved have been reached, there is reason to believe that the contribution of non-sudorific influences to SC is smaller than that of the sweat glands (Roberts, 1967c).

The effector mechanism for SP has not been thoroughly investigated, but the fact that the SP response usually consists of a negative-going increase in potential that is almost invariably accompanied by an increase in SC indicates that SP responses are due to a polarization process that is highly correlated with, if not actually produced by, sudomotor activation (Wilcott, 1967). One possibility, suggested by Lykken (1966), is that changes in SP are produced by a mechanism for sodium reabsorption that reclaims this ion from sweat as it is secreted within the sweat gland tubule. Some evidence in support of this view has recently been provided by Fowles and Venables (1968), who found significant correlations ($r = .46$ to $.61$) between SP level and indices of sodium reabsorption obtained from salivary secretions.

When investigating electrodermal phenomena, the characteristics of the recording system used must be taken into consideration. In experiments with small animals, SC has been usually measured by the use of grid techniques in which a sub-threshold current is passed through a grid-floor on which the animal moves freely (e.g., Carran, Yeudall, and Royce, 1964; Kaplan, 1963b). While grid techniques have the advantage of allowing the animal freedom of movement, they are also subject to a number

of measurement artifacts which complicate interpretation of the data. For example, Roberts (1967b) and Deutsch (1967) have demonstrated that large increments in SC occur to either a conditioned or a natural fear stimulus. However, these increments in SC are also accompanied by freezing behaviour which could artifactually increase conductance in several ways. One possibility is that SC was increased through an increase in the number of grid bars contacted by the freezing animal. This increase in contact sites would have provided additional conduction paths in parallel with the current source and would, therefore, have increased SC. Another possible explanation of the SC change correlated with freezing is that the animal strengthened its grip on the grid bars, resulting in increased contact pressure and contact area, and, therefore, an increase in SC. Other artifacts associated with grid techniques which can increase SC, such as the presence of urine on the grid bars, are summarized by Roberts (1967a).

In view of the inadequacy of traditional grid techniques, investigators have attempted to devise improved grid methods of SC recording. One method recently tested by Woll (1968) employs a commutator which sweeps the grid bars approximately two times every second, connecting each bar in and out of the measuring circuit successively. Thus, at any instant in time only one grid bar is connected, and conductance measurements are taken only from the area of the skin in touch with this bar. This method effectively eliminates increments in SC due to an increase in number of grid bars contacted by S, since in this system the additional grid bars are electrically inactive. However, increased contact pressure and possibly urination artifacts are still left to confound the SC measures. Furthermore, Woll found this technique unsuitable for the recording of SP. A recording technique which

eliminates all of these artifacts is clearly called for.

Objectives of the Present Research

Several investigators have employed grid techniques of electrodermal recording to examine changes in SC which occur during the conditioned suppression of operant responding. In one of these studies, Anderson, Plant, and Paden (1967) first trained rats to approach a goal box to obtain food, and then examined the effect on running behaviour of the presentation of a buzzer that had previously been paired with shock. They found that rats which had been shocked in the presence of the buzzer showed suppression of the running response and had significantly higher SC levels than control rats for which the buzzer had not been paired. Roberts (1967b) examined electrodermal correlates of the suppression of lever-pressing behaviour during discriminative CER training in the mouse. He found that conditionined suppression was attended by a large increase in skin conductance, and that the SC response and the suppression of operant behaviour were conditioned, discriminated, and extinguished at approximately the same rate. These results were corroborated by Woll (1968) in a CER study using rats. Woll's study also provided evidence suggesting that an increase in the number of grid bars contacted by S occurs during suppression and contributes to the SC response. One interpretation of the relationship between SC activity and suppression demonstrated by these studies is that the increase in SC attending suppression is an artifact produced by an increase in the number of grid bars contacted by S, or an increase in contact area that occurs when S strengthens its grip upon the recording electrodes. An alternative explanation is that both responses are controlled by a central neural mechanism which regulates fearful behaviour.

A major objective of the present study was to demonstrate electrodermal correlates of conditioned suppression that could not be the result of measurement artifacts. This was accomplished by developing a technique for attaching electrodes directly to the feet of the animals, thereby eliminating contact area artifacts inherent in grid methods of electrodermal measurement. Recordings of SP as well as SC were taken because, although SP is regulated to an important extent by the same peripheral structures which influence SC, it is independent of contact area (Lykken, Miller, and Strahan, 1968). Therefore, if increments in SC attending suppression are due to variations in contact area even when fixed electrodes are used, parallel changes in SP would not be expected.

A second objective of the present study was to examine the relationship between conditioned changes in SP and SC, and also between electrodermal responses and suppression. This information was expected to provide valuable clues regarding the neural mechanism underlying electrodermal correlates of conditioned suppression.

METHOD

Subjects

The Ss were nine male hooded rats purchased from the Quebec Breeding Farms in St. Eustache, P.Q. They were three to five months old at the time of testing. The rats were reduced to 75% of their normal weight before the experiment started and were maintained at this weight throughout testing.

Apparatus

The Ss were restrained on a Lucite platform that contained openings for the hind legs and scrotum. Restraint was accomplished by taping S's tail to the platform and surrounding the rear limbs and back with a plastic collar that prevented chewing of the tape applied to the tail. S was able to move its shoulders, head, and forepaws freely. A lever, mounted on a metal panel located 50 mm. in front of the S, was made available to the forepaws. In the centre of the metal panel was a round white stimulus light. The reinforcement, liquid chocolate Metrecal, was dispensed through a 15 gauge hypodermic needle located vertically through a small hole in the platform directly in front of the rat's head. The administration and size of the reinforcements were controlled by a Davis LR-131 syringe pump.

The procedure for insertion was to first tape S's tail firmly to the platform. S's rear limbs were then drawn through the two hind-leg apertures and the plastic collar was attached. With the exception of two rats on the first day of bar-press training, the Ss were not etherized before being placed into the apparatus.

The active recording electrodes, made of pure zinc, had a small

rubber grommet taped to them. These grommets, which were filled with a unibase paste contained a .07 molar solution of sodium chloride (Miller, 1968) were used to eliminate variation in contact area and contact pressure. The active electrodes were taped to the hind feet of the rat, with the grommet carefully placed over the four bulbous inter-digital pads. A single reference electrode was used to record SC and SP. This electrode, a curved strip of pure zinc (length - 70 mm., width - 12 mm.), was placed over a section of the rat's tail that had been abraded with adhesive tape. The tails of all rats were abraded approximately every 4 or 5 days after the first day of preliminary training. SC was measured by a constant voltage circuit (.4v) inserted between the reference electrode on the tail and the active electrode attached to the right rear foot, while SP was recorded as the potential between the reference electrode and the active electrode attached to the left foot.

Shock was administered to the rat's tail through a pair of zinc strips 70 mm. long and 10 mm. wide. They were placed approximately 5 mm. apart and were always situated behind the reference electrode, about 7 cm. from it. Tail electrodes were held in place firmly by three rubber bands stretched over the back of the lucite collar and all made good contact with the sides and dorsal surface of the rat's tail. These electrodes were also coated with unibase paste and all electrodes were sanded with an emory cloth before being applied to S. The electrical stimulus was the discharge, through a 51 kilohm series resistor, of a 40 μfd capacitor that had been charged to 250 v. This arrangement provided a constant-current shock of approximately 3.2 ma.

Testing was carried out in a sound-proof chamber which was

electrically shielded with copper mesh. The chamber was dimly illuminated by a house light and also contained a fan for air circulation and temperature control. Auditory stimuli were fed into the chamber through a loudspeaker attached beneath and to the side of the restraining platform. SC and SP were recorded on a type RP Beckman oscillograph at a paper speed of 1 mm/sec. SP was recorded through an electrometer coupler to prevent degradation of the signal by variation in the resistance of the footpad or the footpad-electrode junction. Foringer programming equipment was located in a room adjacent to the experimental room.

Procedure

CER training was carried out in two phases, preliminary training and conditioning. In the preliminary training phase, Ss were adapted to the apparatus for five daily sessions of two hours each during which bar-press training was carried out. Ss were reinforced with liquid Metrecal on a VI-20 sec. schedule (range 3 to 35 sec.). Each reinforced bar-press was accompanied by a flash of the white stimulus light situated in front of the rat. This was done to make the delivery of a reinforcement more discriminable. The last two days of preliminary training were pretest days during which the two auditory stimuli used in conditioning were presented to the Ss four times each without shock. The two CSs consisted of a clicker (70 db.SPL, 33 pps) and a tone (65 db.SPL, 1667 Hz) that were equated for apparent intensity by E. CS presentations lasted three minutes each and were given in a mixed order with a variable intertrial interval that averaged eleven minutes (range = 8-14 min.).

Following preliminary training, discriminative CER conditioning was carried out for eight consecutive days. The procedure for conditioning was identical to that of the pretest days of preliminary training, except that a 3.2 ma. shock of 1 sec. duration overlapped the termination of one of the two auditory stimuli (CS+). Four Ss had the clicker as CS+, whereas the remainder received the tone as CS+. Four CS+ trials and four CS- trials were presented in a mixed order during the daily two-hour sessions, the order being reversed every other day. For example, CS- was presented first to each rat on conditioning day 1, the sequence of trials being CS-, CS+, CS+, CS-, CS+, CS-, CS-, CS+; while the reversed order on conditioning day 2 was CS+, CS-, CS-, CS+, CS-, CS+, CS+, CS-. A few exceptions in this reversed daily order occurred.

