DISHABITUATION OF THE HEAD - SHAKE RESPONSE IN THE RAT

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This is to certify that the

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ABSTRACT

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RESPONSE IN THE RAT

By

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Habituation (response decrement accompanying repeated stimulation) has recently attracted considerable attention among psychologists and physiologists as a basic type of behavior modification. One of the phenomenon's fundamental features is dishabituation, that is, the reinstatement of responding when a novel stimulus follows habituation training. Dishabituation has been studied widely and interpreted in various ways, but there have been few systematic investigations. The present study was designed to investigate the characteristics of dishabituation of the head-shake response (a rapid rotation of the head about the front-to-rear axis, elicited by a stream of pressurized air directed in the ear) in the laboratory rat. A secondary purpose was to evaluate the relevance of the two major theoretical interpretations of dishabituation (removal of habituation versus superimposed sensitization).

In Experiment 1 (between-trial dishabituation), 30 male Holtzman rats, 80 days old, received three dishabituation sessions 24 hr. apart. The dishabituatory stimulus, consisting of a vibratory-auditory pattern provided by a buzzer mounted on the test stand, appeared once during each 30-sec. inter-trial interval separating 10 30-sec. habituation trials with the air stimulus. Dishabituatory stimuli preceded the following trial by 1, 15, or 29 sec., and each of these positions was repeated three times in each session. The set of dishabituation sessions was preceded and followed at 24-hr. intervals by no-buzzer control sessions.

In Experiment 2 (within-trial dishabituation), 30 additional rats received essentially the same treatment as in Experiment 1, except that the dishabituatory stimulus was presented during habituation trials (5, 15, or 25 sec. following the trial onset), and air stimulation continued for 30 sec. after the dishabituatory stimulus.

The combined results of both experiments revealed a biphasic pattern of dishabituation. The buzzer produced a brief period (2-3 sec.) of enhanced responding, followed by a 10-15 sec. period of response suppression. This biphasic pattern was superimposed upon a background of more general response suppression, which demonstrated the strong inhibitory influence of the buzzer. The finding of dual effects suggests that dishabituation is more

complex than is generally acknowledged; neither major theory explains the results of these experiments.

Dishabituation in the present instance must be characterized as a superimposed process of sensitization, very brief in duration. Although the buzzer appeared to retard across-trial habituation, long-term dishabituatory effects were largely obscured by the inhibitory effects of the buzzer. There was no evidence for habituation of dishabituation. That is, the excitatory effect increased across trials and across sessions, although the post-excitatory suppression effect habituated across sessions.

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INTRODUCTION

When a response is repeatedly elicited by the presentation of the same stimulus, the response exhibits decrement along one or a number of dimensions (e.g., frequency, magnitude, latency). This waning of responsiveness has been labeled habituation and has been the subject of considerable discussion and experimentation. General discussions and reviews of the research in habituation can be found in Humphrey (1933), Harris (1943), Thorpe (1963), Thompson and Spencer (1966), Hinde (1966), and Ratner and Denny (1970).

In recent years the phenomenon of habituation has attracted attention among psychologists and physiologists as a major type of behavior modification (i.e., a type of learning). The reasons for its rise to eminence are several. In the first place, it is alleged to be the simplest type of learning (Thorpe, 1963; Ratner and Denny, 1970). While this conception is somewhat misleading (many conceptualize it as the simple dropping out or weakening of a response, which is often an oversimplification), it is essentially non-associative, and only one exteroceptive sensory field is necessarily involved. In the second place, the phenomenon occurs across the entire phylogenetic range, from ameba to

humans (Harris, 1943). This aspect renders the process particularly valuable to the comparative study of behavior. Finally, habituation is considered to have profound ecological importance for the individual animal. Considerable economy of activity is achieved by ceasing to respond to stimuli which have no significant consequences (Thorpe, 1963).

One of the most fundamental conceptual features of habituation is the notion that it should be clearly distinguished from response decrement due to peripheral effects. Virtually every investigator in the area (e.g., Harris, 1943; Thompson and Spencer, 1966) asserts that decrement due solely to either receptor adaptation or effector fatigue cannot be considered habituatory. This is in line with the idea that habituation is a form of learning, and, therefore, must involve changes in the central nervous system and/or changes which are somewhat lasting. Despite the difficulties in effectively separating these various processes in the intact organism, certain procedural tests do allow inferences to be made about the physiological processes underlying an observed instance of response decrement. One of the more common among these tests is the dishabituation procedure. This involves the presentation of an extraneous stimulus during the course of habituation to a constant stimulus. If the novel stimulus produces reinstatement

of or an increase in responsiveness to the standard stimulus, the response decrement cannot be accounted for by either effector fatigue or sensory adaptation alone. The logic behind this reasoning is simply that peripheral fatigue or adaptation would preclude immediate reversibility of the response decrement.

The general importance of dishabituation can be traced to several factors. Its utility in shedding light on the internal processes underlying a given instance of response decrement, especially in vertebrates and higher invertebrates, has already been suggested. Indeed, Thompson and Spencer (1966) point out that dishabituation is commonly used to demonstrate that habituation has occurred. The importance of dishabituation is also underscored by Ratner and Denny (1970), who suggest that one of the general characteristics of habituation is the fact that concurrent stimulation (e.g., dishabituatory stimuli) affects the course of habituation. Likewise, Thompson and Spencer (1966) include dishabituation as one of nine "parametric characteristics" of habituation. Finally, the possible ecological utility of the phenomenon has been discussed by Evans (1965), who suggests that habituation "may be of biological importance because it enables habituated (organisms) to react to a previously innocuous stimulus if there is a sudden change in the background stimulation."

Because of the general importance attached to dishabituation, the phenomenon has been given considerable attention by investigators working with habituation. Thompson and Spencer (1966) have concluded that "This phenomenon appears to be as ubiquitous as habituation itself" (p. 19). While this assertion may contain some validity as a general rule, review of the literature reveals that Thompson and Spencer's statement of the case is somewhat over-enthusiastic.

Much of the data on dishabituation comes from studies of the orienting response (OR). Bartoshuk (1962) found that after habituation of neonatal heart rate acceleration to tones, an increase in tone intensity produced partial recovery of the response. Grim and White (1965) presented a novel light after 16 habituation trials with a red light and observed partial dishabituation of the human GSR which lasted only one trial. When habituation of the vasoconstrictive OR was produced by presenting numbers <u>seriatim</u>, Unger (1964) reported that a number out of sequence resulted in dishabituation. Incomplete recovery of the vasoconstrictive OR has also been noted by Zimny and Miller (1966) when a police whistle followed habituation to a hot or cold stimulus. Likewise, Zimny and Schwabe (1965) found that when habituation trials to a 500 cps tone were followed by a 4000 cps tone, partial recovery of the GSR in humans

resulted. However, dishabituation effects were apparent for only one trial, and a 1000 cps tone failed to produce any dishabituation. A later study by Zimny and Kienstra (1967) provided only weak evidence for dishabituation when a tone interpolated every 6-10 trials in a series of weak habituating shocks produced partial recovery of the GSR in humans only on the first presentation. In the same study, the tone interpolated in a series of shock-plus-tone stimuli failed to produce any dishabituation. Evidence for dishabituation of the OR in monkeys has been reported by Bagshaw, Kimble, and Pribram (1965) and Kimble, Bagshaw, and Pribram (1965), who observed that the previously habituated GSR to a tone was fully reinstated after presentation of two novel tones. However, no dishabituation of the GSR in humans was found by Fried, Korn and Welch (1966) when a novel light was presented once half-way through 80 serial presentations of four colored lights. Likewise, McDonald, Johnson, and Hord (1966) found no dishabituation of the OR in humans during sleep when novel tones were interpolated among presentations of a standard tone. Finally, Wickens, Nield, and Wickens (1966) obtained no dishabituation of the GSR in cats when weak shock was presented on trials 1 and 5 of the 11th and 12th habituation sessions, with tone plus light as the habituating stimulus. The results of studies of the OR are thus divided:

dishabituation does not always occur. When dishabituation does occur, it is often weak, and its effects are usually short-lived.

Various other types of orienting behaviors have provided evidence relating to dishabituation. Sharpless and Jasper (1956), studying the arousal response in cats, produced dishabituation by presenting novel tones. Hayes, Hertzler, and Hogberg (1968) observed that reversing the direction of rotation of a striped drum produced partial recovery of the habituated opto-kinetic response in turtles. However, simply stopping the drum for an equivalent period produced as much recovery. Dishabituation of orienting behavior in the waterbug Notonecta glauca has been reported by Wolda (1961). In this study, a series of right stimuli presented during the end of a 5-7 min. recovery interval augmented recovery of the orienting response to left stimuli. No augmentation of recovery occurred when the dishabituating stimuli were presented at the beginning of the recovery interval.

