EVOKED POTENTIALS RECORDED FROM THE SCALP DURING CONDITIONING OF THE GALVANIC SKIN RESPONSE IN MAN

by

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T A B L E O F C O N T E N T S

I. INTRODUCTION

The evoked responses which occur in the brain as a result of a brief external stimulus are electrical signs of neural activity whose physiological and behavioral significance remains unexplained. A number of attempts have been made to find systematic changes in evoked potentials (EPs) during or after conditioning, the more obvious of these changes being alterations in amplitude and latency.

The present study is concerned with changes in scalp-recorded EPs to clicks in human subjects during conditioning of a Galvanic Skin Response (GSR) to the click. Both amplitude and latency of the EP will be examined for changes.

Amplitude is not difficult to measure, requiring only sufficient amplification to give reliable measurements of the response, which may range from a few microvolts to several millivolts. Among the variables affecting amplitude, an important one is the distance between the recording electrode(s) and the tissue which is generating the electrical signal. The closer the electrode to the source, the larger the signal amplitude. When electrodes are relatively distant, as in recording from outside the scalp, amplitude becomes somewhat more complicated to measure. The brain signal in this case is very small indeed and requires special measuring techniques in order for it to be differentiated from background "noise."

Latency of EPs has been specified (Dustman and Beck, 1963; Geisler and Rosenblith, 1962; Williams, Morlock, Morlock,

and Lubin, 1964) by measuring the time from stimulus onset to the first positive or negative peak or to salient later peaks. Because the EPs for a given subject show a characteristic pattern (Dustman and Beck, 1963; Geisler, Frishkopf, and Rosenblith, 1958, and see Plate 2.), it is usually possible to single out a particular positive or negative component which occurs within a characteristic latency range. Comparisons can then be made of the latency of this component during successive measurements under the same or different conditions.

Although changes in latency have not received much attention in experiments examining EPs during conditioning, changes in amplitude have been reported in many instances.

The earliest report of such a change in amplitude is apparently a study by Artemyev and Bezladnova in 1952 (reported in Morrell, 1961). Using cats, they conditioned a response of leg flexion. The conditioned stimulus (CS) was a tone and the unconditioned stimulus (US) was shock to the paw. They observed that EPs recorded from auditory cortex grew larger as acquisition of the flexion response progressed.

In 1956 Galambos, Sheatz, and Vernier reported essentially the same result. Using cats they conditioned what was apparently a combined orienting response plus an "emotional" response of crouching, snarling, etc., using a click CS and thoracic shock. They demonstrated that EP amplitude increased even in cats under Flaxedil, and that animals immobilized by Flaxedil throughout the CS - US pairings exhibited both the increase in EP and the presence of overt responses after the

Flaxedil wore off. The EPs were recorded by means of chronic implanted electrodes in their ten cats. Galambos et al reported that the enhancement of EP amplitude died away after hours or days. They also reported that preliminary monotonous repetition of the CS produced a decrease in EPs, which they referred to as habituation.

There have been several reports of the effects of classical appetitive conditioning on EP amplitude. Kogan (1960) and Roitbak (1960) found that EPs in the auditory cortex of cats decreased when the CS was followed by a food reward. Both of these studies reported that EP amplitude increased during habituation.

In a study by Hearst, Beer, Sheatz, and Galambos (1960), several different conditioning paradigms were investigated using monkeys. Electrodes were implanted in various cortical and sub-cortical locations. An acoustic CS was used. The following results were obtained: (1) Classical conditioning with a food reward produced increased amplitude of EPs to the Cs. (2) Classical aversive conditioning involving a discrimination produced increases only in the EPs to the positive stimulus. Reversal of the discriminative stimuli did not produce the expected changes in the amplitudes of the EPs corresponding to those stimuli. In this study operant reward and avoidance conditioning were also reported. During these two types of conditioning EPs tended to decrease, but

only in the sub-cortical locations. It should be pointed out that only a small number of animals was used. One animal received the classical appetitive conditioning, and three animals received the classical aversive conditioning. The EP changes were reversible by an extinction procedure and some were repeated a number of times for each animal. Decreased EPs were reported after habituation training.

In a study using a number of cats and monkeys, Galambos and Sheatz (1962) again obtained EP increases with classical aversive conditioning. They used either click or flash as CS in a series of experiments, with EPs submitted to an averaging procedure for more reliable analysis. The overt response was eyeblink to an air-puff US. All subjects were conditioned and extinguished many times. Behavioral extinction usually occurred long before the EP returned to its pre-conditioning level. EPs were reported to decrease under habituation to the CS. Habituation training prior to conditioning was given until the EP was judged to be minimal, which took as long as several weeks in some cases.

A serious objection to all of the studies cited so far is that they included no controls for sensitization or pseudo-conditioning. Gerken and Neff (1963) made this point and sought to assess its effects in an experiment using six cats, each with four different electrode placements in auditory cortex. The CS was a train of clicks, the US was shock. They gave all the cats extensive pre-conditioning with the CS alone,

then gave two cats avoidance conditioning, two cats classical conditioning, two cats "pseudo-conditioning" (random unpaired click and shock) followed by avoidance conditioning for one and classical conditioning for the other. Their results indicated that (1) EPs increased during pseudo-conditioning but increased more in conditioning not preceded by pseudo-conditioning; (2) conditioning after pseudo-conditioning did not produce increased EPs; (3) during pre-conditioning some EP components increased while some decreased; (4) changes in overt responses often preceded changes in EP, and there was no systematic relation between EP magnitude and the presence or absence of an overt CR. Again, it is fair to object that these results are based on only a few animals. However, this study certainly casts doubt on the interpretation of the earlier conditioning studies, in the light of the increased EPs observed in the unpaired pseudo-conditioning control group. The variability of the EP changes in this study, as well as the failure to demonstrate any systematic relationship between EPs and CRs, suggests very strongly that if EP correlates of conditioning do in fact exist, larger numbers of subjects are needed to find them.

Other studies in which increased EPs during classical aversive conditioning have been reported include Jouvet and Hernandez-Peon (1957), John and Killam (1959), and Marsh, McCarthy, Sheatz, and Galambos (1961). The same objections of small N and lack of adequate controls apply to their conclusions.

The reader will note that no studies using human

subjects have been mentioned. This is because of the apparent dearth of such studies. For reasons which are discussed more fully below, scalp-recording of human EPs is technically difficult and the extensive study of such records has become feasible only in recent years. A study by W. Grey Walter (1964) is the only human conditioning study which has come to the attention of this writer. Walter trained a large number of subjects in an instrumental conditioning situation whose results will not concern us here; however, in preliminary training each subject was exposed to a type of classical conditioning which does have some bearing on the present discussion. In some cases the CS was a flash followed by a repetitive click US, in other cases the CS was a single click and the US was repetitive flash. The intensity of the flash is not reported, but the click was "startling at first but not painful" and estimated at 80 db. The US followed the CS by 1 second. Walter found that the scalp-recorded EP to the CS increased in amplitude, while that to the US decreased. There are several difficulties in interpreting this data. No overt response which could be used as an index of conditioning was measured. Once again, no controls were included for sensitization or pseudo-conditioning. Another objection which can be raised to Walter's and the other conditioning studies mentioned, except for those of Gerken and Neff, and Marsh et al, is that comparisons of data are not supported by statistical tests. Further, although sample individual records are given in each report, quantitative

comparisons which include all the data are omitted in nearly all the reports.

Changes in EPs not associated with conditioning have been observed as a function of a number of variables such as stimulus intensity (Geisler, Frishkopf, and Rosenblith, 1958), stages of sleep (Williams, Morlock, Morlock, and Lubin, 1964), and the degree of physical activity of the subject (Thompson and Shaw, 1965). One such variable which may be significant during conditioning is the state of arousal or attentiveness of the subject. Although it is difficult to define "attention", in several studies operational definitions of attention have been used to demonstrate a correlation between attention and EP amplitude. Thompson and Shaw (1965) found that EPs recorded from association cortex in cats were inversely related to attention as defined by the degree of behavioral orienting. In a number of studies using human subjects, the opposite effect has been detected (Davis, 1964; Haider, Spong, and Lindsley, 1964; Satterfield, 1965; Spong, Haider, and Lindsley, 1965). In these studies EP amplitude appears to be directly related to attention as defined by having the subject count successive stimuli or judge their intensity. The discrepancy between the animal and the human data suggests that the problem of definition remains unsolved. The usefulness of "attention" as a construct to help interpret changes during conditioning is as yet very limited.

In the experiment which will be reported at length below, the GSR was chosen as the response to be conditioned because it has been shown to be readily conditionable (Kimble, 1961; Prokasy, 1965) and because it is relatively easy to measure by the potential method (Wang, 1964). Furthermore, it is a response which does not conflict with EP measurements by necessitating bodily movements on the part of the subject.

An Enhancetron, one of the special class of on-line data-processing devices which provide "averaged" responses, was used to measure EPs. When electrodes are placed outside the scalp the EP recorded is not only much smaller than that which could be recorded directly from the cortex, but also is obscured by a very poor signal-to-noise ratio. Sources of noise include other electrical events in the body such as the EEG, retinal potentials, myogenic potentials from ear and other muscles, as well as 60-cycle and high-frequency interference originating outside the subject's body. A great improvement in the signal-to-noise ratio can be produced by summing together many consecutive evoked responses from a given subject and taking the average magnitude at each instant after stimulus onset. Because the evoked changes in potential occur at characteristic latencies after the stimulus, while extraneous electrical events are not time-locked with the stimulus, averaging a number of responses together serves to emphasize the

reiterated evoked changes while extraneous changes tend to cancel each other out.[1] The larger the number of responses averaged together, the better the signal-to-noise improvement and the fidelity of signal representation, up to a limit dictated by the particular recording and averaging equipment used. Many studies in which human EPs have been averaged in this way report the summing together of hundreds of consecutive EPs. However, as has been pointed out elsewhere (Brazier, 1964), it may be dangerous to combine large numbers of EPs in a single average, for changes occurring over time may be lost. This objection is particularly forceful in a conditioning study, where it is the lability of the EP over a limited number of trials, probably less than 100, that is the variable of interest. W. Grey Walter also recognized this, and in his study he combined no more than 12 EPs in his average EP measurement. The present writer experimented with different numbers of EPs averaged together and decided upon 15 as an acceptable compromise between optimal signal enhancement and the time limitation imposed by the use of human subjects for conditioning.

[1] The averaging procedure assumes that the variable individual EP measurements exemplify "an invariant response plus independent random noise" (Rosenblith, 1962). Rosenblith demonstrates that this assumption is inaccurate for some data at least. Analysis of the dispersion of instantaneous voltages about a sample averaged EP indicated that the dispersion varied as a function of time. No doubt part of this variation in dispersion is due to the variation of EP latencies from one response to the next. Whether there are other significant causes of the changes in dispersion remains to be determined. A further point is that the dispersion is never zero. An averaged EP is always more "blurred" than an individually recorded response.

II. MATERIALS AND METHODS

Subjects

The plan of the experiment called for a total of thirty subjects (Ss), ten in each of three groups. In order to get thirty acceptable protocols, fifty-three Ss were run through the full experiment, and twenty-three protocols were discarded for the following reasons:

11	equipment failure
5	measurements of EP were too noisy to yield adequate averaged responses
3	averaged EPs lacked consistent or identifiable wave form, making amplitude or latency comparisons hazardous
4	GSR during most or all trials was totally absent or too low to measure reliably.

The Ss were male and female paid volunteers between the ages of eighteen and thirty-five, drawn principally from among the graduate and medical students at the University of Oregon Medical School and first year psychology students at Portland State College. The sample also included some employees of the Medical School and friends and relatives of some of the students.

Prospective Ss were told by the experimenter (E) that they would receive "weak electric shock to the wrist."[1] Since the prospect of receiving electric shock proved to be a strong deterrent, especially to female volunteers, a special policy with regard to administering shocks was adopted. The Ss were informed that the shock

[1] In compliance with regulations and policies of the National Institutes of Health and the University of Oregon Medical School regarding the use of human subjects in experiments, no subject was included in the experiment unless he had signed a prior release stating that he agreed to the use of electric shock stimuli.

used in the experiment would be "annoying but not painful," and that the shock intensity would be determined according to their consent prior to beginning the experiment.