SP and SC were measured continuously throughout preliminary training and conditioning. Lever-pressing was also recorded continuously, using a Gerbrands cumulative recorder as well as a marking stylus on the Beckman oscillograph. In addition, the number of lever-presses occurring during each minute of the interstimulus interval, and also during each minute of a three minute control interval that immediately preceded CS onset, was recorded by a print-out counter.

Analysis of SC and SP

Measurements of SC and SP were taken every 20 seconds during the interstimulus interval, beginning 10 seconds after CS onset. Readings were also taken every 20 seconds during the three-minute control interval that immediately preceded CS onset. The difference between the mean of measurements taken during the pre-CS control interval and the mean of measurements taken during the interstimulus interval was designated ΔSC (or ΔSP). ΔSC and ΔSP were calculated to reveal the effect of CS

presentation on SC and SP.

Two further measures were calculated for SC only. The first of these, ΔSC^{10}, was the difference between the largest value of SC observed during the first 10 seconds of the interstimulus interval and the tonic level of SC observed immediately prior to response onset. The second measure, ΔSC^{ISI}, was the difference between the largest value of SC observed anywhere during the interstimulus interval and SC level immediately prior to the initial response to CS onset. Thus, the reference point for measurement of ΔSC^{10} and ΔSC^{ISI} was the same, i.e., the tonic level of SC observed prior to the initial deflection to onset of the CS. The latencies of ΔSC^{10} and ΔSC^{ISI} were also measured.

RESULTS

By the end of preliminary training all Ss were responding steadily on the VI-20 sec. schedule. Response rate on the second pretest day averaged 3,107 presses per hour.

The tonic levels of SC and SP throughout preliminary training and conditioning are presented in Fig. 1. The average maximum and minimum SC and SP values as well as the mean value of SC and SP observed during the pre-CS control interval have been plotted. In the case of SC a steady decrease in tonic level over days was clearly seen. Similar adaptation effects over preliminary training occurred with SP, but this trend reversed during conditioning. The gradual increase in negativity of SP seen during conditioning appeared to be due to an artifact arising from increased resistance at the reference electrode which occurred in the records of three rats, #40, #52, and #64. The most plausible explanation is that resistance at the reference site in these three rats increased toward the end of conditioning because the rats' tails had not been abraded often enough. As the ruptured epidermal membranes gradually repaired and resistance increased, the voltage dropped at the reference site by the conductance circuitry likewise increased and drove the reference electrode positive with respect to the footpad, resulting in the apparent increased negativity of SP.

Insert Fig. 1 here

The major observation of the study was that substantial changes in SC and SP accompanied the suppression of operant behaviour during CER conditioning. Fig. 2 presents a graph of the combined responses of all Ss during pretest and conditioning days. The lower section of the

figure shows what happened to bar-pressing behaviour throughout CS presentations. The suppression ratios shown here were calculated by dividing the number of responses during the three-minute CS-UCS interval by the total number of responses made during this interval plus the three-minute control interval that immediately preceded CS presentation (Annau and Kamin, 1961). A ratio of 0 thus indicates complete suppression of operant behaviour while a ratio of .50 indicates total absence of suppression of bar-pressing to the CS. It is evident that, by conditioning day 2, bar-pressing behaviour showed almost complete suppression to CS+, while pressing during CS- presentation was not disrupted.

Insert Fig. 2 here

Suppression of operant behaviour to CS+ was accompanied by increments in SC and increased negativity of SP, as shown in the upper sections of Fig. 2. Only uniphasic, negative-going SP responses were observed throughout the experiment. The changes in electrodermal activity shown here are ΔSC and ΔSP, i.e., the difference between electrodermal levels observed during CS presentation and those observed during the pre-CS control interval. It can be noted that there was a tendency for suppression and SC response amplitude to reach their highest level on conditioning day 4 and then decrease slightly from days 5 to 8. For SP, this tendency was discernible up to day 6 but was reversed on days 7 and 8, with a sharp increase in negativity occurring on day 8. This increase in response amplitude appeared to be the result of the artifactual increase in resistance at the reference site mentioned earlier,

since response amplitude was the largest for the three rats showing the highest tonic levels of SP.

When the data of individual animals were plotted separately, the response pattern shown in Fig. 2 was found to be characteristic of all nine Ss. Each S showed a clear discrimination between CS+ and CS-, with suppression and attending electrodermal changes first becoming evident on either the second or third day of conditioning. The impression received from an examination of Fig. 2 and the graphs of each S's data was that electrodermal responses and suppression were conditioned and discriminated at approximately the same rate. However, detailed statistical analyses revealed a tendency for the electrodermal variables to show a discrimination between CS+ and CS- somewhat earlier than suppression. The relevant findings are summarized in Table 1, which reports the outcome of t-tests performed on the amplitude of SC and SP responses and the suppression ratios observed on the two pretest days and the first three days of conditioning.

Insert Table 1 here

The manner in which this table is read can be illustrated by taking conditioning day 1 as an example. The positive values of t listed under column CS+ indicate that SC, SP, and the rate of bar-pressing increased when the CS was presented. However, none of these increments was statistically significant. The negative values of t listed under CS- indicate that conductance and potential decreased when the CS was presented, whereas the positive values of t for bar-pressing shows that operant behaviour was again facilitated rather than suppressed. Of these three, only the decrease in SC was significant ($p < .05$, two-tailed test). The

third column indicates that the difference between response amplitudes on positive and negative trials was significant for both SC and SP, but not for bar-pressing. Thus, while these results fail to show conditioning to CS+ on day 1, they do provide some evidence of discrimination between CS+ and CS- by SC and SP. However, it appears that the discrimination based on the electrodermal variables was due more to a decrease in electrodermal levels during CS- presentation (negative t values under CS- column) than to an increase in SC and SP during CS+ presentation. One possible explanation of these findings for conditioning day 1 is that conductance and potential are more sensitive indices of conditioning than is the suppression of operant behaviour, and that the early form of the electrodermal CR consists of an arrest of a tendency for SP and SC to adapt to lower levels during the intertrial interval. While this view might have some validity, a more likely explanation is that the discrimination between CS+ and CS- evidenced by the electrodermal variables was simply an artifact of the sequence in which the positive and negative trials were delivered.

An account of this artifact is as follows. Inspection of the oscillographic records showed that the tendency for SC and SP to adapt to lower levels was most pronounced immediately after insertion into the apparatus, and also following shock on CS+ trials. On conditioning day 1, CS- trials occurred during these periods of adaptation; in particular, all Ss received CS- first, and a CS- trial followed a CS+ trial on two occasions. Therefore, it appears that an accidental confounding of trial sequence with adaptation effects produced decrements in SC and to a lesser extent in SP during CS- which in turn led to an apparent discrimination between positive and negative trials.

The interpretation of other findings reported in Table 1 is relatively straightforward. There was a significant increment in potential to the CSs on the first pretest day of preliminary training, but these responses disappeared during the second pretest day. Conductance responses also occurred on the first and second pretest days, but were not significant. There was a tendency for bar-pressing to accelerate during trials on both pretest days of preliminary training, but this acceleration was not significant and was opposite to the direction of the responses seen during conditioning, where suppression of operant behaviour was invariably displayed. The results for the second and third days of conditioning show a significant increase in SC and SP and a significant decrease in rate of bar-pressing during CS+. It is evident that CS- had little effect, and that all three response measures showed a clear discrimination between positive and negative trials. The pattern of findings shown on conditioning days 2 and 3 persisted for the remainder of the experiment.

Fig. 3 shows the form of the electrodermal and bar-pressing responses during positive and negative trials averaged over the fourth, sixth, and eighth days of CER conditioning. Data for these three days only were combined because, except for a tendency for response amplitude and suppression to diminish, the characteristics of the responses did not change beyond the third day on conditioning. The arrows in Fig. 3 indicate the start and end of CS presentations. It is evident that SC and SP increased steadily throughout the presentation of CS+ and that bar-pressing was almost completely abolished. In contrast, CS- had no effect on SC, SP, or the rate of operant responding. While bar-pressing recovered almost immediately after CS+ offset, both SP and particularly SC showed

more gradual returns to their original levels, with peak responses occurring at the time of shock. This wave-form reflects a genuine steady increase in electrodermal activity and is not just an averaging artifact, since individual Ss also showed this steady increase throughout CS+ presentation. For example, the oscillographic record in Fig. 4 shows responses during a typical CS+ trial. At CS+ onset bar-pressing immediately stopped, SC increased in amplitude and SP increased in negativity throughout the trial until shock, after which pressing resumed and the electrodermal responses started to taper off to lower levels.