A number of studies dealing with startle or withdrawal responses have dealt with dishabituation. Berg and Beebe-Center (1941) reported that a novel stimulus produced full recovery of the human cardiac startle to pistol shots. Similar dishabituation of the rat's respiratory startle to a buzzer has been produced by pinching the leg (Lehner, 1941). Prosser and Hunter

(1936) also dishabituated the rat's startle response to a click by presenting visual and auditory stimuli. The earthworm's withdrawal response to a buzzer, after being habituated to zero-level criterion, is fully reinstated by a single tactile stimulus (Gardner, 1969). And polychaetes' withdrawal to a change in illumination has been dishabituated by both tactile and electrical stimuli (Evans, 1965, 1966). However, investigations of polychaetes' withdrawal response to moving shadows by Clark (1960a, b), with mechanical shock as the interpolated stimulus, failed to provide substantial evidence for the occurrence of dishabituation.

Among a large number of studies investigating habituation of nystagmus responses in various organisms, only a few have looked at dishabituation. Crampton and Schwam (1961) have demonstrated dishabituation of postrotatory nystagmus in the cat by auditory or cutaneous stimuli. However, three varieties of visual experience during acceleration failed to modify the course of habituation in cats (Crampton, 1962), providing evidence against dishabituation. Similarly, Brown (1966) reported that interacting angular accelerations interpolated between standard single accelerations failed to retard habituation. And Collins (1967) found that alternating the direction of acceleration and interrupting the nystagmus response in one of these directions failed to retard habituation.

It would thus appear that dishabituation of nystagmus responses is anything but a universal phenomenon.

Various cutaneous and flexion reflexes have been examined in the study of dishabituation. The human abdominal reflex can be dishabituated by a pat on the abdomen (Lehner, 1941). This finding has been verified and extended by Hagbarth and Kugelberg (1958). Hollis (1963) reported that the abdominal reflex to electric shock in pupae of Tenebrio molitor was universally reinstated by a tactile stimulus. Lehner (1941) produced dishabituation of the tail flexion reflex in rats by presenting a tactile stimulus. That various spinal reflexes (e.g., leg and tail flexion, crossed leg reflex) in the rat can be habituated and subsequently dishabituated by strong general excitation has been demonstrated by Prosser and Hunter (1936). And Thompson and Spencer (1966), after habituating the spinal flexion reflex in the cat to electrical stimulation of the afferent nerve, produced dishabituation by a strong pinch of the digits, by increasing the frequency of shock pulses, and by stimulating with stronger shock elsewhere on the limb.

A handful of studies utilizing electrophysiological recording techniques have successfully demonstrated dishabituation. In a study of evoked potentials in the afferent auditory pathways of the cat, Al'tman (1960) found that skin shock produced full recovery of the

amplitude of evoked potentials after habituation to 5 hr. of click stimuli. Buno, Velluti, Handler, and Garcia-Austt (1966), investigating auditory evoked potentials in the guinea pig, found that "interference" stimuli (e.g., light flash, electrical shock to the pinna, air puff) produced a decrement in the amplitude of evoked potentials before habituation but an increase (dishabituation) after habituation to acoustic stimuli. Hernandez-Peon (1960) summarized results of several studies which demonstrated dishabituation of evoked potentials in the auditory, visual, olfactory, and tactile afferent pathways, as well as in non-specific projection systems, in the cat. Dishabituation was typically produced by introducing an extraneous stimulus, or by briefly changing the rhythm or intensity of the habituating stimulus. Finally, in a study of post-synaptic potentials of motoneurons in the flexor twitch pathway of the cat, Spencer, Thompson, and Neilson (1966) used tetanic electrical stimulation to produce dishabituation following habituation to electrical stimulation of the afferent nerve.

It should be noted that in all of the studies mentioned above the dishabituating stimulus was followed by continued presentations of the habituating stimulus. The dishabituation measure thus reflected disruption of the response decrement to the habituating stimulus, rather than responsiveness to the dishabituating stimulus itself.

The latter measure has frequently been confused with dishabituation, although it is more accurately a measure of generalization of habituation. Studies which have employed a generalization procedure, rather than a true dishabituation procedure, will not be dealt with here.

It has occasionally been observed that, when a dishabituatory stimulus is presented repeatedly, its disruptive influence wanes. This may be considered as a special case of habituation, and it has been included in Thompson and Spencer's (1966) set of "parametric characteristics" of habituation. Habituation of dishabituation has been reported by Bagshaw, Kimble, and Pribram (1965), Evans (1966), Lehner (1941), Hagbarth and Kugelberg (1958), Hagbarth and Finer (1963), Crampton and Schwam (1961), and Thompson and Spencer (1966). While Zimny and Miller (1966) and Zimny and Schwabe (1965) reported no habituation of dishabituation, only three or four presentations of the novel stimulus were given in both studies. However, no waning of dishabituation was noted by Wolda (1961) when a series of interrupting stimuli was repeated. Although it may well be that sufficient repetitions of a novel stimulus would eventually lead to a loss of dishabituation effects, the universal occurrence of habituation of dishabituation has yet to be established.

From a conceptual point of view, the phenomenon of dishabituation has been interpreted in two major ways.

The classic approach has been to view dishabituation as the neutralization, or induced recovery, of the process of habituation (Humphrey, 1933). A variation of this theme is that of Sokolov (1960), who has proposed that the dishabituating stimulus disrupts the cortical "model" which is built up during training with the habituating stimulus. A second approach is to view dishabituation as a more or less independent and temporary process of sensitization (Thompson and Spencer, 1966; Evans, 1965; Spencer et al., 1966), excitation (Al'tman, 1960), or facilitation (Wolda, 1961). Similar interpretations have been made by Bartoshuk (1962) and Sharpless and Jasper (1956), who regard dishabituation as an increase in arousal level. These interpretations have been prompted by such observations as the failure to find dishabituation if the arousing effects of the novel stimulus are allowed to wear off before the familiar stimulus is repeated (Sharpless and Jasper, 1956; Wolda, 1961). These findings would not be expected if one regards dishabituation as the neutralization of habituation. There are, therefore, rather simple procedural means by which to test the validity of these two major theoretical positions.

The Present Study

A relatively new experimental situation which has recently been employed in the study of habituation is the

head-shake response in the laboratory rat (Askew, Leibrecht, and Ratner, 1969). The head-shake response consists essentially of a rapid rotation, or twisting, of the head about the front-to-rear axis. A stream of pressurized air directed at the ear serves as the eliciting stimulus. Several desirable characteristics recommend the head-shake response as a particularly well-suited experimental situation for the study of habituation. The response can be easily elicited and identified, has a low base rate, and can be adequately characterized by frequency of occurrence alone. It is quite stereotyped within and between animals (Leibrecht and Askew, in press), and is highly reliable.

Previous work with the head-shake response (Askew et al., 1969; Leibrecht and Askew, in press) has shown that repeated presentations of the air stimulus lead to two decremental phenomena. The first of these is a rapidly occurring, short-term frequency decrement which occurs within 30-sec. stimulus presentations. The second is a slower, relatively long-term frequency decrement which occurs across trials and takes more than 6 hrs. to recover fully.

Pilot work aimed at demonstrating dishabituation of the head-shake response has produced mixed findings. When a variety of stimuli (e.g., mild shock to the tail, tapping the stand, stroking the fur) were presented

during the inter-trial interval after a considerable number of habituating trials, no significant dishabituation resulted. At the same time, tapping the stand during presentations of the air stimulus produced nearly universal recovery of the head-shake response. In view of these somewhat perplexing findings from pilot research and from published studies reviewed earlier, and considering the importance generally attached to dishabituation, a major investigation of dishabituation of the head-shake response would appear to be worthwhile.

The main purpose of the present experiment was to investigate the characteristics of dishabituation in a well-established experimental situation, and secondarily to evaluate the relevance of the two major theoretical interpretations of dishabituation. Accordingly, the study has examined the effects of an extraneous stimulus on the course of habituation of the head-shake responses. In two separate experiments the point in time at which the dishabituatory stimulus is presented has been manipulated both between trials (Experiment 1) and within trials (Experiment 2). In addition, possible habituation of dishabituation, both across trials and across repeated sessions, has been investigated.

EXPERIMENT 1: BETWEEN-TRIAL DISHABITUATION

The first experiment involved the presentation of an extraneous stimulus during inter-trial intervals. As a test of the nature of the dishabituation process (sensitization vs. neutralization of habituation), the proximity of the extra stimulus to the following habituation trial was varied. Three positions within the inter-trial interval, ranging from immediately preceding to relatively remote from the following trial, were employed, with the expectation that position should have no effect if dishabituation simply reverses the accumulated habituation. On the other hand, if dishabituation actually represents a temporary process of sensitization, or facilitation, then the extra stimulus should have greater disruptive effects when it is closer to the following trial. An individual dishabituation session contained presentations of the extraneous stimulus in all three positions, and each position was repeated three times to test for habituation of dishabituation. In addition, possible changes in the dishabituation process were charted across repeated sessions.

Method

Subjects. Thirty male Holtzman albino rats, 80 days of age, served as subjects (Ss). All were experimentally naive, and testing did not begin until 1-2 wk. following arrival in the laboratory. Animals were housed in groups of 4-5 per cage under conditions of constant light and temperature, with water and food available ad lib.