Recording Techniques

(1) Preparation of Subjects; Electrodes

When the S arrived at the laboratory he was seated and the scalp electrode was applied first. It was a standard tin EEG electrode, 3/8 in. in diameter, cup-shaped and filled with EEG gel (Beckman Offner Paste, consisting of glycerine, gum tragacanth and saline). The scalp electrode was located on the midline one to two inches behind the vertex at a point corresponding to the midpoint between C_z and P_z in the ten twenty electrode system (Jasper, 1958). This was a location which preliminary testing had shown to be one where EPs to click stimuli were large. The electrode was held in place with collodion, after the scalp area underneath was cleaned with acetone. In most cases a firm, long-lasting connection was obtained resulting in a minimum of "noise" visible during recording. The indifferent electrode used as a reference for EP recording was a second EEG cup electrode soldered to a screw-type earring. This electrode was also filled with EEG gel and was firmly attached to the right earlobe after the earlobe was cleaned with acetone.

The S was then shown into the room where the experiment would take place and was seated while GSR and shock electrodes were applied. The GSR electrodes were standard, consisting

of 3/8 in. silver-silver chloride discs imbedded in flexible plastic molds which provided an overall diameter of 1 1/8 in. The molds were filled with EEG gel which made the actual electrical contact between the S's skin and the metal electrode. Three locations were prepared for electrodes by sandpapering and cleaning with acetone: the palmar and dorsal surfaces of the hand and the ventral surface of the forearm about eight inches above the wrist. Double-faced Grass adhesive collars were used to hold the three GSR electrodes firmly in place. The GSR electrodes were attached to flexible shielded leads. Recording was done between palmar and dorsal locations with the forearm as a ground.

Two additional electrodes for presenting the electric shock stimuli were placed close to the GSR ground electrode on the forearm. Location was dictated by the necessity of minimizing stimulus artifact and varied among Ss. These electrodes were also EEG cup electrodes filled with gel, and were held in place by adhesive tape and a wad of gauze bound to the arm with a wide rubber cuff. Care was taken not to occlude circulation in the arm.

During the application of the electrodes E made every effort to engage S in conversation and attempted to put S at ease. The E explained what S's part in the experiment would be and reassured S concerning sources of anxiety which had been mentioned most commonly by Ss in pilot testing. No rigid formulation was used but the following points were always

covered:

(a) S should sit as quietly as possible throughout the experiment, therefore S should try to find a comfortable, relaxed posture to begin with. S would be in the sound proof room (SPR) approximately one hour. S should try not to go to sleep.

(b) The double door to the SPR could be opened from within if at any time S should find it absolutely necessary. Also, an intercom was set up so that E could hear S speak in case S needed to. However, E emphasized that these were emergency precautions and that it was hoped no further communication would take place between S and E after the experiment began.

(c) S should not be surprised or tell E if there were intervals when no stimuli occurred.

The S was then shown into the electrically-shielded SPR (double-walled, Industrial Acoustics Company type 1204, inside dimensions approximately 7' X 7' X 6 1/2')and seated in a padded chair with head and arm rests and adjustable back. During the actual experiment the SPR was dark except for a very faint light which leaked in around the edges of the aluminum foil used to block out the SPR window. After leads were connected to the various recording and shock electrodes E checked the recording apparatus, situated outside the SPR, to make sure that all signals were being properly received.

(2) Recording Apparatus

GSR potentials were led into a differential

preamplifier (Tektronix 122) in the SPR, from which the signal was led outside the SPR to a Tektronix 3A74 4-trace amplifier plugged into one side of a dual-beam oscilloscope (Tektronix 565). Sweep time was set at approximately thirteen seconds. A second trace was used for a time mark with one-per-second pulses from a Tektronix 161 pulse generator appearing as regularly spaced dots on the oscilloscope.

Scalp potentials were led into a second 122 preamplifier in the SPR, thence out of the SPR into a Tektronix 2A61 low-level preamplifier plugged into the other side of the oscilloscope. Sweep time was set at 500 msec. The output of the 2A61 was led into a Nuclear Data ND 800 Enhancetron, and also to a channel of the 4-trace amplifier for the purpose of monitoring 2A61 output to insure that the Enhancetron input range of $\pm$ .5 V was not exceeded. This trace was monitored only at the beginning and end of each experimental session and was not present on the oscilloscope face during photographing of responses. In order to limit DC voltage input to the Enhancetron a capacitor was inserted between the 2A61 and the Enhancetron. The Enhancetron was set for an averaging epoch of 500 msec and maximum scale deflection. The output of the Enhancetron was led into a third trace of the 4-trace amplifier, where it could be displayed at the proper 500 msec sweep time when the corresponding oscilloscope time base was controlled by the Enhancetron time base via the "External Horizontal In." During the actual experiment the sweep time for this

side of the oscilloscope was controlled by the 13-sec. time base setting appropriate for GSR measurements, except for the brief interval following each set of fifteen trials when the averaged response computed by the Enhancetron was displayed on the oscilloscope face and photographed.

A Grass C-4 camera placed approximately twelve inches from the oscilloscope face photographed GSRs and EPs as they occurred (see Plate 1.)

Stimuli

All control equipment for timing and triggering of stimuli was outside the SPR.

Initiation of trials was accomplished by a 3-channel tape programmer (4.75 mm/sec. tape speed) with a standard punched tape for each of the three experimental conditions. Triggering of camera exposures was also controlled by these tapes. Triggering of oscilloscope sweeps, Enhancetron averaging epochs, and stimuli was accomplished by various combinations of Tektronix 160-series units as described below. A trial was initiated when the tape programmer triggered a "master" Tektronix 162 wave-form generator. CS - US and US - CS intervals were controlled by the output sawtooth duration of the master 162 unit.

The CS was a single click, approximately fifty-three db above threshold, produced by a 0.3 msec square pulse from a Tektronix 161 pulse generator fed into an audio amplifier (Eico 20) and thence through the SPR wall into a loudspeaker

(Acoustic Research, Inc., AR2-a) approximately four feet from S's head.

The US was a train of shocks produced by a variable voltage, high internal resistance source which produced constant current output when external resistance varied from 500 to 50,000 ohms (manufactured especially for this study by the Research Instrument Service of the University of Oregon Medical School). The DC square wave voltage output was triggered by a 100-msec train of 5-msec pulses at 100/sec, produced by a series of Tektronix 160-series pulse and wave form generators, and led through the SPR wall to the shock electrodes on S's forearm. A fail-safe series of high sensitivity (2 mA) fuses built into the voltage source, and restriction of all shock and GSR electrodes to the same arm, were designed to protect S against the possibility of unexpectedly high voltage in the shock circuit. The output of the voltage source could be controlled by E by means of a variable dial. Output was roughly exponential starting at .05 mA at a dial setting of zero to a maximum of 1.65 at a dial setting of 100. A graph of the shock output may be seen in Appendix A.

In order to establish the intensity of shock which would be used for the experiment E gave a trial shock to S at a standard intensity gauged to be moderate, and then asked S to rate how annoying it was on a scale ranging through "very, moderately, slightly, not at all." The E discussed the shock stimulus at some length with S, explaining that it was

important for the experiment that the shock not be too weak, and that often the first shock felt disproportionately strong because it was novel. By this means most Ss were persuaded to try two to five additional shocks of increasing intensity until an intensity was reached that was as high as S would accept. In a few cases the shock had to be decreased from the standard intensity. The S was then told that for various reasons such as changes in the electrode contact or alterations of shock presentation between the two shock electrodes, moderate fluctuations in shock intensity might be noticed as the experiment progressed.

Shock stimuli of the order of magnitude used in most experiments with human subjects tend to elicit progressively smaller GSRs when repeated a number of times. A stratagem which has been reported to maintain the reinforcing properties of shock stimuli (Aronson, Hind, and Irwin, 1958; Champion and Jones, 1961; McDonald and Johnson, 1965) is to increase shock intensity in stages. In the present experiment shock intensity was increased three times.The particular trial at which each increase was made varied slightly among Ss. Due to individual differences in reaction to shock, the amount of increase was also varied among Ss, the constant attempt being to maintain GSR to the shock US. The trials at which increases occurred are listed in Appendix A. In order to ascertain that initial shock intensity and subsequent increases were not related to experimental condition, the mean starting shock intensities and increases for each group are compared in Table 1. below.

Table 1.

Mean Initial Shock Intensities & Subsequent Mean Increases for Each Group

		E	B	R
Starting I	(mean)	1.14 mA.	1.14 mA.	1.06 mA.
	range	.25-1.65	.44-1.45	.40-1.65
1st Increase	(mean)	.157	.211	.255
	range	0-.41	.12-.36	0-.57
2nd Increase	(mean)	.118	.128	.120
	range	0-.28	0-.25	0-.30
3rd Increase	(mean)	.108	.084	.105
	range	0-.45	0-.25	0-.25

The reason for entries of zero in the above table is that some Ss started the experiment with the maximum available output of 1.65 mA, so for them no increases were possible. These Ss were about evenly distributed among groups. A record was considered acceptable so long as GSRs to the shock remained evident. Low and high starting intensities can be found in each group. The mean starting intensity for female Ss is lower than for males. Large and small increases are also quite evenly distributed among groups. A complete listing of all shock intensities and increases can be found in Appendix A.

An additional procedure was employed to reduce habituation of the GSR to shock: polarity of the input to the two shock electrodes was reversed six times during the experiment. This probably resulted in a slight difference in the location of effective current since there were two ground electrodes

on the S's arm. This procedure did result in changes in the subjective sensation of the shock, as confirmed by reports from Ss after the experiment, and in many instances by very evident changes in GSR to the first shock administered after polarity was changed.

Although this procedure resulted in effective shock intensity changes in both directions, it was felt that the desirability of adhering to a standard schedule of changes for all subjects, as a control against possible experimenter bias in administering shocks, outweighed the disadvantage that part of the time the polarity change produced decreases in effective intensity. It should be noted that the GSR diminution with repeated shock presentation may be an example of habituation. Decreases in stimulus intensity have been reported to produce dishabituation (see discussion in Thompson and Shaw, 1966). The trials in which polarity was reversed are listed in Appendix A.

Procedure

After initial shock intensity had been established E checked the reception of S's voice over the intercom, cautioned S to be particularly careful to keep the arm and hand bearing the GSR electrode quiet, and closed the SPR doors.

The E allowed two or three minutes to pass before starting the tape which programmed the trials. During this interval most Ss showed a gradually stabilizing GSR base line and decreasing frequency of spontaneous GSRs.

Because considerable time was usually necessary to change the apparatus from one experimental condition to another, it was not practicable to assign Ss to conditions after they were closed in the SPR. Instead the order of obtaining the thirty different records (ten in each of three conditions) was pre-determined randomly and each record was in its turn obtained from whatever S happened to be available on that day. Some departures from the original random ordering occurred occasionally because of inadequate time for the necessary apparatus changes between conditions. These departures are not believed to have operated to select Ss in any systematic way. In addition the attempt was made to equate numbers of male and female Ss between groups. The resulting distribution was as follows:

Group E - - - 5 M, 5 F

Group B - - - 6 M, 4 F

Group R - - - 5 M, 5 F

The stimulus schedules for each group were as follows:

Phase I . . . Baseline measurements; habituation training

All groups received fifteen trials of 0.3 msec click alone. Mean intertrial interval (ITI) was 45 sec., obtained by means of a random sequence of equal numbers of 35, 45, and 55 sec. ITIs.

Phases II, III, and IV . . . Acquisition

Group E (forward conditioning) . . . fifteen trials as follows:
ten trials of click CS followed after 6 sec. by shock US, with five CS - alone trials randomly interspersed. The US was a 100 msec train of 5msec pulses at 100/sec. The restriction was made that the first trial was always reinforced. Average ITI was the same as in Phase I.

Group B (backward conditioning) . . . fifteen trials as follows:
ten trials of shock US followed after 6 sec. by click CS, with five CS - alone trials and all other details the same as for Group E.

Group R (random unpaired) . . . fifteen clicks and ten shocks presented in a random sequence during the same total time as that required for the fifteen trials of Group E and B (11.25 min.) Average interstimulus interval (ISI) was thus 27 sec., obtained from a random sequence of equal numbers of 22, 27, and 32 sec.ISIs.

Phase V . . . Extinction

All groups received fifteen trials of click alone. Average ITI was 45 sec., obtained by means of a random sequence of equal numbers of 35, 45, and 55 sec. ITIs.