Insert Figs. 3 and 4 here

The results presented in Figs. 2 through 4 and in Table 1 suggest a high degree of association among electrodermal responses and suppression. This association was studied further through a trial-by-trial examination of the oscillographic records of each S to determine whether there were instances in which (a) the tonic level of SC increased in the absence of a change in SP, or vice versa, (b) suppression occurred without attending changes in SC and SP, and (c) the tonic level of electrodermal activity changed without clear evidence of suppression. Because electrodermal changes and suppression were very small or non-existent on the first day of conditioning, and also because responding to CS- was never very clear-cut, only CS+ trials from conditioning days 2 through 8 were examined. The following results do not include the data from one rat, #50, whose performance will be discussed shortly. For the remaining 8 Ss, a total of 224 CS+ trials was examined. On none of these trials was there an instance of a clear dissociation between electrodermal responses, i.e. a tonic change in conductance was also accompanied by a change in potential

and vice versa, with the time course of the two electrodermal responses being highly synchronized. Nor were any instances found in which suppression was evident without parallel changes in conductance and potential. However, there were three possible occasions on which electrodermal responses may have occurred in the absence of suppression. These occurred on days 7 and 8 of conditioning and involved three different rats, #52, 56, and 65. The suppression ratios for rats #52 and #56 on these trials were .41 and .46 respectively, showing a tendency towards suppression that made firm judgments of response dissociation difficult to render. The remaining S, #65, showed clear-cut electrodermal changes with a suppression ratio of .49, but in this instance the large suppression ratio was due more to the absence of responding in the pre-CS period than to the absence of responding during the interstimulus interval. The clearest impression received from this analysis was that conductance and potential responses and suppression appeared together rather than singly, with the suppression of operant responding being associated with an increment in SC and increased negativity of SP.

In contrast to the other animals, rat #50 showed a clear dissociation between suppression and electrodermal responses on the first two CS+ trials of conditioning day 2. Suppression was clearly evident on these two trials (suppression ratio = .06 and .09), but no change in potential or conductance was discernible. The remaining two CS+ trials on this day showed the complete response pattern, i.e., clear suppression with unambiguous increments in the tonic level of SC and SP. On subsequent days of conditioning the entire response pattern was evident, but in many cases the amplitude of the conductance and potential responses was very small. A number of facts indicate that a defective electrodermal

effector was the cause of the dissociations observed between suppression and electrodermal responses for rat #50. The most important observations are that this S displayed extremely low tonic levels of conductance and potential, and that spontaneous electrodermal activity was almost entirely absent throughout preliminary training and conditioning. For example, on the day on which suppression and electrodermal responses were dissociated (c-2), the tonic level of conductance at the beginning of the session was .2 micromho (skin resistance = five million ohms), while the tonic level of skin potential was 14 mv. positive. There was no evidence of electrodermal activity of any kind until the second CS+ trial, when an increment in conductance and potential level in response to shock was observed. Following this trial the tonic level of electrodermal activity increased, with the appearance of spontaneous electrodermal responses during the intertrial interval. Thus, for this animal the appearance of electrodermal responses to CS+ on the second day of conditioning was preceded by an increase in the tonic level of conductance and potential, the appearance of unconditioned responses to shock, and also the appearance of spontaneous phasic fluctuations in SC and SP during the intertrial interval. These findings, taken together with the fact that in other respects rat #50 was indistinguishable from the remaining Ss in the experiment, strongly suggest that the dissociations between suppression and electrodermal activity on conditioning day 2 were caused by a defect in the peripheral effector for conductance and potential. The nature of the peripheral defect is difficult to specify, but, whatever the cause, it is clear that conductance and potential were affected equally.

The fact that SC and SP responses were so highly synchronized might lead one to suspect that changes in SP were merely artifacts produced

by fluctuations in potential at the reference site that followed from increased SC. However, the evidence against this view is conclusive. In the first place, the largest change in potential at the reference electrode produced by an SC response was very small, about 400 μv, whereas the SP response to CS+ averaged about 12 mv. beyond the third day of conditioning. Secondly, the SC and SP waveforms were not completely identical. For example, although the use of a slow chart speed (1mm/sec.) made a detailed analysis impractical, the latency of the SP response appeared to be consistently shorter than the latency of the SC response. Also, the form of the two responses often differed. As can be seen in Fig. 4, the initial SP response consisted of a sudden increase in negativity followed by a decrease, whereas the SC response was unidirectional and consisted of a sustained, gradual increase in tonic level. Finally, SP responses were still evident when the conductance circuitry was switched off. Therefore, the apparent synchronization of SC and SP responses was not the result of biasing of the reference electrode by the conductance circuitry, but rather, was a reflection of synchronized activity of electrodermal effectors in the two footpads.

The latency and amplitude of the largest SC response occurring within the first ten seconds of CS presentation (ΔSC^{10}) and also of the largest SC response occurring anywhere within the three-minute interstimulus interval (ΔSC^{ISI}) were calculated. The results of this analysis are presented here for the sake of completeness and for any interest which they may hold, although they will not be discussed further. Zero-response

---

Insert Figs. 5 and 6 here

---

trials were excluded from the amplitude and latency measurements. As seen

in the upper section of Fig. 5, the latency of ΔSC^{10} showed no clear-cut effects, except perhaps for a tendency, starting at conditioning day 6, for the latency of CS+ responses to shorten and the latency of CS- responses to lengthen. Amplitude of ΔSC^{10} responses (Fig. 5, lower section) to CS+ and CS- showed a clear-cut separation throughout pretesting and conditioning, CS+ responses being greater than CS- responses. There was also a tendency for the separation between CS+ and CS- to increase during the initial stages of conditioning. Past conditioning day 3, a steady decline in responses to both stimuli occurred. Fig. 6 shows the latency and amplitude of the largest interstimulus interval response, ΔSC^{ISI}. The latency of ΔSC^{ISI}, illustrated in the upper section of Fig. 6, showed a steady increase over conditioning on both CS+ and CS- trials (cf. Kimmel, 1964). This indicates that the peak response within a CS presentation tended to occur closer to the termination of the CS with the passage of conditioning days. The findings regarding response amplitude are shown in the lower half of Fig. 6. Response amplitude to CS+ increased up to conditioning day 4, after which it dropped off, while response amplitude to CS- showed a steady decline over conditioning days. Thus, there was again a tendency for the separation between the CS+ and CS- responses to increase over conditioning.

Insert Fig. 7 here

Since there were a number of trials in which SC and SP responses were not observed within the first ten seconds of CS presentation, measures of response frequency on ΔSC^{10} were also taken. Furthermore, in order to determine whether the frequency of responding to CS- was greater than the spontaneous rate of responding, the occurrence of spontaneous responses during a ten-second control interval ninety seconds before CS-

onset was tabulated. Fig. 7 shows the results of these analyses. It can be seen that the frequency of ΔSC^{10} responding to CS+ increased over days while frequency of responding to CS- decreased. However, the frequency of CS- responding was still greater than the frequency of spontaneous responses, although these responses followed the same course over conditioning days.

DISCUSSION

Role of Measurement Artifacts

In traditional grid methods of electrodermal recording, interpretation of SC results is usually confounded by the presence of a number of measurement artifacts. It was noted earlier that previous demonstrations of an increase in SC during conditioned suppression may have been due to S's touching additional grid bars or gripping them more firmly, both of which would have increased contact area and therefore SC. In the present experiment a fixed electrode technique was used to eliminate these artifacts. Therefore, (a) S did not have the opportunity to touch additional contact sites when freezing because the recording electrodes were attached directly to the footpads, and (b) variations in contact area and contact pressure did not occur since the footpads were separated from the recording electrodes by a rubber grommet, eliminating the possibility of S grasping the electrode more tightly during CS presentation. The fact that conditioned suppression was accompanied by increased negativity of SP also argues against artifactual interpretations of the SC response, since SP is highly correlated with SC but is independent of contact area (Lykken et al, 1968; Martin and Venables, 1966). The performance of rat #50, which was discussed above, also helps to rule out an artifactual interpretation of electrodermal changes accompanying conditioned suppression. If increases in contact area and contact pressure accounted for the electrodermal correlates of suppression, one would always expect to see changes in SC and SP along with suppression. However, since #50 displayed a response pattern of good suppression but very small or no SC or SP changes on several conditioning trials, it cannot be argued that such artifacts (increase in contact area and contact pressure accompanying suppression)

are the basis of electrodermal responses.

Thus, it can be concluded that the SC and SP correlates of conditioned suppression seen in this study were determined by central and peripheral processes and were not the result of artifacts in the transducer system.

Classical Conditioning of SC and SP

Previous studies on the classical conditioning of skin conductance and skin potential have frequently been criticized for failing to control for the effects of sensitization (e.g., Stewart, Stern, Winokur, and Fredman, 1961). The difference between sensitization and true conditioning is that responses which are due to the latter process are specific to the cue that is paired with the UCS, whereas responses which are due to the former process occur to any stimulus event, presumably as the result of a general lowering of response thresholds that is brought about by the introduction of an aversive UCS. The present study, which can be considered a classical conditioning experiment with long interstimulus and intertrial intervals, employed a discrimination procedure to control for the effects of sensitization. The fact that large electrodermal responses and suppression followed CS+ but not CS- indicates that the results obtained were due to conditioning rather than sensitization.

In the present study, SC, SP, and the suppression of operant responding were conditioned at approximately the same rate. However, de Toledo and Black (1966) found that heart-rate responses conditioned much more slowly than did suppression, pre-CS and CS response rates being significantly different from each other on the second day of conditioning for suppression and on the sixth day of conditioning for heart-rate.

The difference between these results regarding heart-rate and those of the present study regarding SC and SP may simply be due to differences in the two conditioning procedures employed. Evidence in support of this view has recently been provided by de Toledo (1967), who showed that the asynchrony displayed between the acquisition of cardiac and suppression CRs disappears when shock intensity is relatively low and an appropriate CS is used. On the other hand, it may be that SC and SP are more sensitive indices of central motivational states than is heart-rate, perhaps because electrodermal functioning is not subject to homeostatic influences which distort the relationship between cardiac responding and neural activity in central motivational mechanisms. If this is true, then Overmeir's (1966) conclusion that autonomic variables are less sensitive indices of fear conditioning than overt behavioural responses might not apply equally to all autonomic systems.