Apparatus. Movement during test sessions was restricted by placing the animals upon an elevated 2 x 6½ x ¼ in. platform (of ¼-in. hardware cloth), open at the top so that short sides extended upward on all edges. The platform rested atop a supporting column 29 in. above the floor. Hanging downward and sloping outward at an angle of 10° from the edges of the platform were sections of galvanized sheet metal, forming a "collar" 10 in. long to discourage escape attempts. The base of the wooden column which supported the platform was mounted on a 4-in. lazy susan ball bearing so that the entire test stand could be rotated in either direction.

The test stimulus consisted of a stream of pressurized air from a Silent Giant aquarium pump, Model 120. The air was delivered through a hand-held rubber tube with an inside diameter of 1 mm. at the tip. With the aid of two valves in the system, the pressure at the tip of the tube was adjusted daily to a constant value.

Adjustment was accomplished by holding the tip of the tube 1 mm. from the end of a 1.5-mm. open manometer and setting the valves so that a 12 cm. column of alcohol was displaced 2.5 cm. It should be noted that a low-frequency low-intensity "humming" sound was produced by the pump and carried through the tube, providing a complex air-tone stimulus.

The dishabituatory stimulus was provided by a 6-volt buzzer (Edwards "Dixie" buzzer, Cat. No. 725) attached with screws to the base of the test stand, with a double layer of cardboard (1.5 mm. total thickness) between the buzzer and the wooden base. This particular arrangement was selected on the basis of preliminary work with five pilot animals. Power for the buzzer was supplied by a 6-volt AC transformer. When activated, the buzzer set up a stimulus pattern with both auditory and vibratory components. The auditory intensity where S rested (27 in. from the buzzer) was 85 db. The vibratory pattern in the center of the platform on which S rested was characterized by an acceleration of 35.4 ft./sec.^{2} (1.10 g), a velocity of .106 in./sec., and a displacement of .0004 in. The duration of the buzzer presentation, controlled by a Hunter Interval Timer (Model 111-C) was set at .10 sec.

Timing of intervals and programming of events were accomplished by means of electromechanical equipment,

which was housed inside a sound-insulated box. A light, not visible to S, signalled periods of stimulation to the experimenter, and a counter displayed the trial number. Responses were recorded with the aid of a Gerbrands event recorder, operated by a micro-switch held in the experimenter's hand. In order to mask incidental noises a white noise level of 75 db was maintained.

Procedure. The head-shake response was elicited by moving the stimulus back and forth across the center of the ear at an approximate rate of three cycles per sec. The distance of the tube from the ear was 3/8 to 1/2 in., and the locus of stimulation was approximately 3/8 in. wide. Whenever S moved its head an appreciable distance during stimulation, the experimenter moved his hand accordingly to keep the stimulus directly in S's ear. If S turned around on the stand, the experimenter rotated the stand until S was again in the proper orientation, at which time the normal stimulus pattern was resumed.

The basic habituation session consisted of an initial 5-min. unstimulated adaptation period on the test stand, followed by 11 30-sec. stimulus presentations in the left ear separated by a fixed 30-sec. inter-trial interval. All Ss received a total of five such sessions separated by intervals of 24 hr. The first and fifth sessions served as control sessions

(no dishabituatory stimulus) and were modified only to include a 12th trial during which the air stimulus was directed away from the ear (test for classical and/or temporal conditioning).

During each of sessions 2, 3, and 4 (dishabituation sessions) the dishabituatory stimulus (.1-sec. buzzer) was presented three times in each of three positions (1, 15 or 29 sec. preceding the next trial), with buzzer presentations counterbalanced in blocks of three trials according to a within-subjects Latin square procedure. A total of 12 such counterbalanced schedules were possible, and these were randomly assigned to each session with the restriction that no S receive the same schedule twice. Trials 2-10 constituted the nine dishabituation trials; trials 1 and 11 were never preceded by the buzzer.

A single experimenter delivered the air stimulus and operated a micro-switch with his other hand whenever a head-shake response occurred.

The basic datum, number of head-shake responses, was obtained by placing a transparent plastic template over individual trials on the paper recording tapes. Using as guides the marks denoting the onset and offset of the trial made by one pen of the event recorded, the template divided each 30-sec. trial into 12 periods of 2.5-sec. each. The number of responses in each 2.5 sec. time period was then recorded on data sheets. In

addition to response frequency, the latency of the first response in each trial was measured with a transparent plastic scale ruled in millimeters, 1 mm. being equal to 1 sec. on the recording tape.

Four major dependent variables were used in analyzing the results of the first experiment. These were (a) the number of responses in the first 2.5-sec. time period of each dishabituation trial, (b) the total number of responses per trial, (c) the latency of the first response in each trial, and (d) the rate of response decrement within each trial. The last measure was obtained by dividing each trial into six periods of 5 sec. each and noting the first period in which the number of HSRs (head-shake responses) was less than or equal to the number in the last period.

Statistical analyses were carried out by means of analysis of variance (AOV) procedures (Winer, 1962). Subjects (a random factor), and Sessions, Trials and Buzzer Positions (fixed factors) were the usual category variables employed in a single-entry-per-cell design (Winer, 1962, p. 290). Under this procedure, main fixed effects were tested by using the double interaction with Subjects as the denominator in the F-ratio. Similarly, double interactions involving fixed factors were tested against the corresponding triple interactions with Subjects, and the fixed-factors

triple interaction was tested against the four-way interaction. Since a single observation per cell did not permit a within-cell variance estimate, the Subjects effect and interactions involving Subjects could not be tested. Duncan's (1955) multiple range test was used in evaluating individual comparisons.

The parametric AOV procedure was chosen as the chief statistical tool because the assumptions prerequisite to its use appeared to be adequately met. While most distributions showed some skew, especially latency, the departure from normality was not large and should not have appreciably affected the resulting probability levels since the number of Ss (30) was relatively large (Hays, 1963; Boneau, 1960). Heterogeneity of variance, appreciable only for latency and within-trial rate of habituation, should have had little effect because of the equal number of observations in each cell (Boneau, 1960; Hays, 1963). Preliminary inspection of intercorrelations between cells for fixed factors indicated that the assumption of homogeneity of covariance was adequately met. However, since distributions frequently tended to be J-shaped and homogeneity of variance in some cases appeared questionable, no F-values with a probability level higher than .01 were accepted as significant. It should be pointed out that, because multiple AOVs were employed, the overall probability level was somewhat greater than .01.

The general format for presentation of the results, following a brief description of the overt response to the buzzer, has been arranged according to independent variables. Changes across Sessions and Trials are considered first, in an attempt to determine if dishabituation (as might be reflected in a general elevation in responding or a retardation in across-trial habituation) actually occurred. The more specific effects of Buzzer Positions are then taken up, with the expectation that the more remote positions should produce less dishabituation.

Results

Description of buzzer response. The buzzer, of course, elicited its own pattern of responses. This pattern usually consisted of a brief head jerk or head movement followed by freezing (lack of overt activity), often for extended periods of time. This pattern usually became less noticeable across trials and across sessions, showing an habituation effect. For example, the head jerk usually decreased in vigor or disappeared, and the period of freezing became shorter or was replaced by head movement.

Effects of sessions. The means for the four major dependent variables, plus mean numbers of HSRs for trials 1, 2, and 11, are presented as a function of Sessions in Table 1.

TABLE 1.--Means, *F*s, and *p*-values for 7 Dependent Variables as a Function of Sessions for the Between-Trial Experiment.

Dependent variable	Session						
	1*	2	3	4	5*	*F*	*p*
# HSRs, Trial 1	9.16	8.24	8.36	8.20	8.16	1.93	NS
# HSRs, Trial 2	5.63	4.39	3.40	4.23	5.03	9.43	.001
# HSRs, Trial 11	3.17	2.60	3.17	3.06	3.30	1.30	NS
# HSRs per buzzer trial	4.02	2.81	3.11	3.45	3.89	18.48	.001
# HSRs, 1st 2.5 sec. time period	.68	.57	.73	.73	.73	2.78	NS
Latency, first HSR per trial	4.60	8.02	6.46	5.01	4.10	9.55	.001
Rate of within-trial habituation	2.04	1.56	1.78	1.77	1.88	6.75	.001

*Control sessions.