After each phase an interval of approximately one and one-half minutes was necessary to allow E to change the oscilloscope controls so that the Enhancetron average of the preceding fifteen responses could be displayed and photographed. The

average was then "erased" and averaging begun anew for each phase.

An additional restriction was placed on the random schedules of shock presentation:

The last trial in Phase II was reinforced
The first trial in Phase III was not reinforced
The last trial in Phase III was not reinforced
The first trial in Phase IV was reinforced
The last trial in Phase IV was reinforced

This restriction was intended to prevent the prediction by S of shock probability on the trial following the relatively long intervals between phases.

It will be noted that the reinforcement schedule used was a partial one, with thirty of the forty-five acquisition trials being reinforced (67%). The total time required for the overall total of seventy-five trials, including the intervals between phases, was sixty-five minutes.

At the end of the experiment the S was relieved of the various electrodes, paid, and asked not to tell other prospective Ss about his experience.

III. RESULTS

(1) GSR Conditioning

The magnitude of GSR was defined for this experiment in units of potential change from base line. A typical GSR is shown in Plate 1. A latency interval was established as follows: for all groups, GSRs occurring between .5 second after CS onset to 6.5 seconds after it, were counted. Because of the six second CS - US interval, in the trials with paired

Plate 1. Photographic Record of a Typical Trial

The GSR may be seen in the lowest trace. Click CS occurred at the beginning of the sweep. Shock artifact (short vertical line imposed on GSR trace) may be seen at 6 sec. after the start of the 13-sec. GSR trace. The EP (not averaged) may be seen in the middle trace. Sweep time for the EP is 500 msec. The dots in the upper trace are 1-sec. time marks. Positivity is upward for both GSR and EP.

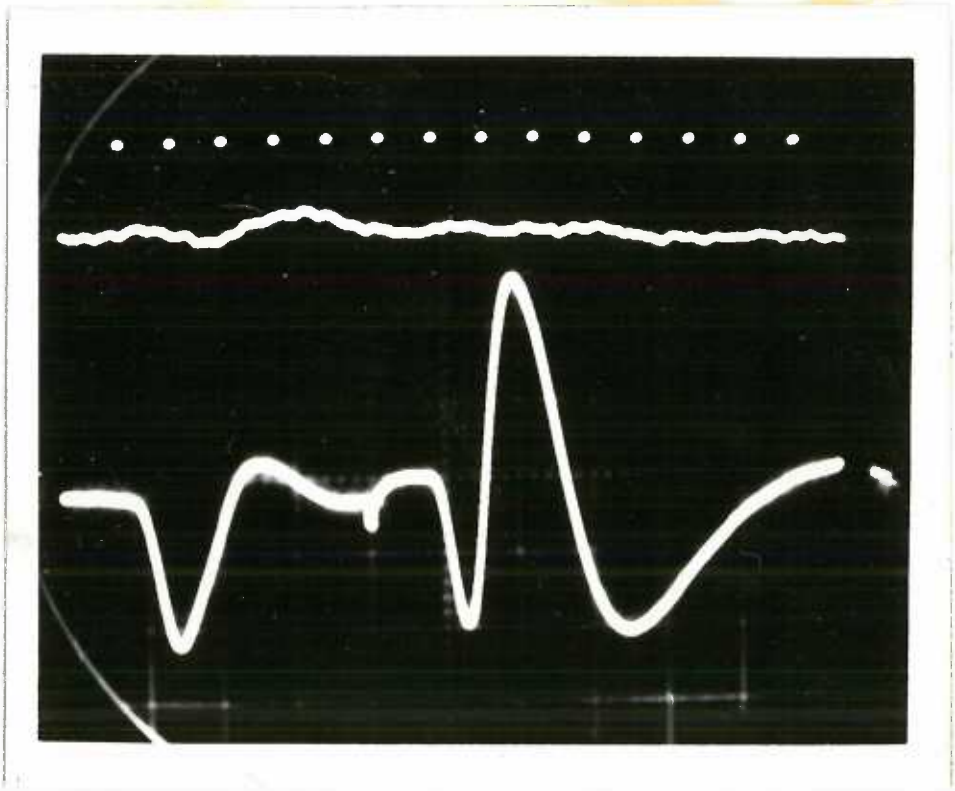

Plate 1. Photographic Record of a Typical Trial

CS - US, the US occurs .5 second before the latency interval ends. The latency of GSR to the shock US observed in this laboratory in a large number of Ss has never been less than approximately one second, always considerably longer than the .5 second by which the CS scoring interval overlaps the US.

The most characteristic GSR is biphasic, negative-positive (reference palm to dorsum). However, many other types of response occur including monophasic in either direction or polyphasic. Scoring of magnitude was therefore carried out as follows:

(a) biphasic R's . . . sum of amplitudes of positive and negative excursions from baseline

(b) monophasic R's . . amplitude of positive or negative excursion from baseline

(c) polyphasic R's . . whichever sum of amplitudes of consecutive positive-negative pairs occurring within allowed latency interval was largest.

After recording all Ss' raw scores the data for Phase I (CS - alone) was examined for signs of habituation. In nearly all cases GSRs decreased markedly over the fifteen trials. In some cases the decrease was erratic, and in a few cases there were such marked fluctuations that a systematic change in magnitude was not discernible. The means and the medians of the last six trials for each S were compared and the means were found to be the more conservative measure of the last six trials, providing fewer zero scores against which to evaluate later increases due to conditioning. This "habituation" score was

Figure 1. Mean GSR Magnitude for Each Group in Each Phase

Mean scores for each group in each Phase were obtained by combining, for the 10 subjects in a given group, each subject's mean score on the 5 test trials in a given Phase. Each subject's habituation score was subtracted from all later scores and a constant of 100 added. Figure 1 shows that there appear to be marked differences between groups, although analysis of variance yielded no significant differences.

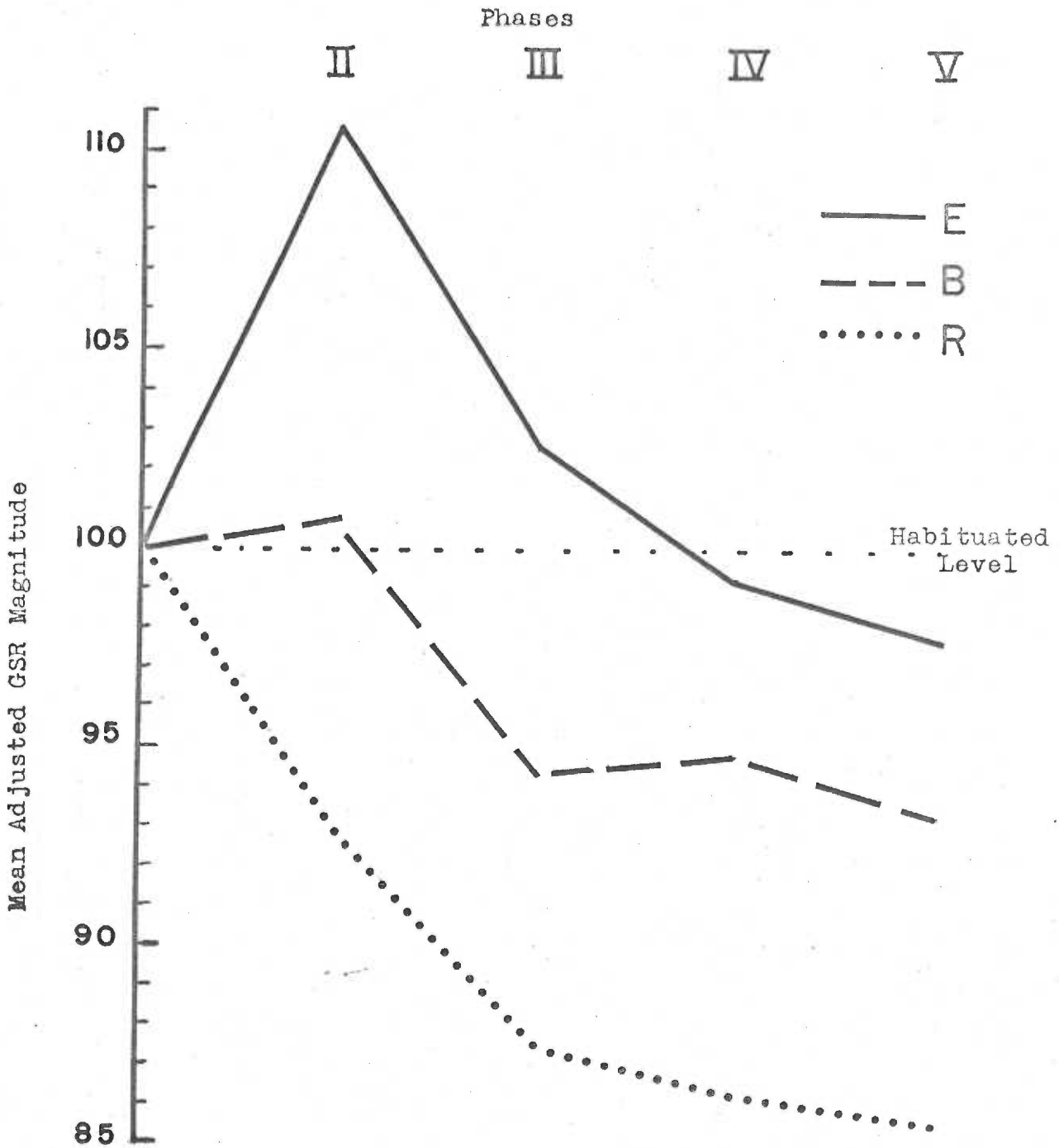

Figure 1. Mean GSR Magnitude for Each Group In Each Phase After Habituation

then subtracted from all subsequent scores for each S.

Phases II, III, and IV yielded five trials each in which the CS occurred alone for the E and B groups. The R group received fifteen trials of CS alone in each Phase, so for the purpose of comparing them to the other groups, five trials in each Phase were selected which corresponded most closely to the times at which the five CS-alone trials occurred for the B and E groups. Thus for each group there was a total of fifteen trials during the acquisition period for which GSR scores could be compared.

In extinction, Phase V, each S's scores in the fifteen trials of CS alone were averaged in blocks of three, yielding five extinction scores. These scores, like those in acquisition, were adjusted by subtracting the"habituated" score. The raw scores may be seen in Appendix B.

Despite the use of adjusted scores, variability between and within individuals was pronounced. Computation of each S's mean GSR magnitude for Phases II through V produced somewhat more stable scores. These individual mean scores were submitted to analysis of variance using a 3 X 4 repeated measures design (Winer, 1962), the results of which are summarized in Table 2. The only significant effect was that due to Phase. As may be seen in Figure 1., all groups show a downward trend after Phase II.

Figure 2. shows a somewhat different comparison between groups. To plot this figure the scores for the ten Ss in a

Source	df	MS	F
Between Ss	29		
Groups	2	2091	2.318
Ss w. groups	27	902	
Within Ss	90		
Phases	3	508	3.722*
Groups X Phases	6	27.5	.202
Phases X Ss w. groups	81	136.5	

*p < .05

Table 2. Analysis of Variance of Mean GSR Magnitude

given group were combined on each "trial" (actually, fifteen acquisition trials plus five blocks of three extinction trials each.) Again, all scores had been adjusted for the "habituated" level. Both the means and the medians were computed for each group of ten scores. The means and medians for each group are presented in Table 3. Figure 2. shows the group medians on each test trial. Inspection of Figure 2 suggests that real differences between groups exist, at least during Phase II, but are prevented from reaching significance in the analysis of variance by the high degree of variability present.

To further test the possibility of differences between groups, a Kruskal-Wallis test (Siegel, 1956) was performed using individual S's scores. The scores used were the mean GSR magnitude for the five trials in a given Phase for each S. The comparison thus involved three sets (one set for each group) of ten mean scores for a given Phase. A significant difference between groups was found (one-tailed, $p < .02$) for Phase II only. Table 4 a. summarizes the Kruskal-Wallis test for each of the Phases II through V.