Response Mechanism for Electrodermal Correlates of Conditioned Suppression

The present results showing synchronization of SC and SP and an association of these responses with suppression raise several questions about the neural mechanism underlying electrodermal correlates of conditioned suppression. Some forms which this mechanism could take are illustrated in Fig. 8. One possibility, indicated by the solid arrows, is that CS+ evokes activity in a central neural mechanism then excites electrodermal centres and somatomotor centres in parallel, leading to increments in conductance and potential with concomitant suppression of operant responding. An alternative view is that electrodermal centres are aroused, not directly by a central mechanism for fear, but indirectly by efferents from motor centres or by feedback from movement. These two possibilities are illustrated by the broken arrows in Fig. 8. A final possibility,

illustrated by the dotted arrow, is that the electrodermal effector is

---

Insert Fig. 8 here

---

activated directly by feedback from skeletal movement. The idea here is that electrodermal correlates of conditioned suppression are mediated at the spinal level and do not involve the activity of higher neural centres. The components of this model and the types of evidence which bear upon various hypotheses regarding the way in which these components are interconnected are discussed below.

Peripheral Effector Mechanisms. On the basis of evidence presented in the introduction, it would appear that the SC increments observed to occur to CS+ in the present study were due to some combination of duct-filling of the sweat glands and depolarization of epidermal membranes. Psychological stimulation, i.e., presentation of CS+, produced autonomic activation, one result of which was an increase in sweat gland activity. As the sweat glands filled with fluid, more conducting paths for current were opened up, and the longitudinal resistance of the sweat gland duct was short-circuited. Both of these effects would have increased SC. Since polarized epidermal membranes also offer resistance to applied current, a second process that was presumably involved in the generation of SC responses was the depolarization of membranes near the skin surface and lining the sweat gland tubules. The fact that iontophoretic induction of atropine eliminates skin conductance responses and greatly lowers SC level indicates that these processes are cholinergically mediated (Lader and Montagu, 1962).

The peripheral mechanism for skin potential is the subject of considerable controversy. The possibility of significant species

differences in the form of SP activity, and in the relation of some of the features of the SP response to the SC response to the SC response, make speculation here somewhat hazardous. For example, SP responses in primates are often diphasic in form, consisting of an increase in negativity followed by a positive wave, whereas, in the present study on rats, only uniphasic negative waves were observed. Wang (1964) states that SP responses in the cat are exclusively negative-going, although this has been disputed by Martin and Venables, 1966). Another possible species difference concerns the relationship of the latency of the SP response to the SC response. In the present study, SP responses appeared to have a shorter latency than the SC responses, and a similar difference has been observed with the cat (Wilcott, 1965a). However, in humans under normal conditions (Jefress, 1928; Wilcott, 1958a) and in monkeys (Wilcott, 1965c) SC and negative-going SP responses appear to have the same latency. Although the significance of species differences in latency characteristics is as yet unclear, Wilcott (1967) takes them to indicate that SP and SC responses in primates are influenced to a greater extent by the same underlying peripheral mechanisms than in the rat and cat.

Despite these complexities, a tentative explanation for the SP responses observed in the present study can still be offered. It may be that SP activity is due to sodium pumping across the sweat gland duct (Fowles and Venables, 1968; Lykken, 1966). While the exact mechanism for the production of potential is as yet unknown, SP may be a secondary result of ionic changes which follow pumping, or it may be that the sodium pump is itself electrogenic (Fowles and Venables, 1968). It is interesting to note that, in the case of rat #50 which appeared to have a defective electrodermal effector, changes in SP during the interstimulus interval

were observed only if SC changed as well. A sodium pumping hypothesis could explain this observation if SP were secondary to pumping and sweat secretion were impaired, or if the pumping process were itself electrogenic and neural input to the sweat glands were somewhat impeded. The fact that negative-going SP responses remain when sweat secretion is abolished through exsanguination of a limb suggests that the sodium pump mechanism is electrogenic in nature (Wilcott, 1958b).

The peripheral effector for suppression obviously involves the musculature, but the precise muscle groups utilized have not been identified. The most reasonable hypothesis is that suppression involves the cessation of activity in those muscle groups which are required for bar-pressing, and also an increase in the activity of those groups leading to freezing behaviour. One might expect an increase in activity of muscle groups associated with freezing since freezing behaviour normally accompanies the suppression of operant responding (Roberts, 1967b).

Central Mechanisms. The central neural structures involved in the control of electrodermal activity were discussed earlier. Most important among these appear to be the anterior hypothalamus, the most powerful excitatory sweat centre in the brain, and the rhomencephalic reticular formation, the most powerful inhibitory centre (Wang, 1964). In addition, Wang (1964) has provided evidence which suggests that the synchronization of electrodermal responses across the limbs of the cat is controlled by a structure in the striopallidum. The neural structures responsible for the control of conditioned suppression have not been as thoroughly studied, but the involvement of structures in the limbic system, most notably the septal area (Brady and Nauta, 1953; Harvey, Lints, Jacobson, and Hunt, 1965), seems quite clear. A thorough discussion of

the neural structures involved in the control of passive avoidance responding and freezing behaviour, and therefore, of conditioned suppression, has recently been provided by McCleary (1966).

The model outlined in Fig. 8 proposed three ways in which central neural processes could generate electrodermal correlates of conditioned suppression. First of all, a central motivational mechanism for fear, involving perhaps the septal-hippocampal-thalamic-hypothalamic areas, could concomitantly innervate both motor and electrodermal neural centres which then initiate effector activity. Muscular responses underlying suppression and electrodermal activity would thus arise as parallel events from common innervation and become attached to CS+ through reinforcement. The second possibility is that electrodermal neural centres are excited indirectly by efferents from motor centres or possibly by feedback from skeletal movement. A third possibility is that the electrodermal effector is activated directly by feedback from skeletal movement.

A general conclusion which emerges from the data presented by Wang (1964) is that sweat gland activity is normally under strong central control. For example, spontaneous electrodermal responses are desynchronized across the limbs of spinal organisms (Fuhrer and Kilbey, 1967), and also in preparations capable of movement, provided that the striopallidum remains connected with the spinal cord (Wang, 1964, pp. 107-108). If spinally-mediated feedback from movement were an important determinant of electrodermal activity, such dependence of responding on higher neural centres would not seem likely. Because of the importance of central neural processes in sweat gland functioning, and also because electrodermal correlates of conditioned suppression are highly synchronized across the rear limbs, the hypothesis that the elctrodermal effector is activated

directly by feedback from movement (the dotted arrow in Fig. 8) seems very unlikely.

The remaining possibilities, according to the model of Fig. 8, are that that electrodermal centres are excited (a) directly by a central fear mechanism, or (b) indirectly by efferents from the motor centres or by feedback from movement. At the present time, one can say only that both views are possible, since both would predict a high degree of association between electrodermal responses and suppression. It is possible, however, to specify the types of behavioural data which bear upon these alternative views. An obvious prediction made by the hypothesis that electrodermal correlates of conditioned suppression are due to the activation of excitatory sweat centres by a central fear mechanism is that the occurrence of electrodermal responses does not depend upon the form of the overt behaviour observed. For example, it may be possible to show that CS+ retains its ability to evoke large increments in the tonic level of SC and SP even though the behavioural response observed is an increase rather than a decrease in rate of bar-pressing. This might be accomplished by presenting CS+ while S was performing a Sidman avoidance response. It might also be possible to demonstrate that CS- in CER training evokes a substantial increase in rate of bar-pressing attended by a decrease in SC and SP. No evidence of such facilitation of bar-pressing or a decrease in SP and SC was found in the present study, but this was probably due to the fact that the baseline level of operant responding was very high (3,107 presses per hour on the second pretest day). In experiments of this type, which are designed to demonstrate a dissociation of overt behaviour from tonic changes in SC and SP, it would be necessary to record measures of somatic responding in addition to lever-pressing, to make sure that some

undetected somatic response, such as muscle tension or respiration, was not responsible for the electrodermal changes observed.

The role of skeletal feedback in the generation of electrodermal correlates could be examined in many ways. One possibility would be to demonstrate that CS+ evokes large increments in SC and SP even though movement cannot occur, e.g., during curarization when the skeletal musculature if temporarily paralyzed and movement is prevented. Another possible approach to examine the dissociation between electrodermal changes and behavioural responses is through the use of brain lesions. For example, there is reason to believe that septal lesions will abolish conditioned suppression (Harvey et al, 1965), but since the septal area does not appear to be an excitatory sweat centre (Holdstock, 1967), electrodermal responses to CS+ might remain relatively intact. If this outcome were observed, it would be difficult to argue that electrodermal correlates of conditioned suppression are due to feedback from behaviours leading to suppression, or to the activation of excitatory sweat glad centres by efferents from central structures which control behaviours responsible for suppression.

SUMMARY

The traditional grid techniques of electrodermal recording are subject to a number of measurement artifacts which complicate interpretation of the data. In view of the inadequacies of these grid methods, the present study employed the use of an improved recording technique which was relatively free of the artifacts inherent in grid techniques. In this fixed electrode technique the animals were immobilized on a restraining platform and electrodermal recording electrodes attached directly to the footpads. Skin conductance (SC), skin potential (SP), and lever-pressing were then examined throughout discriminative CER training while the rats were kept under restraint.