Analysis of variance revealed no significant effects of Sessions on the number of HSRs for trial 1 or trial 11 (see Table 1), indicating that initial and terminal levels of responding did not change across sessions. However, a significant Sessions X Time Periods interaction for the data of trial 1 ($F = 2.18$, $df = 44/1276$, $p < .01$) indicated that the pattern of responding within the first trial varied as a function of sessions. The within-trial patterns can be seen in Figure 1, which shows the mean number of HSRs as a function of 2.5-sec. time periods for all five sessions. Data from both trial 1 and 11 are presented. Inspection of Figure 1 reveals that responding during the first trial of the first session increased initially, then decreased after the first 10 sec. The explanation for this initial increase most likely lies in the fact that the onset of the first presentation of the air stimulus tended to elicit strong orienting or exploratory behavior, which presumably competed with head-shaking. The phenomenon occurred somewhat less strongly again on the first trial of the second session, but it had disappeared by the third session. The lack of a significant Sessions X Time Periods interaction for the data from trial 2 suggests that the initial increase in responding was limited to the first trial. It should be noted that significant within-trial response decrement was still

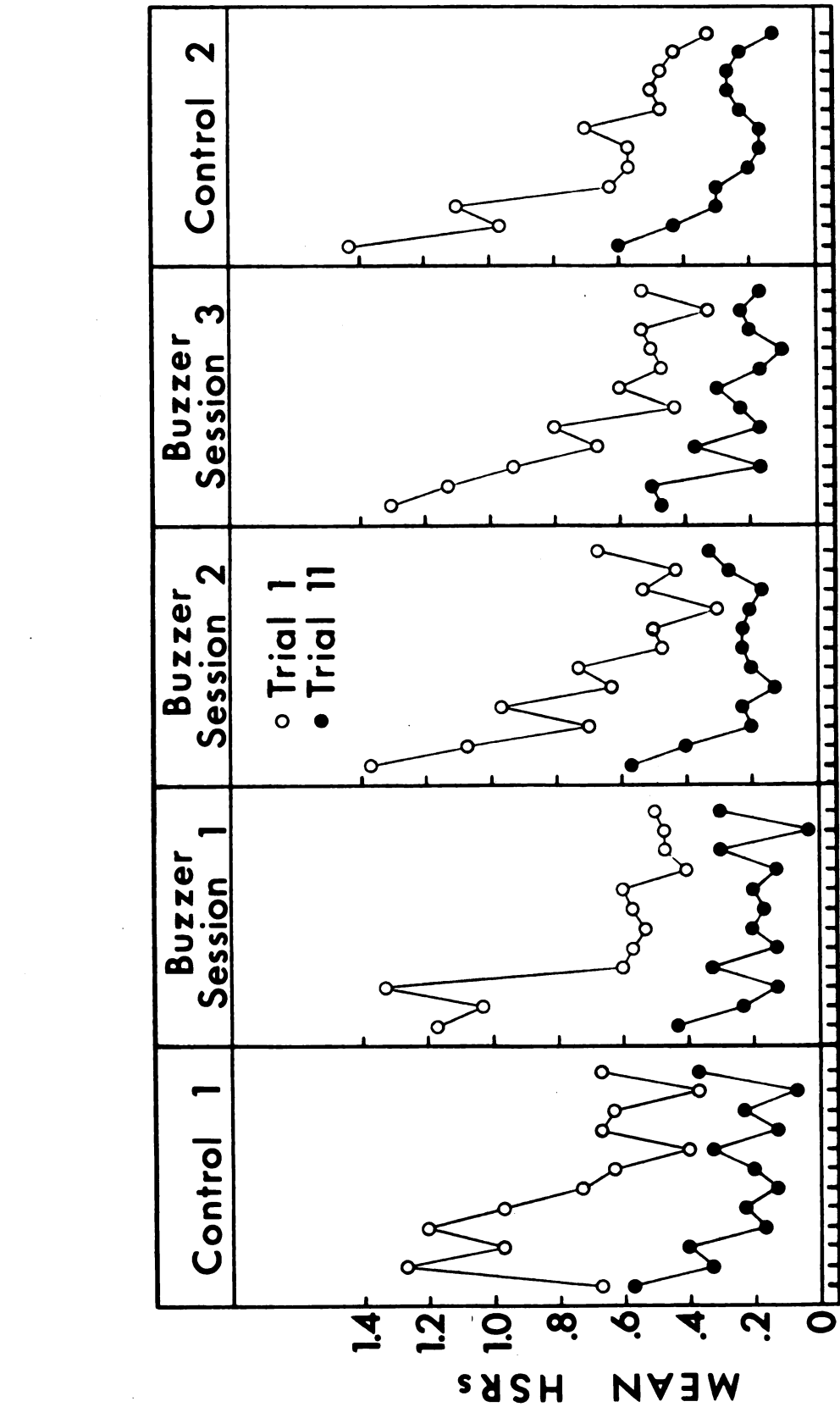

Figure 1. Mean numbers of HSRs as a function of 2.5-sec. within-trial time periods for control trials 1 and 11 of each session (between-trial experiment).

occurring on the last trial of each session (F = 8.40, df = 11/319, p < .001).

With the introduction of the buzzer on the second, third, and fourth sessions, responding was substantially reduced, as shown by the effect of Sessions on number of HSRs per trial (F = 18.48, df = 4/116, p < .001). The means for the three buzzer sessions were all significantly lower than either of the means for the two control sessions, while the latter two means did not differ significantly. Among the means for the three dishabituation sessions a general trend towards increased responding across sessions was noted, with the mean for Buzzer Session 3 being significantly higher than that for Buzzer Session 1. This suggests that the response depression (due presumably to the introduction of the buzzer) which appeared on Buzzer Session 1 gradually became less severe with continued exposure to the buzzer. When the buzzer was withheld on session 5, response level was not significantly different from what it had been on session 1. However, it is possible that a decrease in responding would have occurred with mere repetition of habituation sessions and was not, in fact, correlated with introduction of the buzzer. To test this possibility, 9 of the 30 Ss were given an additional set of control sessions 10 days after the end of the major experiment. Three sessions, identical to the no-buzzer

control sessions administered earlier, were given at 24-hr. intervals. The mean numbers of HSRs per buzzer trial for these three sessions were 3.91, 4.25, and 4.27, respectively. There were no significant differences among these means, nor were they significantly different from the means of the two previous control sessions. This lack of carry-over effects of sessions repeated at 24 hr. intervals has been reported in two previous experiments (Askew et al., 1969; Leibrecht and Askew, in press) and indicates that the response depression during dishabituation sessions was, in fact, due to introduction of the buzzer. The significant lessening of this depression across the three dishabituation sessions shows that habituation of the buzzer's inhibitory or competing effects occurred.

The reduction in responding during dishabituation sessions was accompanied by a significant increase in latency of the first response ($F = 9.55$, $df = 4/116$, $p < .001$). Mean latency showed a significant increase from Control Session 1 to Buzzer Session 1, then gradually decreased with continued sessions towards control level (see Table 1), there being no significant difference between the mean of Buzzer Session 3 and either of the means for the two control sessions. The means for the two control sessions did not differ significantly.

A significant effect of Sessions also occurred for rate of within-trial habituation (F = 6.75, df = 4/116, p < .001). A multiple range test revealed that mean rate decreased significantly from Control Session 1 to Buzzer Session 1, then returned nearly to control level by Buzzer Session 2 (only the mean for Buzzer Session 1 being significantly different from the means for the control sessions). These results show that introducing the buzzer produced faster within-trial habituation, the effect being significant only for the first dishabituation session. As with previous dependent variables, there was no significant difference between the means for the two control sessions, indicating that the three intervening dishabituation sessions had no lasting effect on performance under no-buzzer control conditions.

In order to ascertain possible across-session changes in base rate (no air stimulus) responding, two measures were investigated. These were the number of HSRs in the initial 5-min. adaptation period of each session and the number of HSRs during the 10 30-sec. inter-trial intervals. The means for the initial adaptation periods were .27, .34, .43, .36, and .48 HSRs per 30-sec. interval for sessions 1-5, respectively. A significant effect of Sessions (F = 3.83, df = 4/116, p < .01) indicated that this index of base rate responding increased significantly across sessions. This is

contrary to previous findings reported by Askew et al. (1969). The means for base rate responses during inter-trial intervals were .17, .05, .06, .12, and .18 HSRs per interval for sessions 1 through 5, respectively. Although Ss had as much time to respond during the 10 inter-trial intervals as they did during the initial adaptation period, significantly fewer HSRs occurred after presentations of the air stimulus began ($F = 9.49$, $df = 1/29$, $p < .005$). This reduction in base rate responding during periods interspersed between presentations of the air stimulus did not occur in a previous experiment (Askew et al., 1969). While the reduction appears to be greater during the three dishabituation sessions, the same pattern occurred during the three post-experimental control sessions. Therefore, no statement concerning a possible relation between presence of buzzer and degree of reduction in base rate responding can be made.

Summary of results on effects of sessions. The results of the foregoing section failed to provide evidence that the buzzer produced dishabituation of the head-shake response. Rather, while initial and terminal response levels did not change across sessions, introduction of the buzzer during dishabituation sessions produced a general reduction in responding. This depression was reflected in a reduced number of HSRs

per trial, an increased latency of the first response per trial, and a faster rate of within-trial habituation. These effects were greatest during the first dishabituation session and returned to control levels during succeeding sessions.