The Mann-Whitney U test (Siegel, 1956) was used to test for differences between all possible pairs of groups (E : B, B : R, E : R) in each Phase. The values for the U statistic are given in Table 4 b. The only comparison which proved to be significant was that between E and R in Phase II ($E > R$, $p < .02$, one-tailed). Inspection of Figure 2. indicates that whatever differences there are between the three groups are

Figure 2. Group Median GSR Magnitude

GSR magnitudes for each group for each trial were computed according to the procedure described in Table 3. Group E (solid line) shows significantly greater GSR magnitude than Group R (dotted line) throughout Phase II. The ranking E > B > R is significant for test trials 2, 3, and 4 in Phase II. After Phase II, GSR magnitudes for the three groups do not differ significantly.

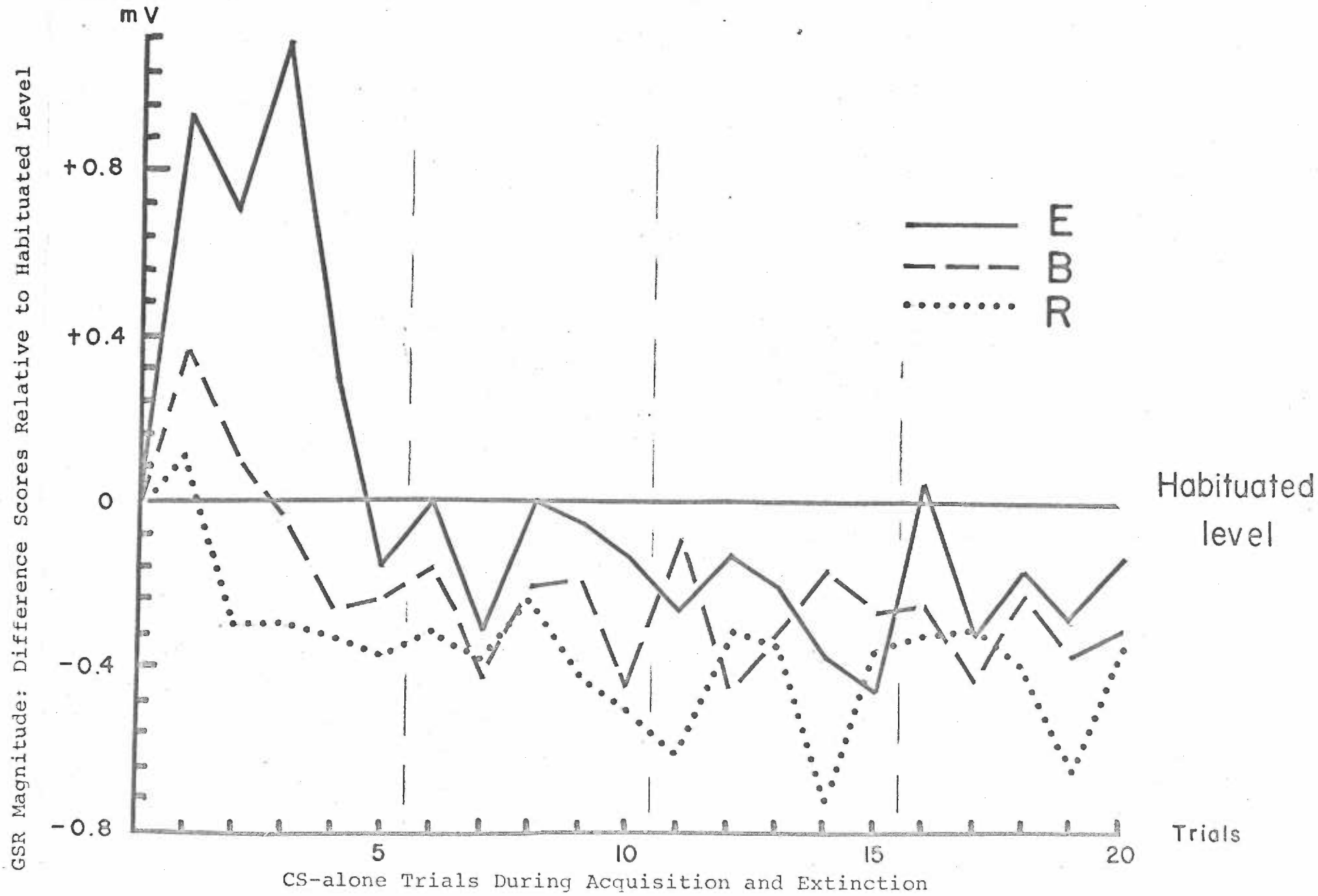

Figure 2. Group Median GSR Magnitude

Table 3

Comparison of group mean and median GSR magnitude for each test trial

Means and medians were computed by combining scores on a given test trial for the 10 Ss in a given group, except for Phase V where mean scores over 3-trial blocks were combined for the 10 Ss in a given group.

Before computing means and medians, each S's scores in Phases II, III, IV, and V were adjusted by the subtraction of the S's habituation score (mean GSR magnitude on last 6 Phase I trials) and the addition of the constant 100 to each score.

	Groups	E		B		R	
	Trials	Mean	Median	Mean	Median	Mean	Median
	1	108.74	111.8	110.7	105.5	99.55	101.5
	2	114.73	108.9	101.3	101.3	93.10	96.1
Phase II	3	118.98	113.9	99.0	99.7	93.70	96.05
	4	106.93	103.7	97.15	96.6	91.05	95.9
	5	103.73	97.9	95.10	97.1	85.35	95.35
	6	101.88	99.7	96.5	98.0	86.75	96.05
	7	100.63	96.1	89.6	94.6	88.60	95.3
Phase III	8	103.58	99.9	98.7	97.5	87.45	97.0
	9	103.43	99.4	98.0	97.55	90.38	94.85
	10	103.08	98.4	88.95	94.3	83.45	93.75
	11	102.93	96.7	98.25	98.95	87.40	92.35
	12	100.93	98.4	89.40	94.3	86.75	96.05
Phase IV	13	102.33	97.4	95.35	96.0	86.00	95.6
	14	95.08	95.4	95.25	98.0	81.15	91.0
	15	97.03	94.3	94.40	96.75	89.45	95.6
	16	100.09	100.55	92.98	96.9	84.84	96.0
	17	96.71	96.0	92.93	94.65	85.94	96.1
Phase V	18	98.25	97.8	93.93	97.25	86.18	95.1
	19	96.38	96.4	93.13	95.45	82.43	92.1
	20	96.11	98.3	92.41	96.25	88.32	95.65

concentrated within Phase II. In order to examine GSR magnitude during Phase II with greater precision, the Whitney extension of the Mann-Whitney test[1] was applied to the three sets of individual scores on each of the five test trials in Phase II. This test is applicable where three independent samples are to be compared and an a priori decision regarding their relative ranks can be made. Since much evidence exists that Ss receiving forward conditioning can be expected to give larger GSRs than those of control Ss, and there is also evidence that backward conditioning of the GSR produces a smaller but still noticeable increase in response magnitude (Champion and Jones, 1961), the prediction could be made a priori that GSR magnitudes in the three groups would be ranked E > B > R. The Whitney test showed this ranking to hold for test trials 2, 3, and 4 in Phase II. Table 4 c. summarizes the values obtained for U and V for each trial. On trials 2, 3, and 4 the predicted ranking is significant with $p < .01$.

A significant difference between groups, in particular during test trials 2, 3, and 4, does appear to have resulted from the experimental procedure. The data illustrated in Figure 2. and Figure 3. also suggest that there is a significant change in GSR magnitude over the course of the experiment. All groups tended to decrease their response level as the experimental session progressed. A Friedman two-way analysis

[1] Referred to in Siegel (1956), and obtained for use in the present paper by personal communication from Dr. F. Robert Brush.

Phase	H	p
II	8.65	$<.02$
III	2.56	$<.30$
IV	1.01	$<.70$
V	1.28	$<.70$

Table 4 a. Summary of Kruskal-Wallis Test on Individual S's Scores

Phase	E : B	E : R	B : R
II	37	16*	31
III	39	30	38
IV	40	38.5	44
V	38	37	45

*$p < .01$

Table 4 b. Summary of Mann-Whitney U Test on Individual S's Scores (one-tailed; $n_1 = n_2 = 10$)

Trial	U	V	p
1	60	47	$<.10$
2	68	34	$<.01$
3	65	25	$<.01$
4	65	35	$<.01$
5	61	45	$<.10$

Table 4 c. Summary of Whitney Extension of the Mann-Whitney U Test for Test Trials in Phase II

of variance on the median scores for each group in each trial (the scores presented in Table 2.) yielded a significant overall trials effect with $p < .02$. The Friedman test bears out the finding in the analysis of variance of the GSR scores mentioned earlier, that there was a significantPhase effect. The use of the non-parametric test increases the significance level for the trials effect from $p < .05$ (analysis of variance) to $p < .02$ (Friedman test).

The persistent downward trend in the GSR data suggests another hypothesis to test: that GSR level at the end of Phase V decreased relative to the GSR "habituated" level. The possibility that further "habituation", beyond that defined by the last six Phase I scores, occurred in many Ss was tested by the use of the Wilcoxon matched-pairs signed-ranks test. The mean of the last six Phase V scores was subtracted from the mean of the last six Phase I scores for each S. This gave three sets of ten difference scores, corresponding to the ten Ss in each of the three groups. Phase V GSR magnitude did not differ significantly from Phase I GSR magnitude for any of the groups. ,

To summarize the GSR data, groups E, B, and R could be differentiated on the basis of magnitude of GSR but only during Phase II. The magnitude changes accompanying conditioning did not endure beyond Phase II, and in fact within that interval were significant only for trials 2, 3, and 4.

Plate 2. Averaged EPs from a Typical Record

The five consecutive average EPs recorded from subject E_8 after each Phase in the experiment illustrate the intra-subject reliability which EPs exhibit in most subjects. In this record the negative peak N_1 at approximately 90 msec and the positive peak P_2 at approximately 140 msec are well marked. Positivity is up. Calibrations, 10 microvolts and 100 msec.

Subject Eg (forward conditioning)

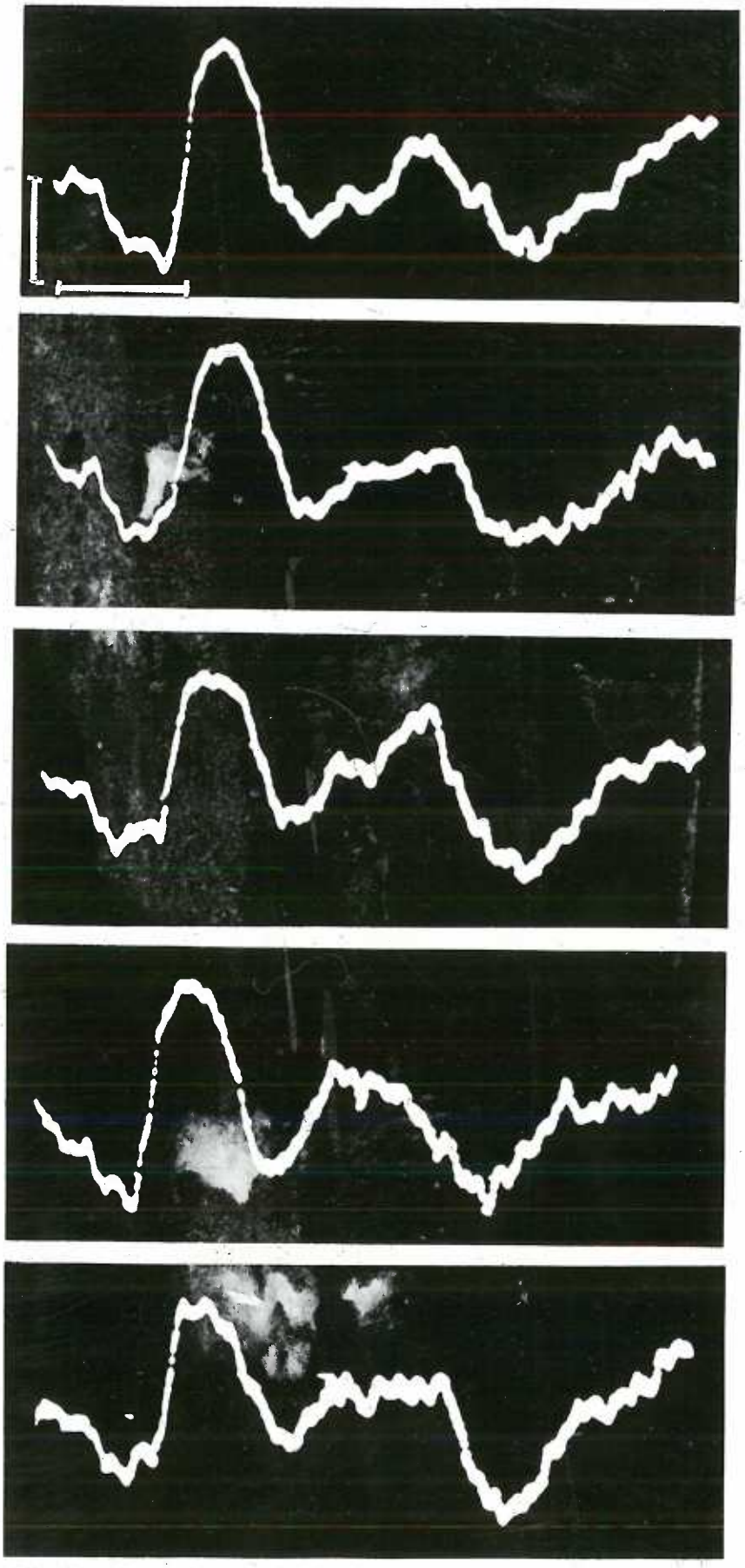

Plate 2. Averaged EPs from a Typical Record

(2) EP Measurements

The EP to click in most, but not in all, human Ss shows a pronounced negative peak at about 80 - 100 msec followed by a large positive peak at about 150 - 200 msec (Davis, Engebretson, Lowell, Mast, Satterfield, and Yoshie, 1964; Geisler, Frishkopf, and Rosenblith, 1958; Williams, Morlock, Morlock, and Lubin, 1964). A typical re cord from the present experiment may be seen in Plate 2. The negative and positive peaks mentioned above are quite noticeable in this and many other records. Therefore, these two components were selected to be measured for latency and amplitude.