Conditioned suppression of operant behaviour was accompanied by an increase in SC amplitude (approximately .8 micromho) and an increase in negativity of SP (approximately 10 mv). Suppression and electrodermal responses appeared to be conditioned simultaneously, with clear-cut effects occurring by the second day of conditioning. The results regarding SP and SC were attributed to neural control of electrodermal activity rather than to measurement artifacts. Several aspects of a model of the underlying response mechanism for electrodermal correlates of conditioned suppression were discussed.

FIGURES AND TABLES

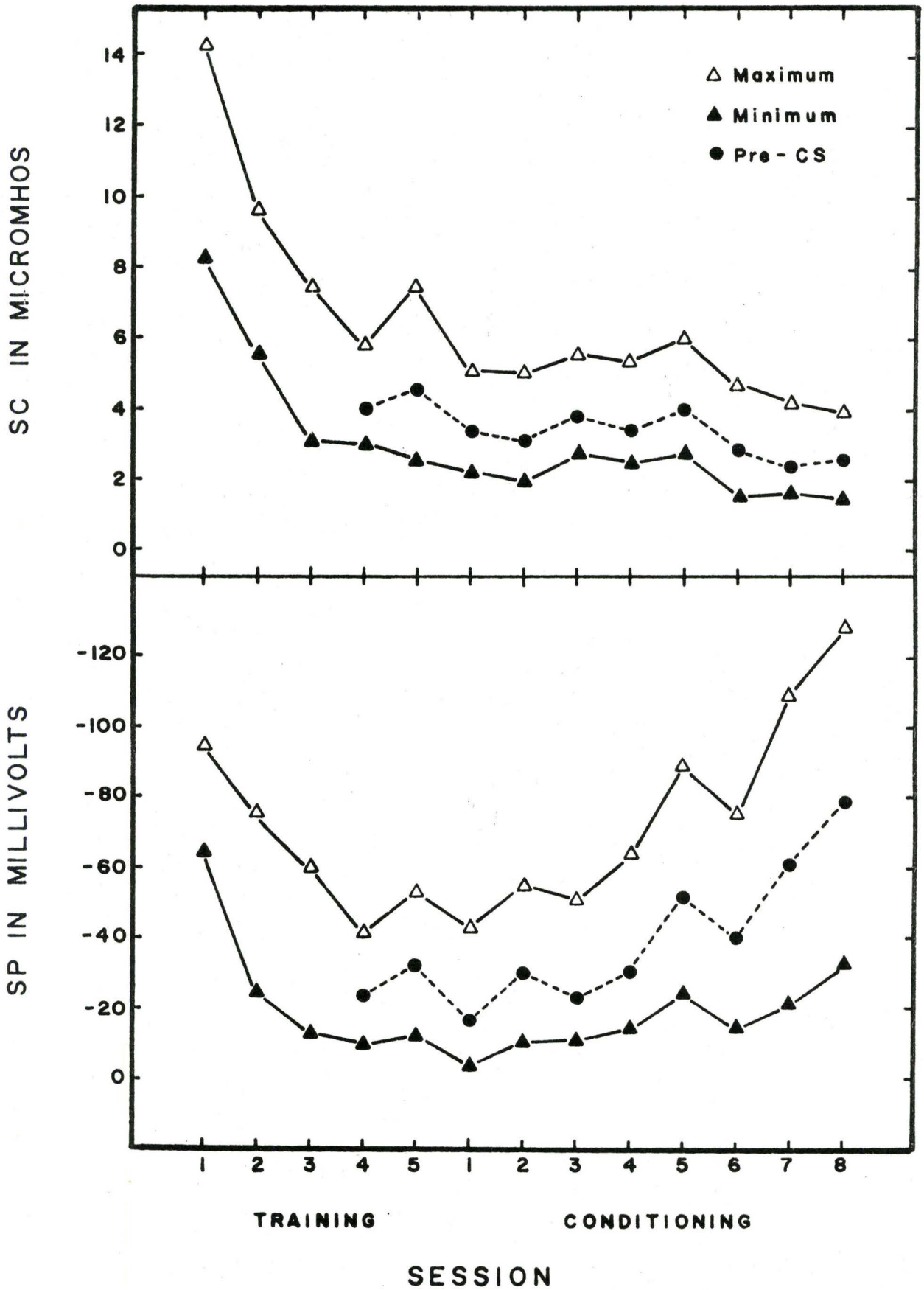

SC IN MICROMHOS
14
12
10
8
6
4
2
0
Maximum
Minimum
Pre - CS
SP IN MILLIVOLTS
-120
-100
-80
-60
-40
-20
0
1
2
3
4
5
1
2
3
4
5
6
7
8
TRAINING
CONDITIONING
SESSION

Figure 1

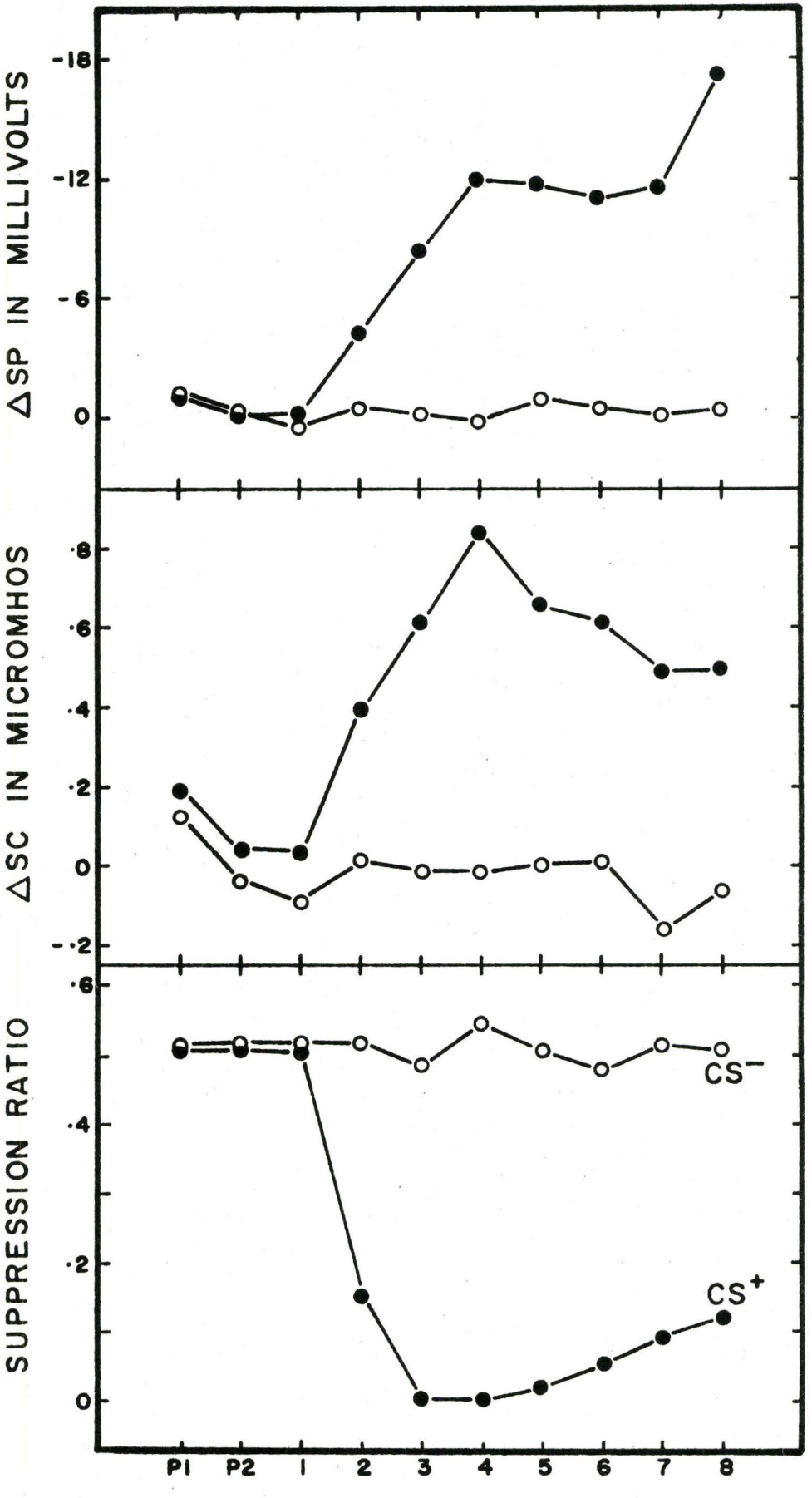

ΔSP IN MILLIVOLTS
-18
-12
-6
0
ΔSC IN MICROMHOS
.8
.6
.4
.2
0
-.2
SUPPRESSION RATIO
.6
.4
.2
0
CS⁻
CS⁺
P1
P2
1
2
3
4
5
6
7
8
SESSION