Effects of trials. The means for the four major dependent variables are presented in Table 2 as a function of 3-trial blocks (it will be recalled that each session contained nine dishabituation trials arranged in three blocks of three trials each). Means for the three dishabituation sessions combined and the two control sessions combined are given separately. Data for the control sessions were analyzed separately from data for the dishabituation sessions.

A significant decrement in the number of HSRs per trial occurred across trial blocks during dishabituation sessions ($F = 14.11$, $df = 2/58$, $p < .001$) and during control sessions ($F = 41.75$, $df = 2/58$, $p < .001$). However, a significant Sessions X Trial Blocks interaction among the three dishabituation sessions ($F = 4.29$, $df = 4/116$, $p < .005$) indicated that the across-trial pattern of responding varied from session to session. Across-trial patterns can be seen in Figure 2, which shows the mean numbers of HSRs per trial as a function of Trial Blocks for separate sessions. The data from the two control sessions have been combined, since

TABLE 2.--Means, Fs, and p-values for 4 Dependent Variables as a Function of 3-Trial Blocks for the Between-Trial Experiment, with Means for Control Sessions in Parentheses.

Dependent Variable	Trial Block				
	1	2	3	F	p
# HSRs per buzzer trial	3.50 (4.59)	3.04 (3.74)	2.83 (3.52)	14.11 41.75	.001 .001
# HSRs, 1st 2.5 sec. time period	.76 (.87)	.68 (.62)	.59 (.63)	7.19 12.63	.005 .001
Latency, first HSR per trial	6.39 (3.43)	6.07 (4.91)	7.03 (4.70)	1.33 4.45	NS .025
Rate of within-trial habituation	1.72 (2.12)	1.77 (1.95)	1.61 (1.81)	2.03 4.00	NS .025

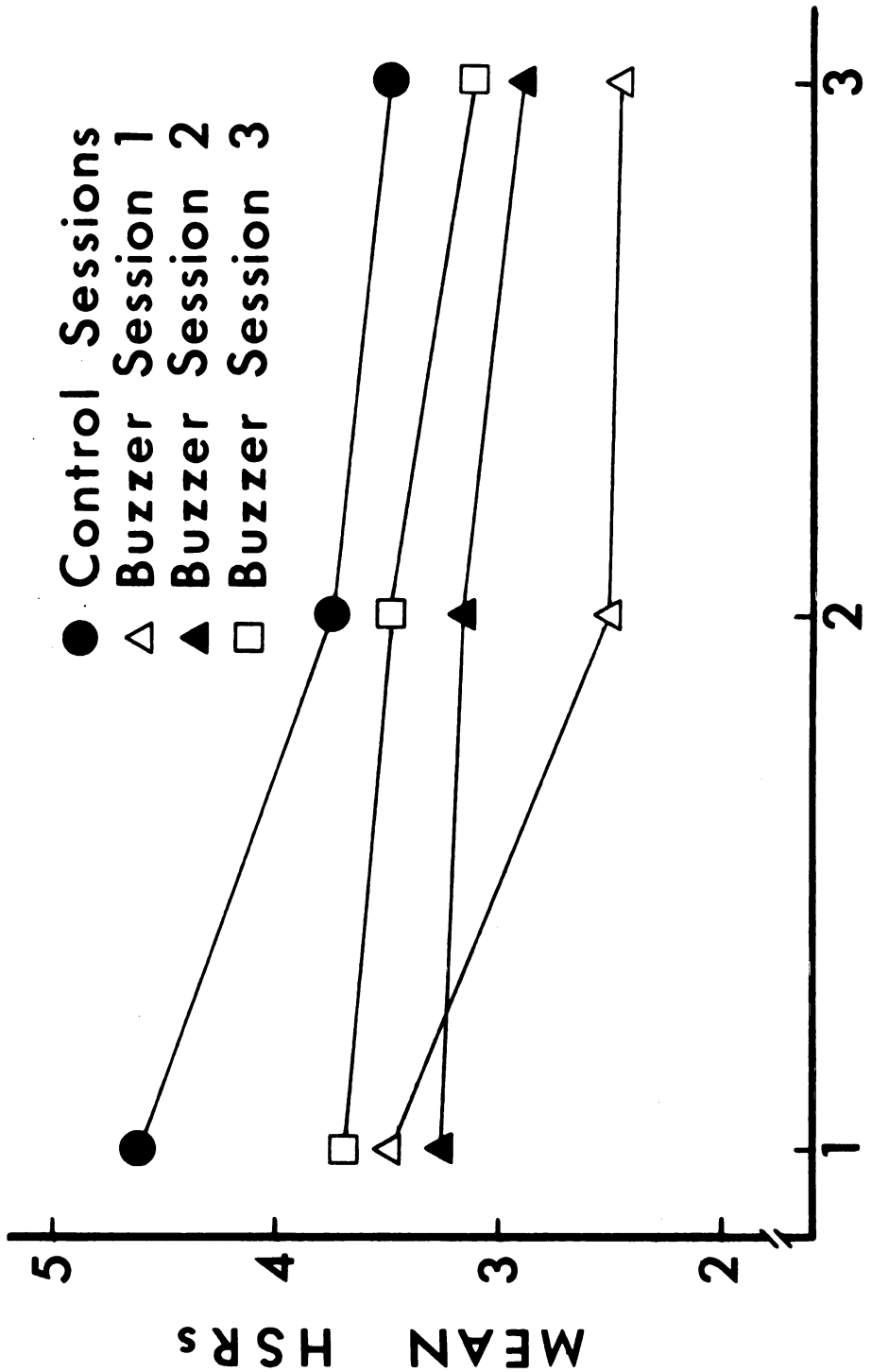

Figure 2. Mean numbers of HSRs per trial as a function of 3-trial blocks for each buzzer session and for combined control sessions (between-trial experiment).

neither the Sessions effect nor the Sessions X Trial Blocks interaction was significant for these sessions. It can be seen from Figure 2 that considerable across-trial decrement occurred during the first dishabituation session, and in a manner similar to that during the control sessions. However, the amount of across-trial decrement was substantially less during Buzzer Sessions 2 and 3. In fact, the decrement across trials was not significant for either Buzzer Session 2 or 3. These results suggest that, while introduction of the buzzer failed to modify the pattern of across-trial responding during Buzzer Session 1 (although the entire curve was lowered), the presence of the buzzer on the following two sessions significantly retarded across-trial decrement in frequency.

The number of HSRs in the first 2.5-sec. time period of each trial showed significant decrement across trial blocks during dishabituation sessions ($F = 7.19$, $df = 2/58$, $p < .005$) as well as control sessions ($F = 12.63$, $df = 2/58$, $p < .001$). The pattern was essentially the same during dishabituation and control sessions and did not change significantly with repeated buzzer sessions. This index, therefore, reflects no modification by the presence of the buzzer.

Neither latency of the first response per trial nor rate of within-trial habituation showed significant

changes across trial blocks during control or dishabituation sessions (see Table 2).

Summary of effects of trials. The retardation of across-trial frequency decrement during the second and third dishabituation sessions provides the only trial-block evidence for dishabituation. Changes in the number of HSRs in the first period of each trial were similar for both control and buzzer sessions. Latency and rate of within-trial habituation during buzzer sessions showed the same lack of significant across-trial changes as during control sessions.

Effects of buzzer positions. It will be recalled that buzzer presentations preceded dishabituation trials by 1, 15 or 29 sec. These three positions will be referred to as the 1-sec., 15-sec., or 29-sec. buzzer positions, respectively. The means for the four major dependent variables appear in Table 3 as a function of Buzzer Positions. Data have been combined for the three dishabituation sessions and for the two control sessions. Data from the dishabituation sessions were analyzed together, while data from the control sessions were analyzed in a separate AOV.

No significant effect of Buzzer Positions was found for any of the four dependent variables during control sessions (see Table 3). On the other hand, a significant Buzzer Positions effect was found for all

TABLE 3.--Means, *F*s, and *p*-values for 4 Dependent Variables as a Function of Between-Trial Buzzer Positions, with Means for Control Sessions in Parentheses.

Dependent variable	Buzzer Position (sec. preceding trial onset)				
	1"	15"	29"	*F*	*p*
# HSRs per buzzer trial	3.35 (3.82)	3.10 (3.98)	2.92 4.07)	7.97 1.33	.001 NS
# HSRs, 1st 2.5 sec. time period	.84 (.72)	.62 (.69)	.57 (.70)	14.69 <1	.001 NS
Latency, first HSR per trial	5.03 (4.36)	7.22 (4.27)	7.25 (4.42)	7.43 <1	.005 NS
Rate of within-trial habituation	1.84 (2.03)	1.65 (1.92)	1.62 (1.93)	7.09 <1	.005 NS

four dependent variables during dishabituation sessions (see Table 3). The mean for the 1-sec. position was significantly different from the means for the other two positions on all variables except the number of HSRs per trial, where only the means for the 1-sec. and 29-sec. positions differed significantly. In no case were the means for the 15-sec. and 29-sec. positions significantly different. That is, the 1-sec. buzzer position produced a significantly greater number of HSRs per trial and per initial time period, a shorter latency, and a slower rate of within-trial habituation than did either the 15-sec. or 29-sec. positions.