Latency was determined by measuring the time from stimulus onset to the negative or positive peaks described above. A representative EP is shown in Figure 3 a. with peaks numbered for identification. Although this EP shows a regular configuration even beyond P_2, a number of EPs do not. It appears from inspection of many EP records that variability in wave form, latency, and amplitude increases with time from stimulus onset.

Measurements of latency of N_1 and P_2 are confounded by amplitude changes. Occasionally two peaks will appear in the approximate location where one peak is found. In such cases the problem is to decide which one to measure for latency. The procedure adopted here was to set up intervals for the group of EP records

as a whole which contained all of the well-defined peaks of a given designation. This interval was then examined in all the questionable records, and the largest peak occurring in that interval was arbitrarily designated the one to be measured for latency.

Figure 3.(b) shows latency measurements for an idealized EP.

Amplitude measurements were of peak-to-peak amplitudes. As may be seen from Figure 3. (b), the amplitude labelled "N_1" is the negative-going change from P_1 to N_1 ; "P_2" is the positive-going change from N_1 to P_2.

(A) EP amplitude changes:

As with the GSR raw scores, EP raw scores exhibit much variability. The EP raw scores are presented in Appendix C. Since amplitude in Phase I appeared to be highly correlated with amplitude in all later Phases, and initial mean amplitudes for the three groups were quite different, analyses of covariance (Winer, 1962) were performed with Phase I scores as the covariate, using a repeated measures design. Table 5. contains summaries of the covariance analyses. For N_1 the only significant effect was that due to Phases, which may be seen in Figure 4, (b) as the tendency for all groups to decrease over time. For P_2 both the effect due to Phases and the Group X Phases interaction were significant. Inspection of Figure 4. (b) suggests that the significant interaction may well be due to the fact that Group E decreases most overall, Group R

Figure 3 a. Components of an Averaged EP

An actual averaged EP is traced in this figure. A negative peak (N_1) at approximately 100 msec and a positive peak (P_2) at approximately 200 msec are typical features occurring at comparable latencies in the EPs of other subjects.

Figure 3 b. Measurement of EP Components

The vertical line at the left represents t_o or stimulus onset. N_1 and P_2 latencies are measured from this ordinate. Amplitude of N_1 and P_2 are measured peak-to-peak as shown.

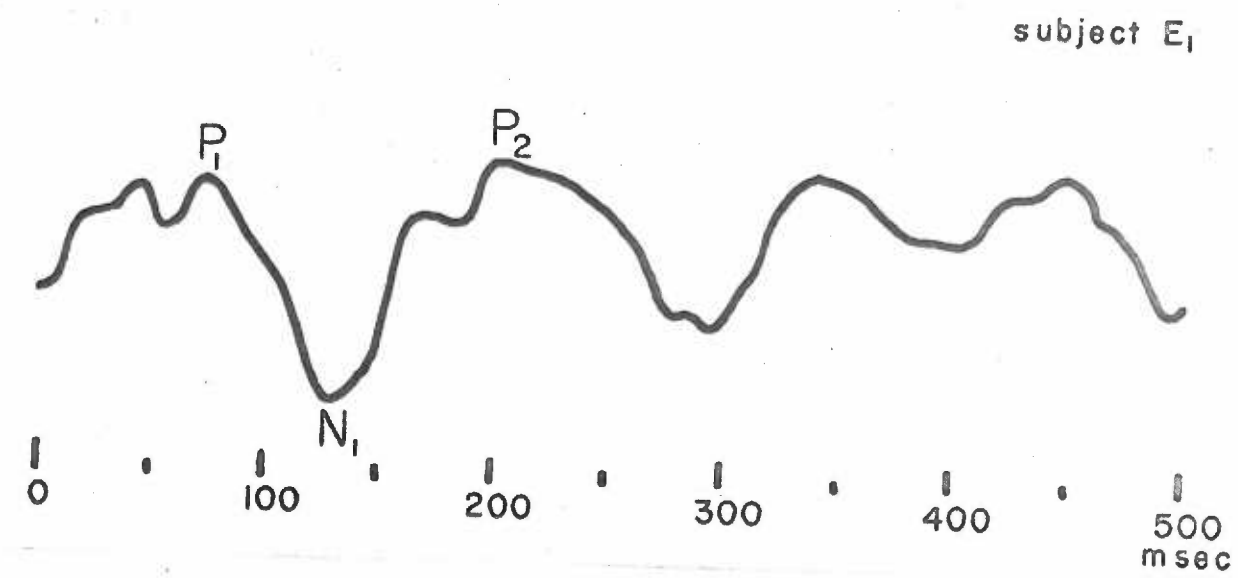

Figure 3 a. Components of an Averaged EP

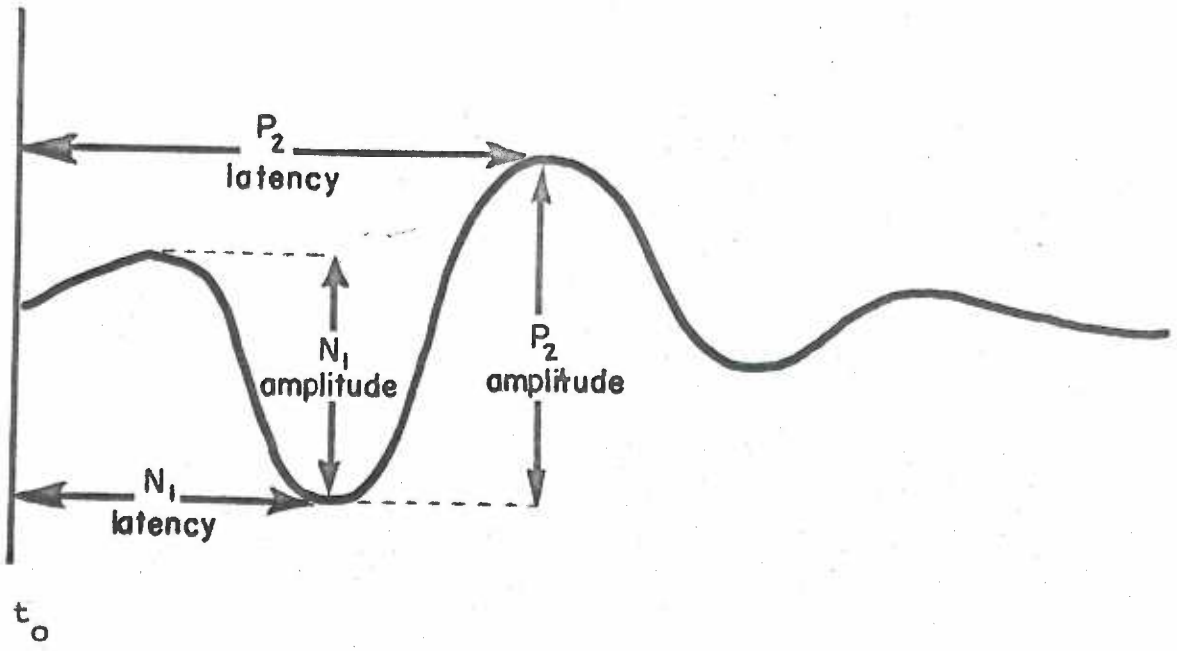

Figure 3 b. Measurement of EP Components

Source	df	MS	F
Between Ss	29		
Groups	2	330.5	1.378
Ss w. groups	27	239.7	
Within Ss	90		
Phases	3	41.3	2.193
Groups X Phases	6	.5	.027
Residual	81	18.83	
Between Ss (adjusted)			
Groups	2	138.5	.796
Ss w. groups	26	173.9	

Table 5 a. Analysis of Covariance for N_1 Amplitude

Source	df	MS	F
Between Ss	29		
Groups	2	460.5	.756
Ss w. groups	27	609	
Within Ss	90		
Phases	3	190	25.2 ***
Groups X Phases	6	28.7	3.82***
Residual	81	7.52	
Between Ss (adjusted)			
Groups	2	81.5	.371
Ss w. groups	26	219.5	

*$p < .001$

Table 5 b. Analysis of Covariance for P_2 Amplitude

Figure 4 a. Group Mean Amplitude of N_1 and P_2

This figure incorporates raw scores and illustrates the difference between the three groups in initial amplitudes of both N_1 and P_2. The overall downward trend throughout the experimental session may be seen in both components.