Figure 2

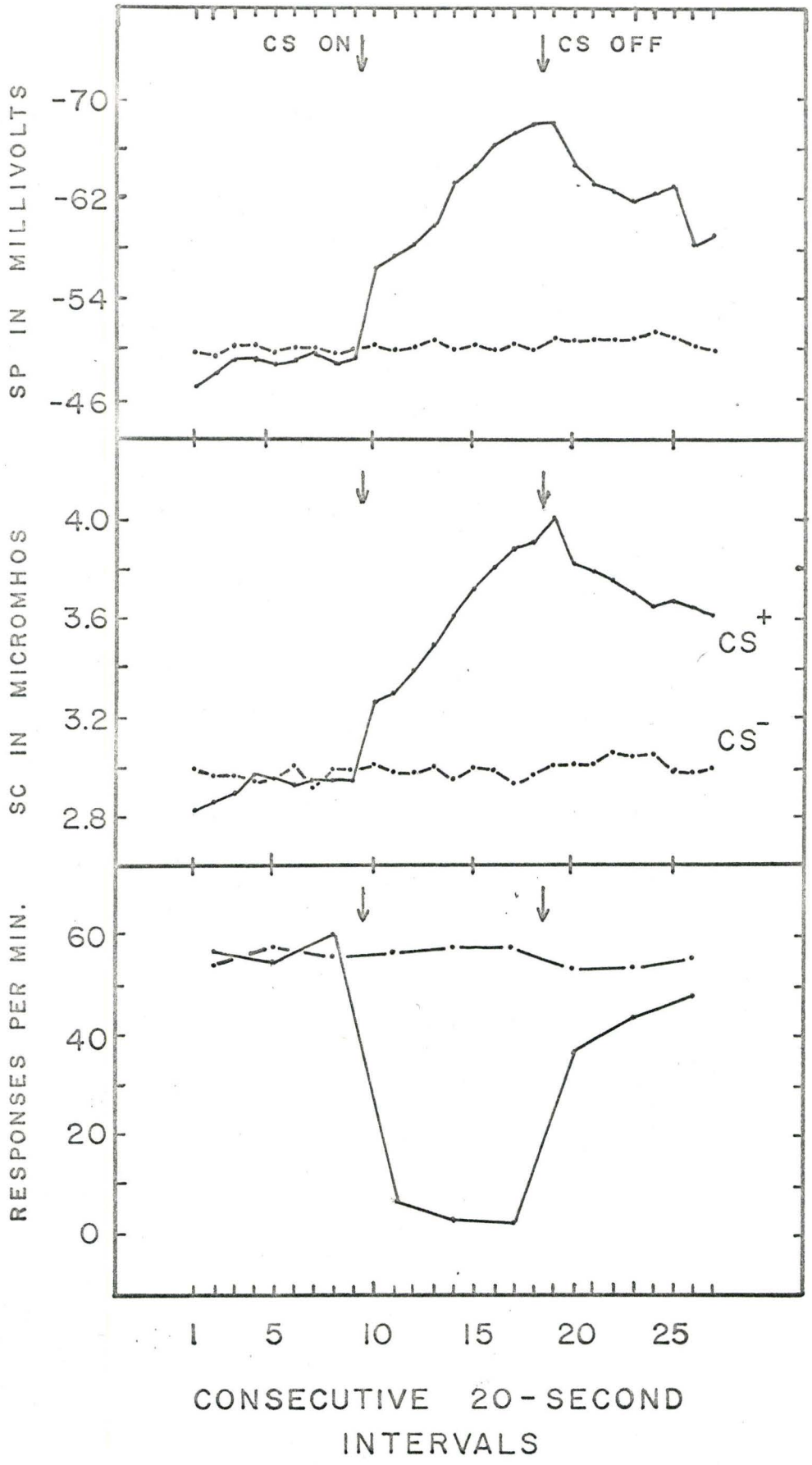

CS ON
CS OFF
SP IN MILLIVOLTS
-70
-62
-54
-46
SC IN MICROMHOS
4.0
3.6
3.2
2.8
CS+
CS-
RESPONSES PER MIN.
60
40
20
0
1
5
10
15
20
25
CONSECUTIVE 20-SECOND INTERVALS