The effects of the various buzzer positions on the within-trial pattern of responding can be seen in Figure 3. This figure presents the mean numbers of HSRs per 2.5-sec. time period as a function of buzzer positions for all five sessions. A progressive, negatively accelerated within-trial response decrement occurred on Control Session 1, with the end points of each curve generally lower than preceding points. Introduction of the buzzer accelerated the within-trial decrement and led to a period of suppressed responding, reaching a low point approximately 10-12 sec. after the onset of the trial. Following this low point, responding tended to increase, at least for the 1-sec. and 15-sec. buzzer positions. In order to test the significance

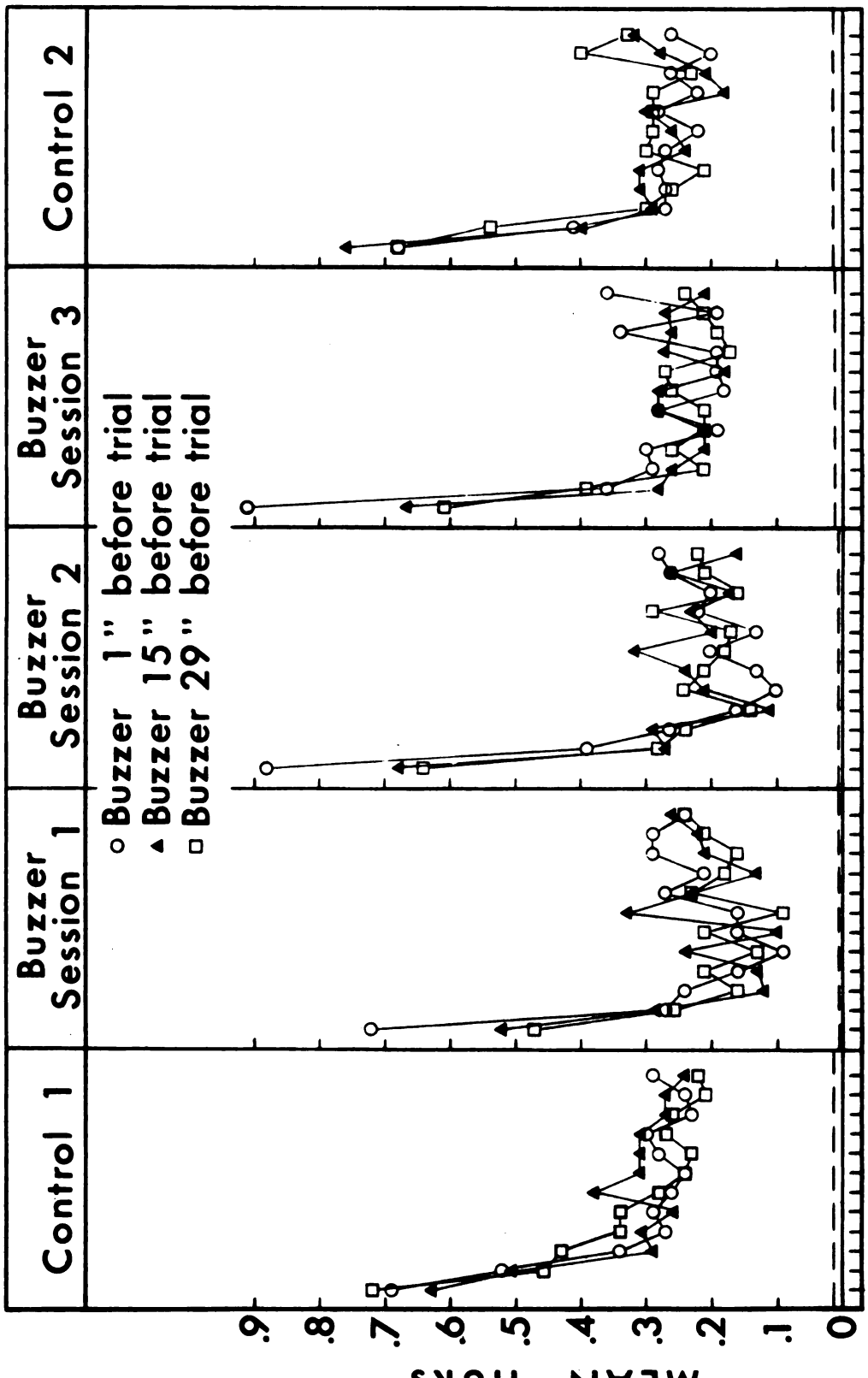

Figure 3. Mean numbers of HSRs as a function of 2.5-sec. within-trial time periods and buzzer positions for each session (between-trial experiment).

of this increasing trend, HSRs were summed across time periods 4-6 (the second quarter of the trial) and compared with the number of HSRs summed across time periods 10-12 (the final quarter of the trial). A significant increase in responding from the second to the fourth quarter was shown by a significant effect of Time Period Blocks ($F = 7.72$, $df = 1/29$, $p < .01$). However, a significant Time Period Blocks X Buzzer Positions interaction ($F = 5.97$, $df = 2/58$, $p < .005$) indicated that changes in responding varied as a function of position of the buzzer. And a significant Time Period Blocks X Sessions interaction ($F = 6.33$, $df = 2/58$, $p < .005$) indicated that response patterns changed across sessions. A multiple range test revealed that during Buzzer Session 1 responding increased significantly from the second to the fourth quarter for the 1-sec. and 15-sec. buzzer positions, but not for the 29-sec. position. During Buzzer Session 2 a significant depression and subsequent increase in responding occurred only for the 1-sec. position, and by Buzzer Session 3 no significant depression and subsequent increase occurred for any of the buzzer positions.

Summary of effects of buzzer positions. The finding that the 1-sec. buzzer position resulted in a greater frequency of HSRs, a shorter latency, and a slower rate of within-trial habituation than did the

more remote positions suggests that presentation of the buzzer produced a temporary excitory or facilitatory effect, lasting only a few seconds. Fine-grain examination of the within-trial response patterns revealed a period of response suppression, reaching a low point about 10-12 sec. after the onset of the trial, for the 1-sec. and 15-sec. buzzer positions. This phenomenon disappeared across sessions.

Intercorrelations. Product-moment correlations among frequency measures showed that animals tended to maintain their relative standings with respect to response level across time. In considering the following results, it should be noted that *rs* equal to or greater than .45 are significant at the .01 level. However, the collective probability level is somewhat greater than .01 with multiple correlations. Initial response level (number of HSRs on trial 1 of session 1) correlated significantly with the total number of HSRs summed across dishabituation trials (r = .40 to .76) and with mean rate of within-trial habituation (r = .38 to .65). The latter result indicates that high responders tended to show slower within-trial habituation.

Correlations comparing the same dependent variable across sessions showed a moderate to high level of reliability. Initial response level (number of responses in the first trial) correlated moderately across

sessions (r = .45 to .68). Greater reliability was found for total number of HSRs summed across buzzer trials (r = .58 to .84).

Conditioning effects. Two contingencies within the experimental procedure constituted conditions conducive for the occurrence of conditioning effects. The first of these was the rhythmic schedule of events set up by the use of a fixed trial duration and a fixed inter-trial interval. This regular pattern could have led to the establishment of temporal conditioning, which might, for example, have maintained an inflated rate of responding. In addition, the visual and auditory components accompanying the actual air stimulus may have become conditioned elicitors of HSRs. In order to test these possibilities, both control sessions were modified to include a 12th "trial" during which the air stimulus was moved back and forth near the ear with the actual air stream directed away from the ear. Visual and auditory components were thus present, but mechanical components of the air stream were eliminated. If classical and/or temporal conditioning had taken place, such presentations should have produced HSRs at a level greater than base rate, at least on Control Session 2. However, no such pattern occurred. On Control Session 1, 6 Ss gave 7 HSRs during the probe trial, while 6 Ss gave 6 HSRs during the immediately preceding non-stimulated inter-trial interval. On

Control Session 2, 4 Ss gave 5 HSRs during the probe trial, while 5 Ss gave 5 HSRs during the preceding ITI. In addition to indicating no differences between non-stimulated base rates and "stimulated" probe trial rates, these data are notable in that the majority of animals gave no HSRs during the probe trial on either session.

The second conditioning contingency arose from the fact that during dishabituation sessions the buzzer preceded onset of the air stimulus by 1 sec. on three occasions. After repeated pairings of this type, the buzzer may have become a conditioned elicitor of HSRs. If this had occurred, HSRs should have followed the 15-sec. and 29-sec. buzzer presentations by a few sec., especially during the last buzzer session. However, only 6 of 37 base rate responses on Buzzer Session 3 followed the 15-sec. and 29-sec. buzzer presentations by 5 sec. or less, which is no greater than chance expectancy. In addition, only 6 Ss gave more than 1 response during inter-trial intervals of Buzzer Session 3. This shows that animals had only a very weak tendency to give HSRs during inter-trial intervals.