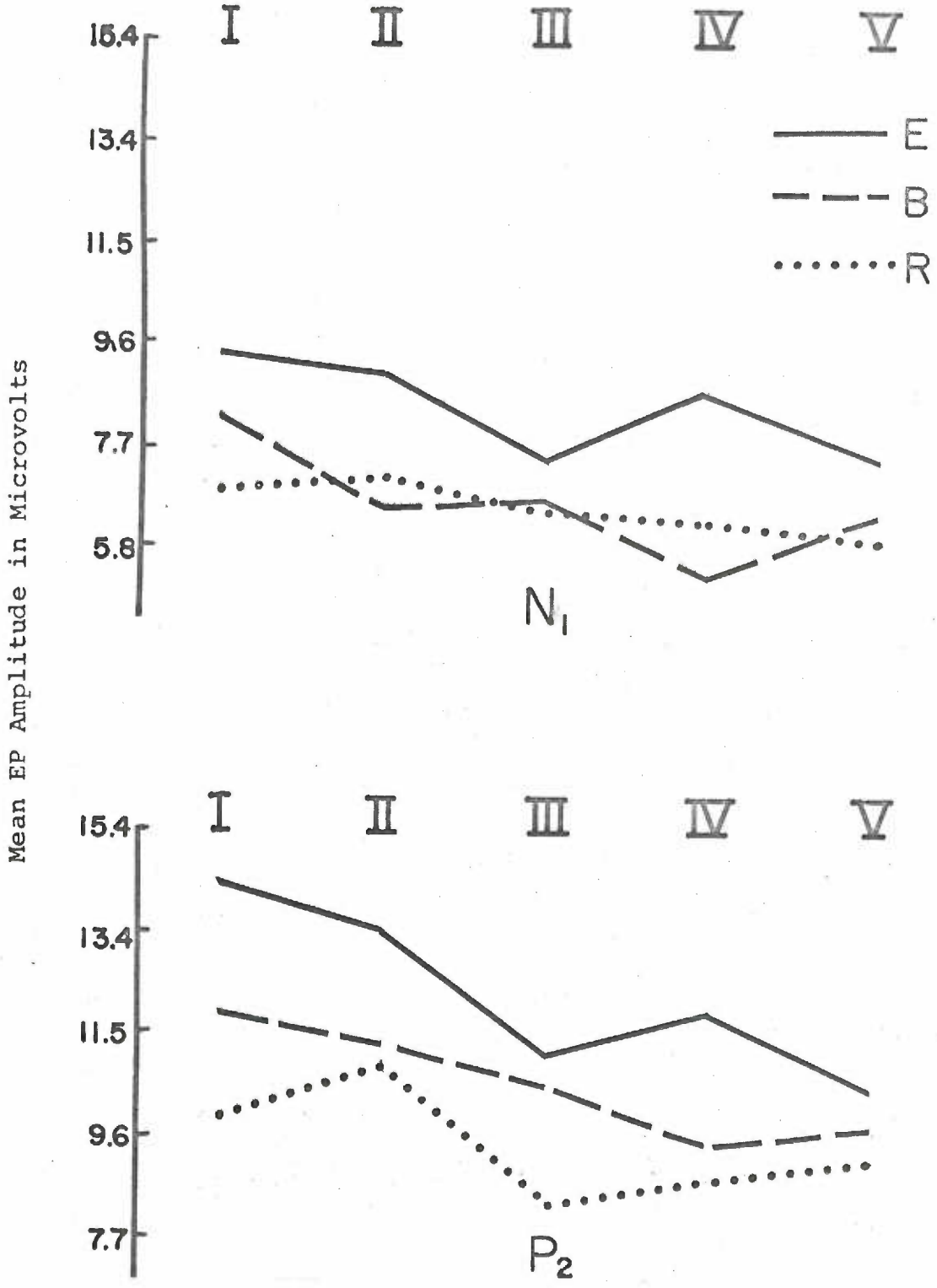

Figure 4 a. Group Mean Amplitude of N_1 and P_2

Figure 4 b. Percentage Amplitudes Relative to Amplitudes in Phase I

In Figure 4 b. the opposite tendency of the amplitude change for Group E compared to that of Group B is well marked between Phase I and Phase II. After Phase II all groups show an overall downward tendency, although there is some evidence in the P_2 amplitudes of differential responding between Group E and Group R as late as Phase V.

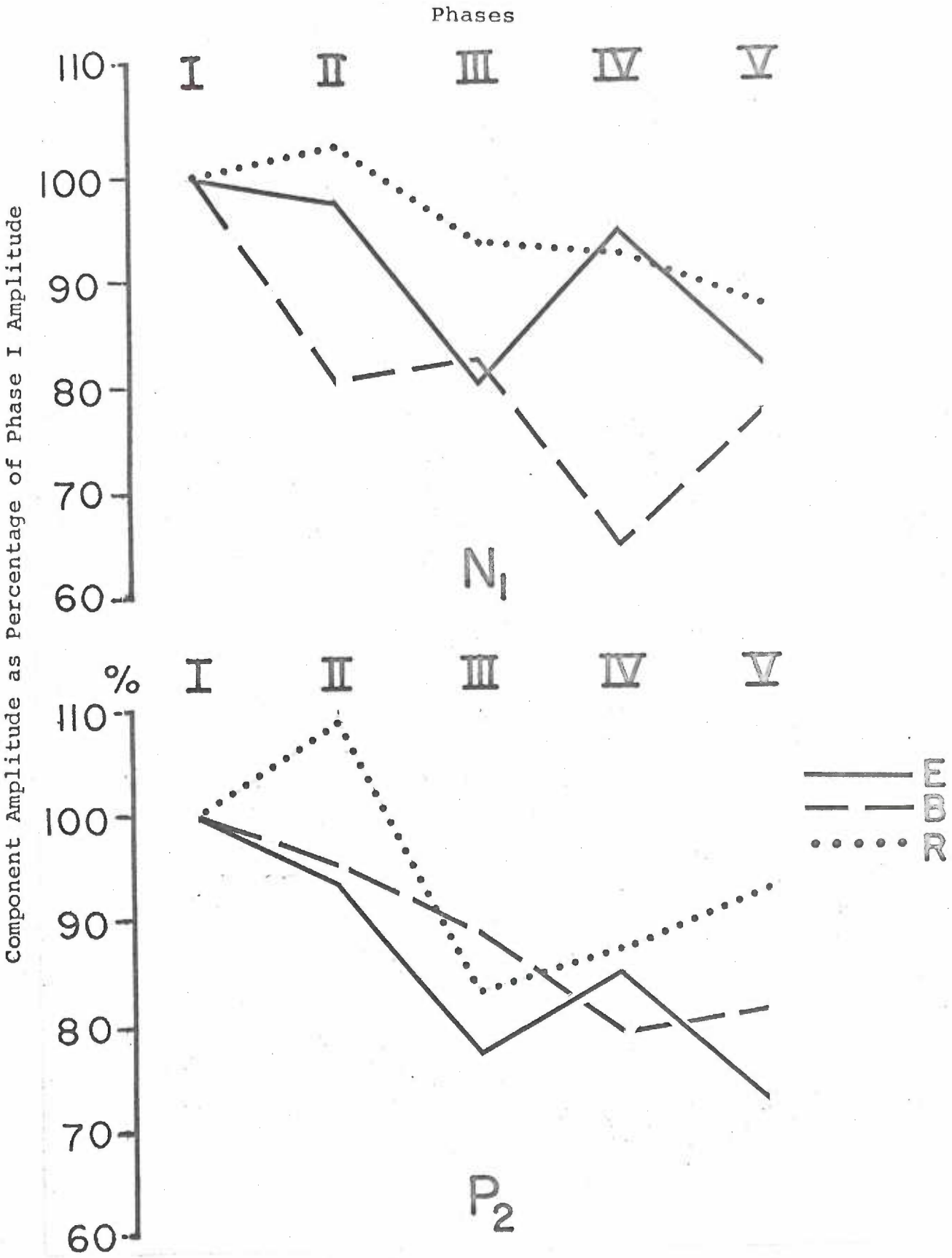

Figure 4 b. Percentage Amplitudes Relative to Amplitudes in Phase I

appears first to increase and later to decrease, and the B Group appears to lie in between.

In order to test for correlation between GSR and EP mean magnitude for each Phase, the adjusted GSR scores for individual Ss (Figure 1.) were compared with individual S's P_2 amplitude scores for Phase II through V. P_2 scores for the correlation were obtained by subtracting the Phase I scores from all later scores for a given S and calculating the percentage change. Since many changes were negative (i.e., decreases in amplitude) a constant of one hundred was added to make all scores positive. The Pearson product-moment correlations between EP and GSR amplitude for each group singly (forty pairs of observations) were:

E : r = .10

B : r = -.25

R : r = .004

For the combined group (one hundred twenty pairs of observations) the overall r = .123. None of the correlations reached significance.

(B) Latency changes:

Analyses of covariance for N_1 and P_2 latencies yielded no significant differences. There were increases and decreases in latency for all Ss between various Phases, with no systematic trend apparent. Summary tables for these analyses are presented in Table 6.

Source	df	MS	F
Between Ss	29		
Groups	2	998	1.11
Ss w. groups	27	898	
Within Ss	90		
Phases	3	158	1.53
Groups X Phases	6	161	1.56
Residual	81	103	
Between Ss (adjusted)			
Groups	2	142	.366
Ss w. groups	26	388	

Table 6 a. Analysis of Covariance for N_1 Latency

Source	df	MS	F
Between Ss	29		
Groups	2	6189	4.604*
Ss w. groups	27	1344	
Within Ss	90		
Phases	3	136.7	1.314
Groups X Phases	6	229.5	2.207
Residual	81	104.0	
Between Ss (adjusted)			
Groups	2	2546	3.02
Ss w. groups	26	843	

* $p < .05$. Note that adjustment for the covariate removes the significant effect due to Groups.

Table 6 b. Analysis of Covariance for P_2 Latency

IV DISCUSSION

The marked enhancement of the GSR in the forward-conditioning group (E) during the first fifteen acquisition trials appears to constitute significant evidence of conditioning. It was originally hoped that the unusually long (six second) CS - US interval in a trace-conditioning paradigm would slow the course of acquisition sufficiently to maintain the increasing trend in the GSR over more than fifteen trials. This hope was not fulfilled, as may be seen from Figures 1. and 2. The transient nature of response enhancement seen here seems to be characteristic of many GSR conditioning experiments. In a study by Kimmel (1959) differences in GSR level of the conditioning groups relative to the control groups lasted approximately ten trials. In another study (Stewart, Stern, Winokur, and Fredman, 1961) a similar tendency was found for the conditioned response to decrease well before the sixteenth trial was reached, at which point any differentiation between control and conditioning groups had nearly disappeared. In general in GSR conditioning a relatively small number of trials constitutes the total acquisition period (Aronson et al, 1958; Champion and Jones, 1961; McDonald and Johnson, 1965; Prokasy, Fawcett and Hall, 1962). The massing of all conditioning trials (not to mention habituation and extinction) within the relatively short space of one hour may be an important factor in the rapid decline of the response . In at least one study (Kimmel, 1964) little decline in GSR level was reported when

acquisition was spread over a number of days. It would have been interesting to see whether there were differences between the three groups in this study in the level of GSR responding to clicks on the day following conditioning. However, in any experimental design there are limitations which must be accepted as inevitable. The use of human volunteers introduces a severe restraint on the number and duration of experimental sessions.

Since differences in GSR responding between the three groups were concentrated within the span of fifteen acquisition trials the burden of demonstrating correlates in the EP falls primarily upon one data-point: the averaged EP measured at the end of Phase II (Figure 4.) Here, again, is a limitation in the method which may be unavoidable. In view of the transient nature of the GSR changes, a finer resolution of the time course of EP changes is needed than is afforded by the necessity of combining fifteen EPs in a single average response.

However, the fact that the forward conditioning group (E) showed a tendency to decreased EP while the control group (R) showed the opposite tendency during the first fifteen trials is highly suggestive that the operation of pairing two stimuli has some consequence for the EP. It is possible that other responses in addition to the GSR became conditioned during the 45 acquisition trials. Various muscular responses which were not monitored, such as a tensing in preparation for shock or a respiratory movement, could have become conditioned. The

existence of such undiscovered CRs might well account for continued differences in P_2 amplitude between Group R and E which appear to be evident as late as the extinction phase (see Figure 4.) and which undoubtedly contribute to the significance of the Groups X Phases interaction.

Examination of EP correlates of attention as defined by behavioral orienting responses in a report by Thompson and Shaw (1965) revealed an inverse relation between amplitude of association cortex EPs and degree of attention. This result seems compatible with the ranking of P_2 amplitudes at Phase II, where E is lowest (corresponding to the most attentive in the Thompson and Shaw rankings) and the R Group is highest. However, it does not seem likely that EPs for all groups would tend to decrease over the entire experimental session if attentiveness is inversely related to the EPs observed here. In fact most Ss found the hour-long session dull. There does not seem to be good reason for supposing that any of the Ss would be more attentive at the end of the session than at the beginning. Of course, the possibility that some other factors (such as increase in electrode resistance) produce progressive attenuation of EP magnitude cannot be excluded. The EPs measured by Thompson and Shaw were of shorter latency (fifty-sixty msec) than those measured in this experiment, and were measured in cats. This does not rule out the possibility that longer-latency EPs from association cortex in human Ss bear a similar inverse relation to attention and are what is being

measured by the scalp electrode in the present case. Association responses of some sort are likely candidates since the electrode was located over non-specific areas of cortex.

Other studies of attentive behavior, in human subjects with electrode locations similar to that used in the present study, have found that EP amplitudes in the 100 - 500 msec range increased with heightened attention (Davis, 1964; Haider, Spong,and Lindsley, 1964; Satterfield, 1965; Spong, Haider, and Lindsley, 1965). "Attention" however was defined by a different sort of operation, namely by tasks which required vigilance or selective responding to certain stimuli but not to others, as in counting particular stimuli or judging their intensity. If these results are compared with the EP levels at Phase II in Figure 4. they would suggest that the R Group is paying the most attention to the clicks and the E Group is attending the least. A reasonable case could be made for this suggestion on the grounds that the R Group constantly tries but never succeeds in finding any pattern or predictability to the stimuli. For the E and B Groups this question is no doubt settled early in the acquisition period. At this point we must concede that evidence of conditioning is not adequate data to support an inference in either direction regarding attentiveness. It should be emphasized that entirely different neural, as well as behavioral , mechanisms may be operating in the three cases of orienting toward a stimulus, selectively attending to it for the purpose of counting it,

or developing a conditioned GSR to it.