Figure 3

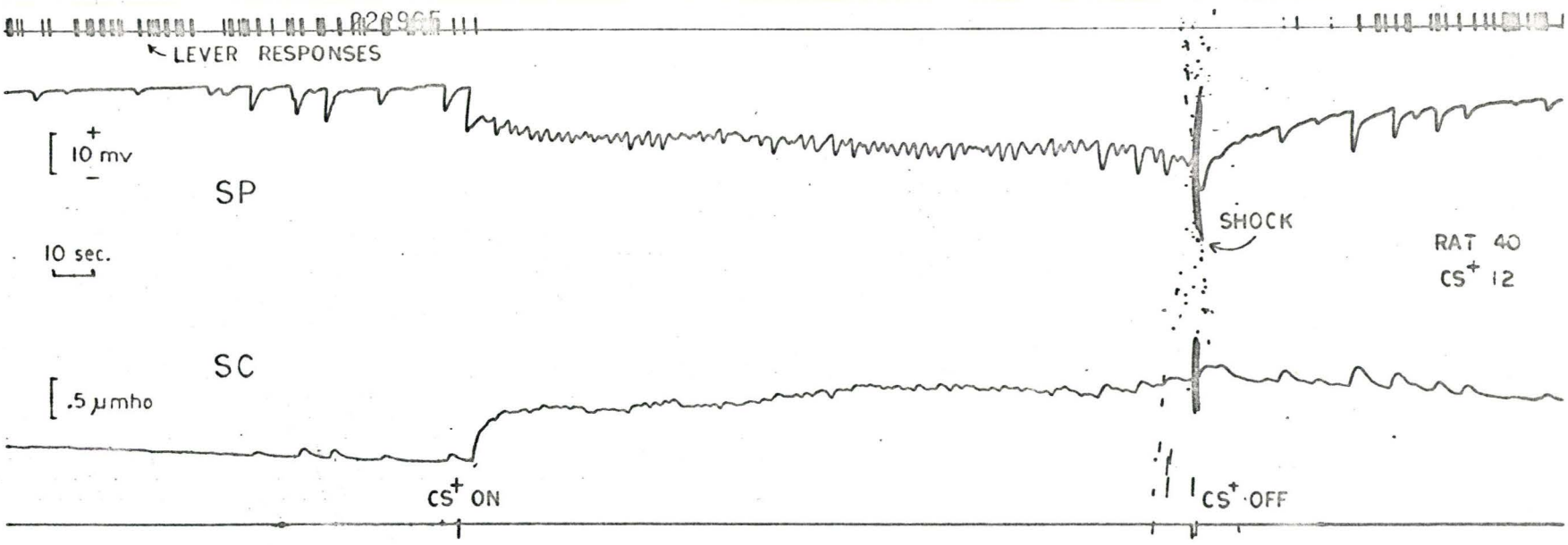

LEVER RESPONSES
+
10 mv
−
SP
10 sec.
SHOCK
RAT 40
CS+ 12
SC
.5 µmho
CS+ ON
CS+ OFF

Figure 4

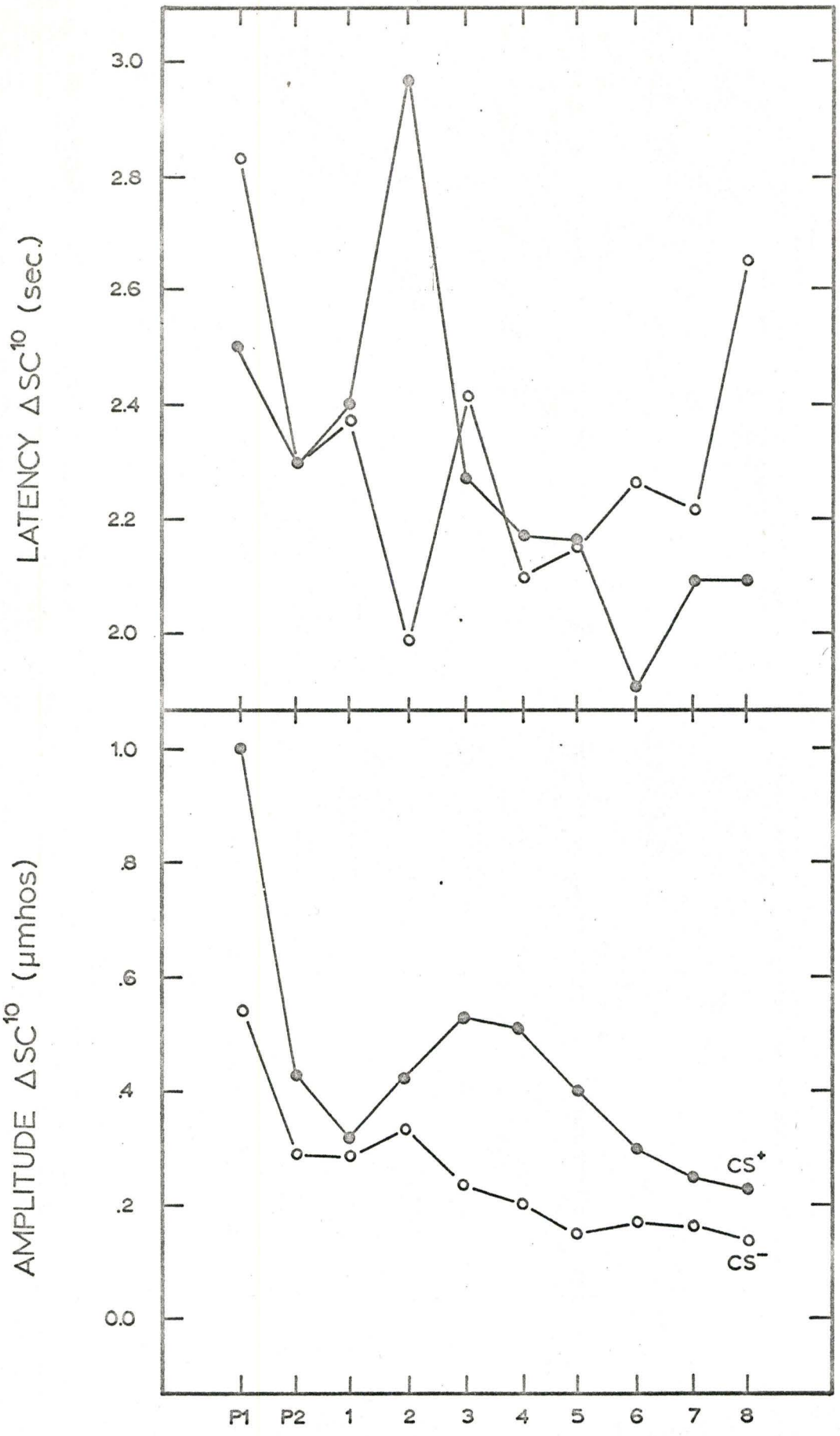
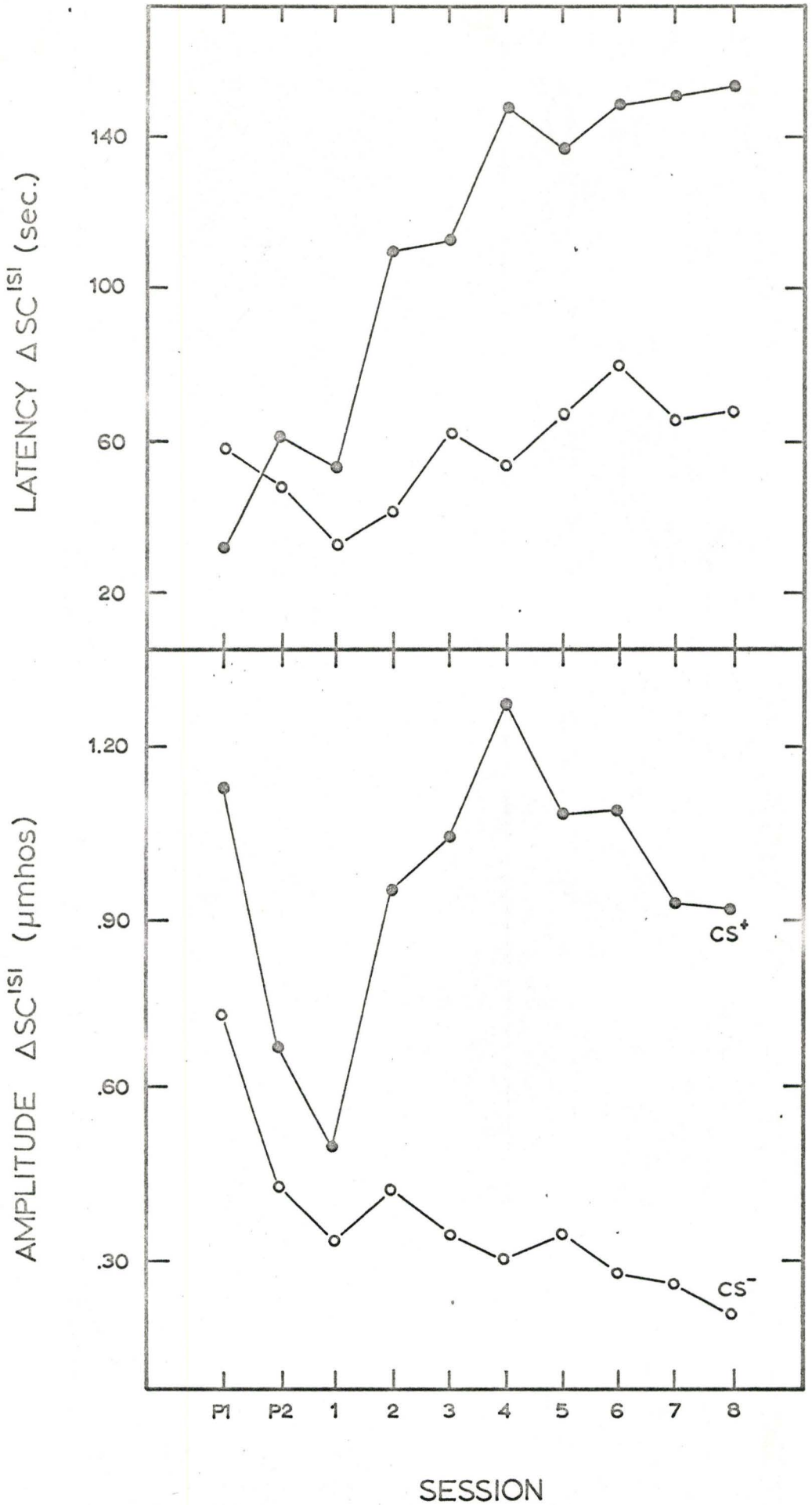

LATENCY ΔSC10 (sec.)
3.0
2.8
2.6
2.4
2.2
2.0
AMPLITUDE ΔSC10 (μmhos)
1.0
.8
.6
.4
.2
0.0
CS+
CS−
P1
P2
1
2
3
4
5
6
7
8
SESSION