EXPERIMENT 2: WITHIN-TRIAL DISHABITUATION

The second experiment involved the presentation of the extraneous stimulus during presentations of the air stimulus. This procedure was suggested chiefly by pilot observations that the within-trial frequency decrement could be reliably disrupted by a novel stimulus. The procedure offered the opportunity to examine the effects of the extraneous stimulus continuously following its application. In order to vary the relative strength of the habituating head-shake response, the extra stimulus was presented with a variable delay following the onset of the air stimulus. An inverse relation between the degree of habituation and the ease of producing dishabituation has been reported by Kozak and Westerman (1966) and Prosser and Hunter (1936). As in Experiment 1, the extraneous stimulus was repeated three times in each position to test for habituation of dishabituation, and repeated sessions tested for long-term changes in the dishabituation process.

Method

Subjects. Thirty male Holtzman albino rats, 80 days of age, served as Ss. All were experimentally naive,

and testing did not begin until 1-2 wk. following arrival in the laboratory. Animals were housed in groups of 4-5 per cage under conditions of constant light and temperature, with water and food available ad lib.

Apparatus. The test stand, test stimulus, dishabituatory stimulus, and programming and recording equipment remained the same as in Experiment 1.

Procedure. The head-shake response was elicited in the manner described for Experiment 1.

The standard dishabituation session began with a 5-min. unstimulated adaptation period on the test stand, followed by a 30-sec. presentation of the air stimulus. There then occurred a series of nine dishabituation trials during which the buzzer was presented 5, 15, or 25 sec. following the onset of the air stimulus. Trials were continued for 30 sec. following the dishabituatory stimulus in order to provide a standard period of sufficient length for post-buzzer observation. Total trial length was therefore variable (35, 45, or 55 sec.), depending on when the buzzer was presented with respect to the beginning of the trial. The nine dishabituation trials were arranged in blocks of three trials, with each block containing one of each of the three types of trials. Buzzer positions were counter-balanced across trial blocks according to a within-subjects Latin square

procedure. A total of 12 such counterbalanced schedules were possible, and these were randomly assigned with the restriction that no S receive the same schedule twice. A final 30-sec. presentation of the air stimulus, with no buzzer presentation, followed the last dishabituation trial. A total of 11 presentations of the air stimulus thus constituted a single experimental session. A fixed 30-sec. inter-trial interval was employed.

Each S received two control sessions, one preceding and one following the three dishabituation sessions. Control sessions were identical to the dishabituation sessions, except that power to the buzzer was disconnected and no dishabituatory stimulus was ever presented. All sessions were separated by an interval of 24 hr.

The remaining experimental procedures were the same as those used in Experiment 1. Number of head-shake responses was obtained from the recording tapes with the aid of a transparent plastic template, which divided each trial into 2.5-sec. time periods. In addition, the latency of the first post-buzzer response in each trial was measured with a transparent plastic ruler.

Five major dependent variables were used in analyzing the results of Experiment 2. These were (a) the number of HSRs in the first post-buzzer 2.5-sec. time period of each dishabituation trial, (b) the total number of HSRs in the 30-sec. post-buzzer period of each trial,

(c) the latency of the first post-buzzer response in each trial (measured from the onset of the buzzer presentation), (d) the rate of re-habituation following each buzzer presentation, and (3) the amount of dishabituation on each trial. The rate of re-habituation was determined by dividing the post-buzzer period into six intervals of 5 sec. each and noting the first interval in which the number of HSRs was less than or equal to the number in the last interval. The amount of dishabituation was obtained by subtracting the number of HSRs in the 2.5-sec. time period immediately preceding the buzzer from the number of HSRs in the first 2.5-sec. post-buzzer time period.

As in the first experiment, the assumptions prerequisite to the use of AOV procedures appeared to be adequately met. Therefore, AOV procedures were employed as the chief statistical tool. Subjects (a random factor), and Sessions, Trials, and Buzzer Positions (fixed factors) were the usual category variables employed in a single-entry-per-cell design (Winer, 1962, p. 290). Under this procedure, main fixed effects were tested by using the double interaction with Subjects as the denominator in the F-ratio. Similarly, double interactions involving fixed factors were tested against the corresponding triple interactions with Subjects, and the fixed-factors triple

interaction was tested against the four-way interaction. Since a single observation per cell did not permit a within-cell variance estimate, the Subjects effect and interactions involving Subjects could not be tested. Duncan's (1955) multiple range test was used for individual comparisons. Since distributions frequently tended to be J-shaped and homogeneity of variance in some cases appeared questionable (especially for latency and re-habituation rate), the .01 level of significance was generally used. As in the first experiment, the over-all probability level was somewhat greater than .01 since multiple AOVs were used.

As in Experiment 1, presentation of results has been arranged according to independent variables. Effects of Sessions are considered first, followed by Trials effects. Finally, more specific questions concerning the effects of Buzzer Positions are taken up.

Results

Description of buzzer response. The response pattern elicited by the buzzer was essentially the same as was described in Experiment 1. A brief head jerk or movement of the head was followed by freezing, which often lasted 10-15 sec. or longer. The pattern usually showed an habituation effect, including changes in topography, across trials and across sessions.

Effects of sessions. Table 4 presents the means for the five major dependent variables, plus mean numbers of HSRs for trials 1 and 11 and the post-buzzer period of trial 2, as a function of Sessions.

Analysis of variance revealed a significant effect of Sessions on the number of HSRs for trial 1 (F = 11.19, df = 4/116, $p < .001$) and trial 11 (F = 3.96, df = 4/116, $p < .005$). Responding on trial 1 decreased significantly from the first to the second session and decreased less rapidly on the two succeeding sessions, with the difference between sessions 2 and 4 being significant. Responding during trial 11 was significantly reduced only on Buzzer Session 1 (due presumably to the introduction of the buzzer), there being no significant differences among the means for the other four sessions. A significant Sessions X Time Periods interaction for the data of trial 1 (F = 2.74, df = 44/1276, $p < .001$) indicated that the pattern of responding within the first trial varied across sessions. The within-trial patterns can be seen in Figure 4, which shows the mean numbers of HSRs as a function of 2.5-sec. time periods for all five sessions. Data from trials 1 and 11 are presented in separate curves. Inspection of Figure 4 reveals that, as in Experiment 1, an initial increase in responding preceded the typical within-trial response decrement during

TABLE 4.--Means, Fs, and p-values for 8 Dependent Variables as a Function of Sessions for the Within-Trial Experiment.

Dependent variable	Session						
	1*	2	3	4	5*	F	p
# HSRs, Trial 1	10.87	9.50	8.91	8.36	8.56	11.19	.001
# Post-buzzer HSRs, Trial 2	4.66	3.73	2.73	3.06	3.73	7.46	.001
# HSRs, Trial 11	3.47	2.63	3.23	3.27	3.57	3.96	.005
# Post-buzzer HSRs per trial	3.46	2.71	2.77	2.86	3.17	10.79	.001
# HSRs, 1st post-buzzer period	.30	.34	.56	.63	.27	20.92	.001
Latency, first post-buzzer HSR	6.57	8.91	7.31	6.10	7.29	4.21	.005
Post-buzzer rate of re-habituation	1.49	1.51	1.50	1.61	1.39	1.73	NS
Amount of dishabituation	-.15	.14	.35	.39	-.08	25.51	.001

*Control sessions.