At least it is clear from the data of this study that the increase in EP found in the animal studies mentioned earlier did not occur in human GSR conditioning. There are many possible reasons for this difference. The evoked response being measured from the scalp electrode may not be the same response as that recorded by Galambos and his associates. Although they obtained a similar wave-form configuration, (Galambos and Sheatz, 1962; Galambos et al, 1956) with the same latency range for both positive and negative components as the components measured in this study, it must be remembered that their electrodes were implanted directly in primary auditory cortex or in sub-cortical areas, while in this study the electrode was very far from those locations. Despite the relatively great distance between scalp electrodes and the cortex, a number of investigators who have studied the resemblance between scalp recordings at a given location and cortical recordings taken at the same spot have found very close resemblances in the EPs for various sensory modalities, except that the cortically-recorded EPs were larger. These investigators have concluded that the scalp electrode samples essentially the same electrical activity as the electrode directly on the cortex (Chatrian, Petersen, and Lazarte, 1960; Geisler et al, 1958; Giblin, 1964; Katzman, 1964). This would imply that in the present study the EPs observed are generated in the non-specific cortex beneath the electrode, i.e., near

the vertex. Although several studies have suggested that potentials from eye, ear, or cervical musculature may be prominent contributors to EPs measured outside the scalp, (Bickford, Jacobson, and Cody, 1964; Davis et al, 1964) these muscle potentials are not thought to affect the later portions of the EP with which we are concerned in the case of N_1 and P_2 (Davis et al, 1964; Domino and Corssen, 1964).

Another major source of difference between the results of Galambos et al and those obtained in this study may be procedural: the number of pre-conditioning or habituation trials. Galambos and Sheatz (1962) report that they continued habituation training for many days if necessary until no further decrease in EPs could be observed. Decreases in EP as a result of habituation have not been conclusively demonstrated, because of much conflicting data not all of which is adequately controlled (cf. the discussion in Thompson and Spencer, 1966). Whether the EP decrease reported by Galambos and Sheatz is truly habituation of the EP is open to question. Nevertheless, in their laboratory decreases in EP coincident with habituation training have been observed in a large number of animals, and conditioning was apparently never begun until the decreasing trend had reached an asymptotic level. It is possible that conditioning did not produce increased EPs in the present study because the Ss were not given a comparable number of pre-conditioning trials. The downward trend between Phases II and V (Figure 3. and 4.) suggests that if habituation is in

fact represented by decreasing EPs, all groups continued to habituate throughout the experiment regardless of conditioning. However, the Figures would appear to show differences between groups in the rate of the hypothesized habituation.

Differences in habituated level seem a very likely factor underlying the different EP tendencies described. There is accumulating evidence that human EPs to various types of stimuli decrease when the stimulus is monotonously repeated over a long time (Giblin, 1964; Haider, et al, 1964; Walter, 1964). However, the requisite control involving EP measurement at the beginning and end of an equally long interval without repeated stimulus presentations in between has not been run. If lengthy habituation training is in fact necessary before reliable changes due to conditioning can be distinguished, the determination of human EP changes specific to conditioning may have to await the cooperation of subjects willing to undergo extremely long recording sessions.

In the study of Gerken and Neff (1963) it will be recalled that their pre-conditioning series was also limited. They did not continue habituation training until a criterion of maximally decreased EP was reached, but gave each animal a pre-determined number of habituation trials. They found both increases and decreases in EPs during habituation, with opposite changes occurring at different electrodes in auditory cortex in the same animal. After pre-conditioning, the animals which received random unpaired click and shock

presentations did not show decreasing EPs to clicks as might be expected if further habituation were occurring during this time. Instead they showed increases as large or larger than those found during conditioning in other animals. This result is consistent with the changes for the R Group (Figure 4.) in this study. This suggests that sensitization may play a large role in producing increased EPs in the conditioning studies which have reported increases.

The increases in EP during conditioning which resulted in the Gerken and Neff study can be reconciled with the results in the present study if we look only at individual records. The former study included only two animals that received pre-conditioning followed by classical conditioning. Among the ten human Ss in this study who received forward conditioning, three showed increased EPs after Phase I in contrast to the overall decrease for the group. Another point is that comparison of individual scores for N_1 and P_2 in this study shows that for a given S, decrease in one component might be accompanied by increase in the other. This type of inconsistency was also reported by Gerken and Neff.

It seems unlikely that the neurophysiological responses of cats and monkeys are typically different from those of human subjects in a conditioning situation where behavioral responses are similar. However, the similarity of the responses used in the animal studies and the GSR used in the present study is certainly questionable. Conditioning of a

skeletal response may well involve different mechanisms than conditioning of an autonomic response such as the GSR.

The preceding discussion suggests that to speak of "the EP changes associated with conditioning" may be an over-simplification. It appears likely that the changes discovered so far are functions not simply of conditioning per se but also of specific variables such as the type of subject, the location of electrodes, and the particular behavioral response employed. Conditioning studies involving human subjects and skeletal responses would appear to be a profitable area for further investigation.

V Summary and Conclusions

The results of this study appear to indicate that human EP magnitude is affected by classical GSR conditioning. The extreme variability of the data recorded in this study necessitates only a tentative formulation of conclusions. It would appear that habituation as a variable affecting EP amplitude needs to be thoroughly investigated, since effects of habituation training may add algebraically with effects of conditioning if the two processes are allowed to operate concurrently.

The results of this study suggest that the EP correlates of classical conditioning, at least in human subjects, cannot be described as simply as has been done in the animal studies and in the study of W. Grey Walter. The possibility exists that no EP concomitants of conditioning per se exist. However the data of this study suggest that such concomitants may be of small enough magnitude that they are obscured by the high measurement variability. It is suggested that greater refinement in the techniques for measuring and comparing EPs, and the use of conditioned responses other than the GSR, may better illuminate the relationship between EPs and overt behavioral changes due to conditioning.

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VII Appendix A: Parameters of Electric Shock Stimulus

I: Magnitude of shock intensity increases

The following table lists initial shock intensities and subsequent intensities after each increase for each S.

Subject	Initial Shock Intensity	Intensity After 1st Increase	Intensity After 2nd Increase	Intensity After 3rd (last) Increase
E_1	1.65	1.65	1.65	1.65
E_2	1.15	1.30	1.50	1.65
E_3	1.65	1.65	1.65	1.65
E_4	1.35	1.55	1.65	1.65
E_5	1.33	1.50	1.65	1.65
E_6	1.45	1.65	1.65	1.65
E_7	1.10	1.27	1.55	1.65
E_8	0.25	0.32	0.40	0.85
E_9	0.44	0.85	1.07	1.20
E_{10}	1.00	1.20	1.35	1.60
B_1	1.35	1.65	1.65	1.65
B_2	1.40	1.65	1.65	1.65
B_3	1.15	1.30	1.53	1.65
B_4	1.27	1.45	1.65	1.65
B_5	1.45	1.65	1.65	1.65
B_6	1.20	1.35	1.50	1.65
B_7	0.85	1.10	1.30	1.40
B_8	1.05	1.20	1.35	1.60
B_9	1.23	1.35	1.60	1.65
B_{10}	0.44	0.80	0.90	1.07
R_1	0.45	0.90	1.15	1.25
R_2	1.15	1.30	1.50	1.65
R_3	1.65	1.65	1.65	1.65
R_4	1.07	1.23	1.40	1.65
R_5	1.40	1.65	1.65	1.65
R_6	0.50	1.07	1.20	1.35
R_7	0.40	0.85	1.15	1.35
R_8	1.35	1.65	1.65	1.65
R_9	0.93	1.15	1.30	1.50
R_{10}	1.65	1.65	1.65	1.65

Appendix A: Parameters of Electric Shock Stimulus (cont.)

II: Trials in which increases in shock intensity occurred

The total number of shock stimuli given was thirty. In general, shock increases were scheduled for (1) the 11th - 12th shock, (2) the 20th - 22nd shock, (3) the 25th - 26th shock. The criterion used throughout to determine the precise trial at which shock intensity was increased, was to maintain GSR to the shock stimulus. Where GSR to the shock US decreased or disappeared before the trial on which shock increase was scheduled, the shock intensity was increased earlier than the scheduled trial. In some cases departures from the schedule occurred because of error on the part of E or because of lack of equipment reliability.

The following tables list for the subjects in each group the particular shock stimulus, in the sequence of thirty shocks received by each S, at which shock increases occurred.

1st Shock Increase

E	B	R
12th shock - 9 Ss	11th shock - 8 Ss	11th shock - 9 Ss
9th shock - 1 S	12th shock - 1 S	8th shock - 1 S
	16th shock - 1 S	

2nd Shock Increase

E	B	R
20th shock - 5 Ss	20th shock - 8 Ss	22nd shock - 5 Ss
17th shock - 2 Ss	22nd shock - 2 Ss	20th shock - 4 Ss
18th shock - 1 S		18th shock - 1 S
22nd shock - 1 S		
23rd shock - 1 S		

3rd Shock Increase

E	B	R
26th shock - 5 Ss	25th shock - 7 Ss	26th shock - 10 Ss
25th shock - 3 Ss	26th shock - 2 Ss	
24th shock - 1 S	23rd shock - 1 S	
27th shock - 1 S		

Appendix A: Parameters of Electric Shock Stimlus (cont.)

III: Trials in which polarity of input to shock electrodes was reversed

For a number of Ss, input of one polarity was substantially more aversive than input of the opposite polarity, judging by the GSR to shock. When one input polarity appeared to constitute a relatively ineffective shock US, the reversal to the opposite polarity was made earlier than the scheduled time. Scheduled times for polarity reversals were the same for all groups. Actual reversals of polarity did not deviate from the schedule by more than 1 or 2 trials, except when error on the part of E or equipment malfunction occurred. The actual reversals of input polarity for all Ss are given in the table below.

Shock for which input polarity was reversed (all groups)

Reversal	Number of shock at which reversal occurred
1	5th shock
2	8th - 10th shock
3	14th - 16th shock
4	20th - 21st shock
5	22nd - 25th shock
6	26th - 28th shock

IV: Shock output from constant current voltage source

In the graph below, current output is plotted against scale reading of the shock source. The output rises in an approximately exponential fashion until the maximum of 1.65 mA is reached.

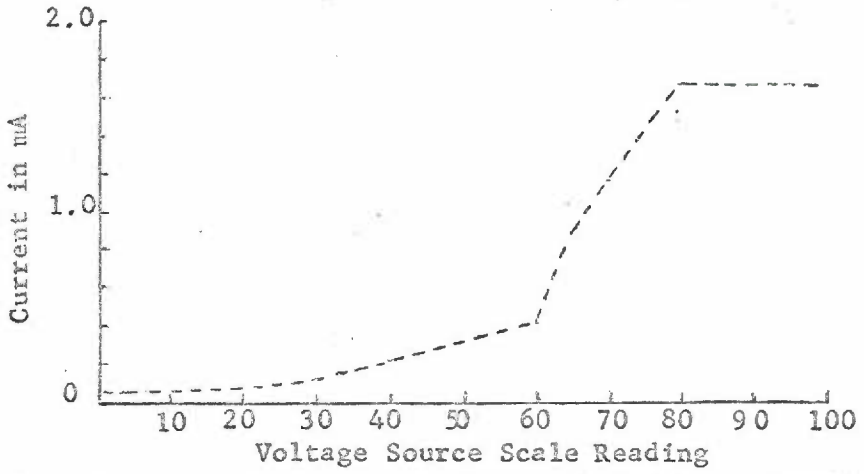

Figure 5. Output of Constant Current Voltage Source

VIII Appendix B: GSR Magnitudes for All Ss

In the table below, GSR magnitudes given for Phase I are the means for the last six trials in Phase I (CS-alone). GSR magnitudes for Phases II, III, and IV are raw scores obtained from CS-alone test trials. GSR magnitudes for Phase V are means for 3-trial blocks during the 15 CS-alone extinction trials. All magnitudes are given in millivolts.