Figure 5


LATENCY ΔSC^ISI (sec.)
140
100
60
20
AMPLITUDE ΔSC^ISI (μmhos)
1.20
.90
.60
.30
CS+
CS-
P1
P2
1
2
3
4
5
6
7
8
SESSION

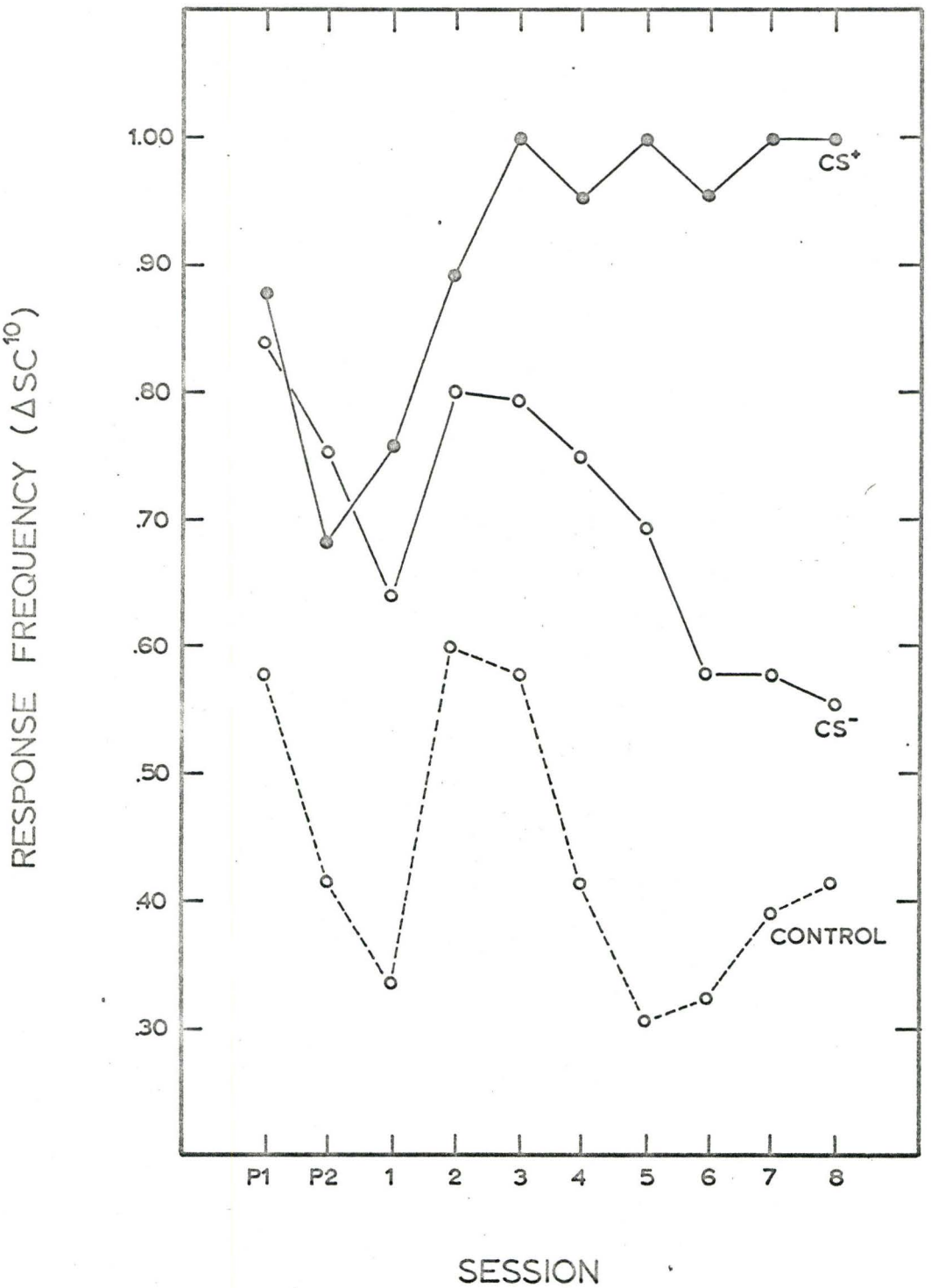

RESPONSE FREQUENCY (ΔSC10)
1.00
.90
.80
.70
.60
.50
.40
.30
CS+
CS-
CONTROL
P1
P2
1
2
3
4
5
6
7
8
SESSION

Figure 7

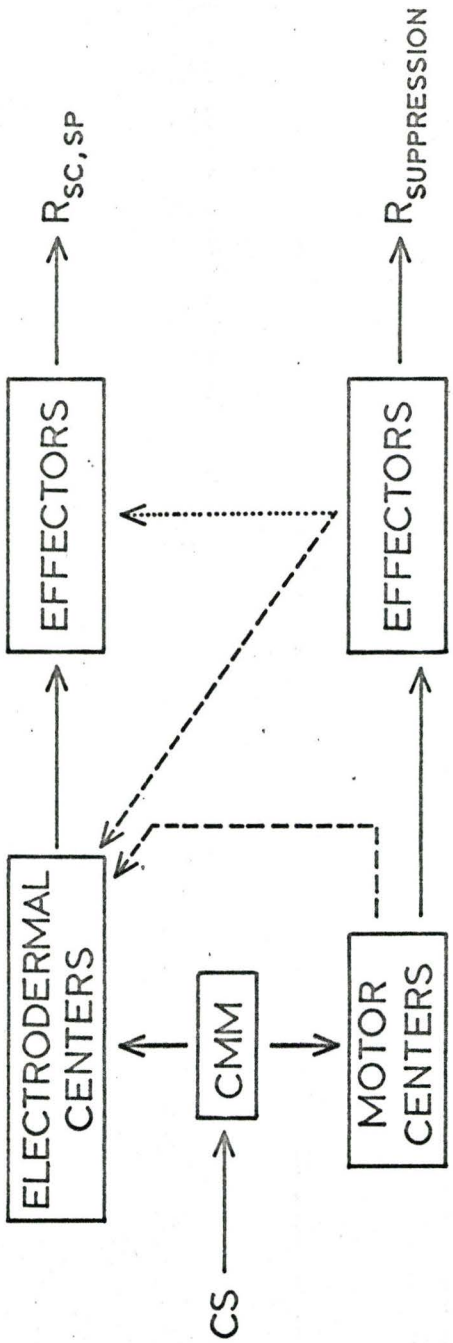

$R_{SC, SP}$
$R_{SUPPRESSION}$
EFFECTORS
EFFECTORS
ELECTRODERMAL CENTERS
CMM
MOTOR CENTERS
CS

Figure 8

Table 1

T-tests on response amplitudes

	PT-1			PT-2			C-1		
	CS+	CS-	CS+-CS-	CS+	CS-	CS+-CS-	CS+	CS-	CS+-CS-
Δconductance	1.821	2.090	.426	1.150	.743	1.745	1.093	-2.647*	2.545*
Δpotential	2.305*	3.977**	-.005	.515	.595	- .045	1.333	-1.632	2.424*
Δbar-pressing	1.940	.855	.444	.615	2.138	- .354	.499	1.931	- .987

	C-2			C-3		
	CS+	CS-	CS+-CS-	CS+	CS-	CS+-CS-
Δconductance	3.761**	.621	3.568**	4.643**	.458	4.021**
Δpotential	4.051**	.766	3.685**	5.986**	.432	5.719**
Δbar-pressing	-5.506**	.780	7.122**	-99.389**	-.402	5.616**

* $p < .05$ two-tailed test
** $p < .01$ two-tailed test

BIBLIOGRAPHY

Anderson, D.C., Plant, C. and Paden, P. Conditioned suppression of a running response as related to competing response drive and basal skin resistance level. J. comp. physiol. Psychol., 1967 63, 282-288.

Annau, Z. and Kamin, L.J. The conditioned emotional response as a function of intensity of the US. J. comp. physiol. Psychol., 1961, 54, 428-432.

Brady, J.V. and Nauta, W.J.H. Subcortical mechanisms in emotional behavior: affective changes following septal forebrain lesions in the albino rat. J. comp. physiol. Psychol., 1953, 46, 339-346.

Carran, A.B., Yeudall, L.T. and Royce, J.R. Voltage level and skin resistance in avoidance conditioning of inbred strains of mice. J. comp. phsyiol. Psychol., 1964, 58, 427-430.

Darrow, C.W. The rationale for treating the change in galvanic skin response as a change in conductance. Psychophysiology, 1964, 1, 31-38.

de Toledo, L. Heart-rate and EMG changes during conditioned suppression in rats. Paper read at Eastern Psychological Association, Boston, 1967.

de Toledo, L. and Black, A.H. Heart-rate: changes during conditioned suppression in rats. Science, 1966, 152, 1404-1406.

Deutsch, R. Changes in skin resistance, defecation, and activity in the presence of a fear-provoking stimulus. Paper read at Eastern Psychological Association, Boston, 1967.

Edelberg, R. Response of cutaneous water barrier to ideational stimulation: a GSR component. J. comp. physiol. Psychol., 1966, 61, 28-33.

Fowles, D.C. and Venables, P.H. Endocrine factors in palmar skin potential. Psychon. Sci., 1968, 10, 387-388.

Fuhrer, M.J. and Kilbey, M. Effects of spinal cord transections on electrodermal activity in man. Psychophysiology, 1967, 4, 176-186.

Grings, W.W. Methodological considerations underlying electrodermal measurement. J. Psych., 1953, 35, 271-282.

Harvey, J.A., Lints, C.E., Jacobson, L.E. and Hunt, H.F. Effects of lesions in the septal area on conditioned fear and discriminated instrumental punishment in the albino rat. J. comp. physiol. Psychol. 1965, 59, 37-48.

Holdstock, T.L. Effects of septal stimulation in rats on heart-rate and galvanic skin response. Psychon. Sci., 1967, 9, 37-38.

Jefress, T.A. Galvanic phenomena of the skin. J. exp. Psych. 1928, 11, 130-144.

Kaplan, R. Rat basal resistance level under stress and non-stress conditions. J. comp. physiol. Psychol. 1963, 56, 775-777 (b).

Kimmel, H.D. Further analysis of GSR conditioning: a reply to Stewart, Stern, Winokur and Fredman. Psych. Rev., 1964, 71, 160-166.

Lader, M.H. and Montagu, J.D. The psycho-galvanic reflex: a pharmacological study of the peripheral mechanism. J. Neurol. Neurosurg. Psychiat., 1962, 25, 126-133.

Lloyd, D.P.C. Secretion and reabsorption in eccrine sweat glands. In W. Montagna (Ed.), Advances in Biology of Skin, Vol. 3. New York: Permagon Press, 1962. pp. 127-151.

Lykken, D.T. Neuropsychology and psychophysiology in personality research. In E.F. Borgatta and W.W. Lambert (Eds), Handbook of Personality Theory and Research. Chicago: Rand McNally, 1966.

Lykken, D.T., Miller, R.D. and Strahan, R.F. Some properties of skin conductance and potential. Psychophysiology, 1968, in press.

Martin, I. and Venables, P.H. Mechanisms of palmar skin resistance and skin potential. Psych. Bull., 1966, 65, 347-357.

McCleary, R.A. Response-modulating functions of the limbic system: initiation and suppression. In E. Stellar and J.M. Sprague (Eds), Progress in Physiological Psychology, Vol. 1, New York: Academic Press, 1966, pp. 210-272.

Miller, R.D. Silver-silver chloride electrodermal electrodes. Psychophysiology, 1968, in press.

Montagu, J.D. and Coles, E.M. Mechanism and measurement of the galvanic skin response. Psych. Bull., 1966, 65, 261-279.

Overmier, J.B. Instrumental and cardiac indices of Pavlovian fear conditioning as a function of US duration. J. comp. physiol. Psychol., 1966, 62, 15-20.

Roberts, L.E. Central, peripheral, and artifactual determinants of skin resistance in the mouse. J. comp. physiol. Psychol., 1967, 64, 318-328 (a).

Roberts, L.E. Changes in skin conductance and operant behavior during discriminative CER conditioning in the mouse. Paper read at Eastern Psychological Association, Boston, 1967 (b).

Roberts, L.E. Experiments on electrodermal phenomena in animals. Mimeographed manuscript, McMaster University, 1967 (c).

Stewart, M.A., Stern, J.A., Winokur, G., and Fredman, S. An analysis of GSR conditioning. Psych. Rev., 1961, 68, 60-67.

Wang, G.H. The Neural Control of Sweating. Madison: Univ. of Wisconsin Press, 1964.

Wilcott, R.C. Correlations of skin resistance and potential. J. comp. physiol. Psychol., 1958, 51, 295-300 (b).

Wilcott, R.C. A comparative study of the skin potential, skin resistance, and sweating of the cat's footpad. Psychophysiology, 1965, 2, 62-71 (a).

Wilcott, R.C. Observations on skin potential, skin resistance, and sweating of two rhesus monkeys. Cleveland: Author, 1965. (Mimeo) (c)

Wilcott, R.C. Arousal sweating and electrodermal phenoma. Psych. Bull., 1967, 67, 58-72.

Woll, S. A subdermal reference electrode technique for studying the electrodermal correlates of conditioned suppression. Unpublished M.A. thesis, McMaster University, 1968.