Ss	E_1	E_2	E_3	E_4	E_5	E_6	E_7	E_8	E_9	E_{10}
Phase I	.55	1.75	1.25	.18	1.05	1.63	0	2.45	.50	1.20
	.43	3.40	1.72	.17	.75	.83	.50	2.63	.47	.78
II	1.70	1.35	3.45	1.20	2.05	2.85	1.70	2.05	1.85	1.40
	.75	1.80	3.50	.75	2.25	1.70	3.40	.40	8.90	2.20
	.90	1.55	3.90	2.60	3.05	3.30	0	3.00	9.90	1.70
	.50	1.60	2.45	0	1.60	1.80	.20	0	7.80	1.90
	1.05	1.25	2.25	0	.55	1.00	0	.10	7.70	.75
III	.55	1.50	1.75	.05	1.35	.15	.75	0	5.85	.85
	1.75	1.60	.30	0	.30	.10	.35	0	5.95	1.20
	.45	1.90	2.05	.40	.35	.05	.95	.05	7.30	1.00
	.55	1.20	.90	0	2.70	0	.50	.80	6.60	1.10
	.45	1.80	.65	0	.50	.15	0	2.40	6.25	1.80
IV	2.00	1.80	2.55	0	.35	.10	.80	1.70	4.05	.50
	3.10	.95	1.90	0	0	0	0	1.00	4.05	8.50
	1.20	1.50	3.20	0	.55	0	0	1.50	5.10	.20
	.25	1.10	.10	.25	.25	0	0	.20	3.05	.80
	0	1.35	.30	.25	.25	0	.90	.20	3.65	1.05
V	.52	1.88	.45	1.05	1.87	0	.82	.17	3.18	1.07
	.88	1.08	1.18	0	.20	.31	.93	.50	2.50	.50
	.77	.97	1.40	1.90	.55	0	.83	.15	2.33	.30
	1.28	1.10	2.02	0	.35	0	.88	.25	1.11	.31
	.98	1.22	1.22	.10	1.87	.05	0	.43	1.11	.05

Appendix B: GSR Magnitudes for All Ss (cont.)

Ss	B_1	B_2	B_3	B_4	B_5	B_6	B_7	B_8	B_9	B_{10}
Phase I	.11	.25	5.81	.77	1.01	1.80	0	1.97	.07	.88
	.97	.05	5.60	.43	.07	1.61	.11	.81	.10	.73
II	.10	.45	1.70	1.85	.70	2.50	1.60	1.15	9.70	2.55
	.45	.50	3.10	2.70	1.10	2.20	2.60	.25	0	0
	.35	0	1.75	1.40	3.50	1.30	.15	0	1.15	1.00
	0	0	3.15	1.90	1.40	1.15	.65	.15	.10	.25
	0	0	2.30	0	.90	2.55	0	.95	0	0
III	.40	0	3.60	.35	.15	2.30	.15	.95	.10	.10
	0	0	0	.90	0	.10	0	.10	0	.10
	0	0	3.15	.25	.05	2.40	.15	4.20	0	.10
	0	0	2.40	.20	.20	2.45	.30	4.05	0	0
	0	0	.10	0	0	.35	.10	0	0	0
IV	1.75	0	5.95	1.00	.20	.15	0	.35	.30	.15
	.15	0	6.10	.10	.20	.10	0	.35	0	0
	.10	0	.40	0	0	.25	0	.20	0	0
	3.20	0	0	.35	0	0	.40	2.90	0	0
	.70	0	3.60	0	.20	1.40	.10	0	0	0
V	1.00	.91	1.60	.27	.25	0	.01	.37	0	.17
	.43	0	2.91	.05	0	.03	.20	.58	.05	.28
	1.43	.70	2.38	.90	.08	.10	.07	.23	0	.27
	.23	.71	2.23	0	.33	0	.05	.07	1.10	.05
	.98	.88	1.35	.08	.31	.03	.03	.35	0	0

Appendix B: GSR Magnitudes for All Ss (cont.)

Ss	R_1	R_2	R_3	R_4	R_5	R_6	R_7	R_8	R_9	R_{10}
Phase										
I	.07	0	.67	3.20	.85	1.65	3.37	9.87	1.70	.08
	.25	.45	.45	1.71	.40	1.18	3.21	10.21	1.03	0
II	.50	0	1.35	3.25	2.00	1.00	1.95	6.95	2.75	0
	.60	0	.20	1.65	.65	.90	1.25	7.10	.95	0
	.30	0	0	1.35	1.10	.55	.95	7.50	2.15	0
	.20	0	0	2.20	0	0	.95	5.35	1.35	0
	0	0	0	0	0	1.05	1.15	1.15	2.10	.10
III	.65	0	0	0	.60	.75	2.00	.55	2.40	0
	0	0	0	3.80	.25	.50	1.95	1.90	.40	0
	.20	.20	0	2.80	0	.30	1.55	1.10	1.50	0
	.50	0	0	.45	.40	.60	2.05	5.70	.90	0
	0	0	.80	0	0	.80	.60	.90	.55	0
IV	.10	0	0	0	1.80	0	0	5.30	.40	0
	0	0	0	1.30	.60	.75	0	1.05	1.15	2.10
	.25	0	0	1.30	2.90	0	0	.70	1.05	0
	.25	0	0	.80	0	0	0	.10	.20	0
	.80	0	0	.95	.65	0	1.20	5.00	1.05	0
V	1.25	.20	.23	.20	.75	.07	.41	1.03	.90	0
	.18	0	0	.17	.90	1.30	.85	1.85	.61	.27
	.63	0	0	.20	2.48	0	.18	1.63	.95	.31
	.05	.10	0	.07	.63	0	0	1.43	.35	0
	1.30	0	.80	.40	1.50	.07	.25	3.95	.98	0

IX Appendix C: Amplitudes and Latencies of N_1 and P_2 for All Ss

I: Amplitude* of N_1

Ss	E_1	E_2	E_3	E_4	E_5	E_6	E_7	E_8	E_9	E_{10}
Phase										
I	24.7	17.0	21.0	22.5	15.3	22.0	32.5	32.0	22.0	34.0
II	19.6	17.9	21.5	20.0	14.5	29.5	27.5	30.5	22.5	32.0
III	11.5	19.1	16.0	14.5	10.6	20.0	21.5	26.5	22.0	34.0
IV	14.0	14.5	23.5	14.0	12.8	16.0	25.5	35.5	29.5	44.0
V	16.6	16.2	21.0	13.0	8.5	15.0	17.0	28.0	28.0	37.0
Ss	B_1	B_2	B_3	B_4	B_5	B_6	B_7	B_8	B_9	B_{10}
Phase										
I	27.0	14.5	16.5	24.0	22.0	15.0	24.8	16.0	15.0	38.5
II	10.0	13.5	22.0	20.0	17.0	13.0	20.8	16.5	2.0	36.5
III	15.0	13.0	15.5	25.0	21.0	22.0	20.8	12.5	6.0	24.5
IV	8.0	9.5	13.0	16.5	12.5	14.0	19.2	8.5	9.5	28.0
V	10.0	7.0	16.5	20.0	11.0	25.0	21.6	16.0	18.0	22.5
Ss	R_1	R_2	R_3	R_4	R_5	R_6	R_7	R_8	R_9	R_{10}
Phase										
I	19.0	16.5	25.9	27.0	5.1	12.0	8.0	38.5	17.0	13.6
II	19.0	18.5	18.3	31.0	2.6	16.0	13.5	18.5	35.0	14.5
III	19.0	12.0	17.4	38.0	6.8	9.5	14.0	12.0	34.0	7.7
IV	19.5	7.5	13.6	26.0	9.4	8.0	11.0	12.0	47.5	12.8
V	15.5	13.0	12.3	35.0	3.4	19.0	16.0	14.0	22.0	8.5

* Amplitudes are given in arbitrary units. To obtain amplitude in microvolts multiply each score by 0.384.

Appendix C: Amplitudes and Latencies of N_1 and P_2 for All Ss

II: Amplitude* of P_2

Ss	E_1	E_2	E_3	E_4	E_5	E_6	E_7	E_8	E_9	E_{10}
Phase										
I	27.2	27.6	36.0	31.0	33.2	37.0	29.5	49.0	52.0	53.0
II	23.8	25.1	29.0	22.0	28.1	49.5	32.5	40.5	45.0	54.0
III	17.9	25.1	23.5	21.5	23.4	38.0	24.5	28.5	40.0	47.0
IV	23.0	20.4	22.0	17.0	18.7	31.5	23.0	53.0	51.0	60.0
V	16.6	20.4	26.0	16.5	16.6	31.5	23.0	32.0	41.0	51.5
Ss	B_1	B_2	B_3	B_4	B_5	B_6	B_7	B_8	B_9	B_{10}
Phase										
I	39.5	18.5	24.0	28.5	37.0	24.0	34.4	24.0	21.0	59.5
II	31.0	19.5	33.0	30.0	24.0	26.0	34.4	27.5	16.5	54.5
III	23.5	15.0	23.5	32.0	27.0	28.5	40.0	21.0	16.0	46.5
IV	16.5	16.5	24.0	23.0	19.5	24.0	25.6	21.5	30.0	44.0
V	25.5	13.0	22.0	32.0	22.0	40.0	28.0	20.5	27.0	22.5
Ss	R_1	R_2	R_3	R_4	R_5	R_6	R_7	R_8	R_9	R_{10}
Phase										
I	29.0	26.5	23.0	56.0	15.3	9.0	26.5	31.5	29.0	13.6
II	34.5	24.5	18.3	60.0	5.5	18.0	31.5	23.0	46.0	21.3
III	29.0	15.0	8.9	58.5	8.5	9.5	27.0	17.0	37.5	6.0
IV	30.0	15.0	9.8	42.0	13.2	10.0	26.0	18.0	47.0	15.3
V	27.5	20.0	12.8	55.0	6.8	22.5	28.0	18.5	37.0	11.0

* Amplitudes are given in arbitrary units. To obtain amplitude in microvolts multiply each score by 0.384.

Appendix C: Amplitudes and Latencies of N_1 and P_2 for All Ss

III: Latency of N_1 in msec

Ss	E_1	E_2	E_3	E_4	E_5	E_6	E_7	E_8	E_9	E_{10}
Phase										
I	130	90	90	80	115	100	85	105	115	100
II	135	90	80	70	110	95	70	90	100	110
III	125	90	85	80	120	90	70	95	85	105
IV	120	90	80	80	105	95	65	90	115	100
V	125	90	90	85	105	95	90	95	105	100
Ss	B_1	B_2	B_3	B_4	B_5	B_6	B_7	B_8	B_9	B_{10}
Phase										
I	95	40	80	90	90	75	95	75	70	95
II	95	85	85	100	90	70	95	100	80	95
III	95	75	80	100	90	75	105	105	40	100
IV	100	90	85	75	95	70	100	105	70	100
V	75	45	85	85	80	75	100	105	40	90
Ss	R_1	R_2	R_3	R_4	R_5	R_6	R_7	R_8	R_9	R_{10}
Phase										
I	90	90	80	70	55	110	65	110	95	95
II	100	100	60	65	75	110	65	100	80	110
III	105	95	80	70	60	65	105	105	110	110
IV	80	115	90	70	70	100	75	110	105	110
V	65	100	65	70	70	85	75	110	90	105

Appendix C: Amplitudes and Latencies of N_1 and P_2 for All Ss

IV: Latency of P_2 in msec

Ss	E_1	E_2	E_3	E_4	E_5	E_6	E_7	E_8	E_9	E_{10}
Phase										
I	200	160	135	120	165	155	160	170	165	165
II	210	150	145	125	165	140	155	175	170	170
III	225	165	170	160	170	140	165	230	155	175
IV	190	160	140	175	170	145	115	175	160	175
V	210	160	145	150	165	150	145	235	165	170

Ss	B_1	B_2	B_3	B_4	B_5	B_6	B_7	B_8	B_9	B_{10}
Phase										
I	135	115	125	140	135	130	165	165	145	165
II	135	115	135	165	145	130	170	145	130	170
III	130	115	150	140	145	135	160	125	120	155
IV	130	135	130	135	140	155	175	140	130	155
V	135	120	145	135	135	140	180	165	125	165

Ss	R_1	R_2	R_3	R_4	R_5	R_6	R_7	R_8	R_9	R_{10}
Phase										
I	135	125	115	175	170	145	160	145	145	145
II	135	150	120	160	170	150	155	160	140	135
III	135	135	135	165	160	145	160	185	145	130
IV	140	165	125	160	190	130	140	165	155	145
V	145	150	110	160	190	150	150	170	140	150