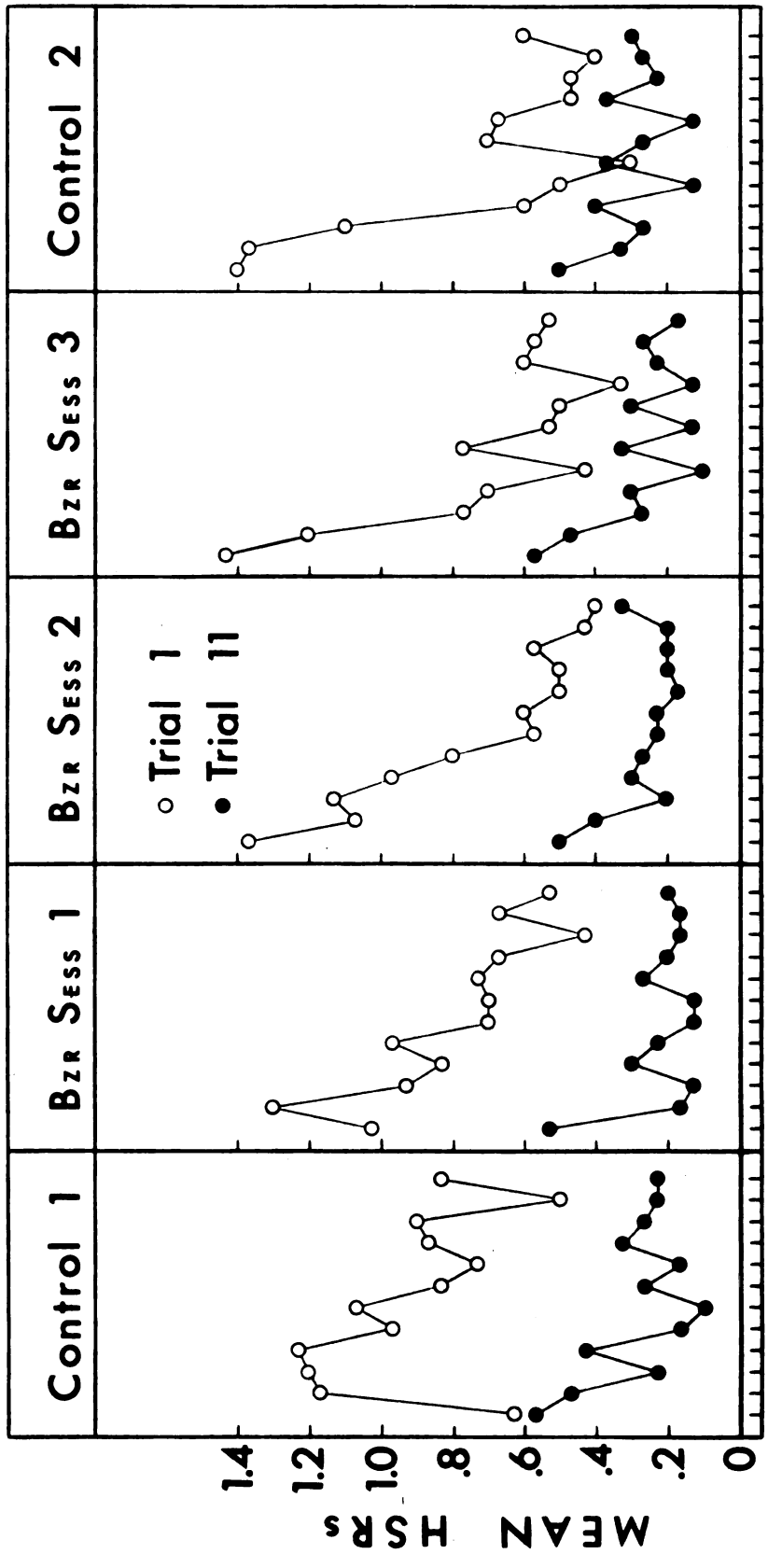

Figure 4. Mean numbers of HSRs as a function of 2.5-sec. within-trial time periods for control trials 1 and 11 of each session (within-trial experiment).

the first trial of the first two sessions. The phenomenon failed to appear on the remaining sessions.

Introduction of the buzzer substantially reduced responding, as shown by a significant effect of Sessions on number of post-buzzer HSRs per trial (F = 10.79, df = 4/116, $p < .001$). The means for all three buzzer sessions were significantly lower than either of the means for the two control sessions, while the latter two means did not differ significantly. A trend towards increased responding across the three dishabituation sessions was not significant. Although response level returned to control level when the buzzer was withheld on Control Session 2, it is still possible that mere repetition of habituation sessions, rather than introduction of the buzzer, was responsible for the reduced responding during dishabituation sessions. As in Experiment 1, this possibility was tested by giving 9 of the 30 Ss three additional control sessions 10 days after the end of the major experiment. These sessions were identical to the no-buzzer control sessions administered earlier and were given at 24-hr. intervals. The mean numbers of post-buzzer HSRs per trial for these three sessions were 3.12, 3.25, and 3.31, respectively. There were no significant differences among these means, nor were they significantly different from the means of the two previous control sessions. These results, similar to those reported in

Experiment 1, indicate that the over-all response depression during buzzer sessions was, in fact, due to introduction of the buzzer. It should be noted, with respect to the earlier mentioned across-session decrement in first-trial responding, that the first-trial means for the three post-experimental control sessions (8.67, 9.22, and 9.00, respectively) showed no across-session decremental trend.

The reduction in post-buzzer responding during dishabituation sessions was accompanied by a significant increase in the latency of the first post-buzzer response ($F = 4.21$, $df = 4/116$, $p < .005$). Mean latency increased significantly from Control Session 1 to Buzzer Session 1, then decreased gradually with continued sessions toward control level (see Table 4), there being no significant difference between the mean of Buzzer Session 3 and either of the means for the two control sessions. The two control means did not differ significantly.

While the number of post-buzzer HSRs and the latency of the first post-buzzer response both showed a general depression of responding during dishabituation sessions, the number of HSRs in the first post-buzzer 2.5-sec. time period showed a significant increase during the same sessions ($F = 20.92$, $df = 4/116$, $p < .001$). Although the increase during Buzzer Session 1 was not significant, the means for Buzzer Sessions 2 and 3 were significantly

higher than the mean for Buzzer Session 1, but the former were not significantly different. The data for number of HSRs in the first post-buzzer period and for amount dishabituation thus provide evidence that significant dishabituation occurred on all three buzzer sessions, with significantly less dishabituation occurring during Buzzer Session 1 than during subsequent sessions.

No significant effect of Sessions was found for rate of re-habituation (see Table 4).

Two measures of base rate responding were investigated. These were the number of HSRs in the initial 5-min. adaptation period of each session and the number of HSRs during the 10 30-sec. inter-trial intervals. The means for the initial adaptation periods were .24, .24, .25, .35, and .49 HSRs per 30-sec. interval for sessions 1-5, respectively. As in Experiment 1, this index of base rate responding increased significantly across sessions ($F = 4.07$, $df = 4/116$, $p < .005$). The means for base rate responses during inter-trial intervals were .04, .03, .08, .18, and .17 HSRs per interval for sessions 1-5, respectively. Significantly fewer HSRs occurred during inter-trial intervals than during the initial adaptation period ($F = 12.23$, $df = 1/29$, $p < .005$), indicating that base rate responding was depressed between alternating presentations of the air stimulus. The same pattern was found in Experiment 1.

Summary of results on effects of sessions.

Frequency of HSRs during trial 11 and during 30-sec. post-buzzer periods, as well as latency of the first post-buzzer response, indicated that introduction of the buzzer during dishabituation sessions produced a general reduction in responding. At least partial remission of this effect occurred with repeated buzzer sessions. On the other hand, frequency of responses during the first 2 1/2 sec. following the buzzer and amount of dishabituation both showed that significant dishabituation occurred during all three buzzer sessions, with greater dishabituation occurring during Buzzer Sessions 2 and 3 than during Buzzer Session 1.

Effects of trials. It will be recalled that each buzzer position was repeated three times during each session, appearing once in each block of three trials. Table 5 presents the means for the five major dependent variables as a function of 3-trial blocks. Means for the three dishabituation trials combined and the two control sessions combined are given separately. Data for the control sessions were analyzed separately from data for the dishabituation sessions.

A significant decrement in the number of post-buzzer HSRs occurred across trial blocks during control sessions ($F = 26.24$, $df = 2/58$, $p < .001$), but not during buzzer sessions (see Table 5). The lack of significant

TABLE 5.--Means, Fs, and p-values for 5 Dependent Variables as a Function of 3-Trial Blocks for the Within-Trial Experiment, with Means for Control Sessions in Parentheses.

Dependent variable	Trial Block			F	p
	1	2	3		
# Post-buzzer HSRs	2.97 (3.82)	2.68 (3.19)	2.69 (2.93)	3.96 26.24	.025 .001
# HSRs, 1st post-buzzer period	.47 (.26)	.54 (.33)	.53 (.25)	1.10 1.64	NS NS
Latency, first post-buzzer HSR	8.20 (6.05)	7.36 (6.76)	6.77 (7.98)	1.77 6.27	NS .005
Post-buzzer rate of re-habituation	1.52 (1.59)	1.62 (1.43)	1.48 (1.31)	2.00 6.76	NS .005
Amount of dishabituation	.17 (-.19)	.36 (-.04)	.35 (-.11)	5.52 1.77	.01 NS

across-trial decrement in post-buzzer responding during dishabituation sessions indicates that introduction of the buzzer retarded across-trial habituation.

A significant increase in the amount of dishabituation across trial blocks occurred during buzzer sessions ($F = 5.52$, $df = 2/58$, $p < .01$) but not during control sessions (see Table 5). A significant Sessions X Trials interaction ($F = 2.91$, $df = 4/116$, $p < .025$) among the dishabituation sessions indicated that across-trial patterns of dishabituation varied from session to session. The across-trial patterns can be seen in Figure 5, which shows the mean amount of dishabituation per trial as a function of Trial Blocks for the three buzzer sessions. Inspection of Figure 5 reveals that during the first trial block of the first dishabituation session the buzzer failed to produce dishabituation. In fact, the buzzer failed to interrupt the within-trial pattern of response decrement, as shown by a negative mean (-.09). However, significant dishabituation occurred during trial blocks 2 and 3 of Buzzer Session 1. During the second and third buzzer sessions significant dishabituation occurred on all trials; a trend toward increasing dishabituation across trials during Buzzer Session 2 was not significant. These results show that the first few presentations of the buzzer do not produce an increase in head-shake responding, but subsequent presentations

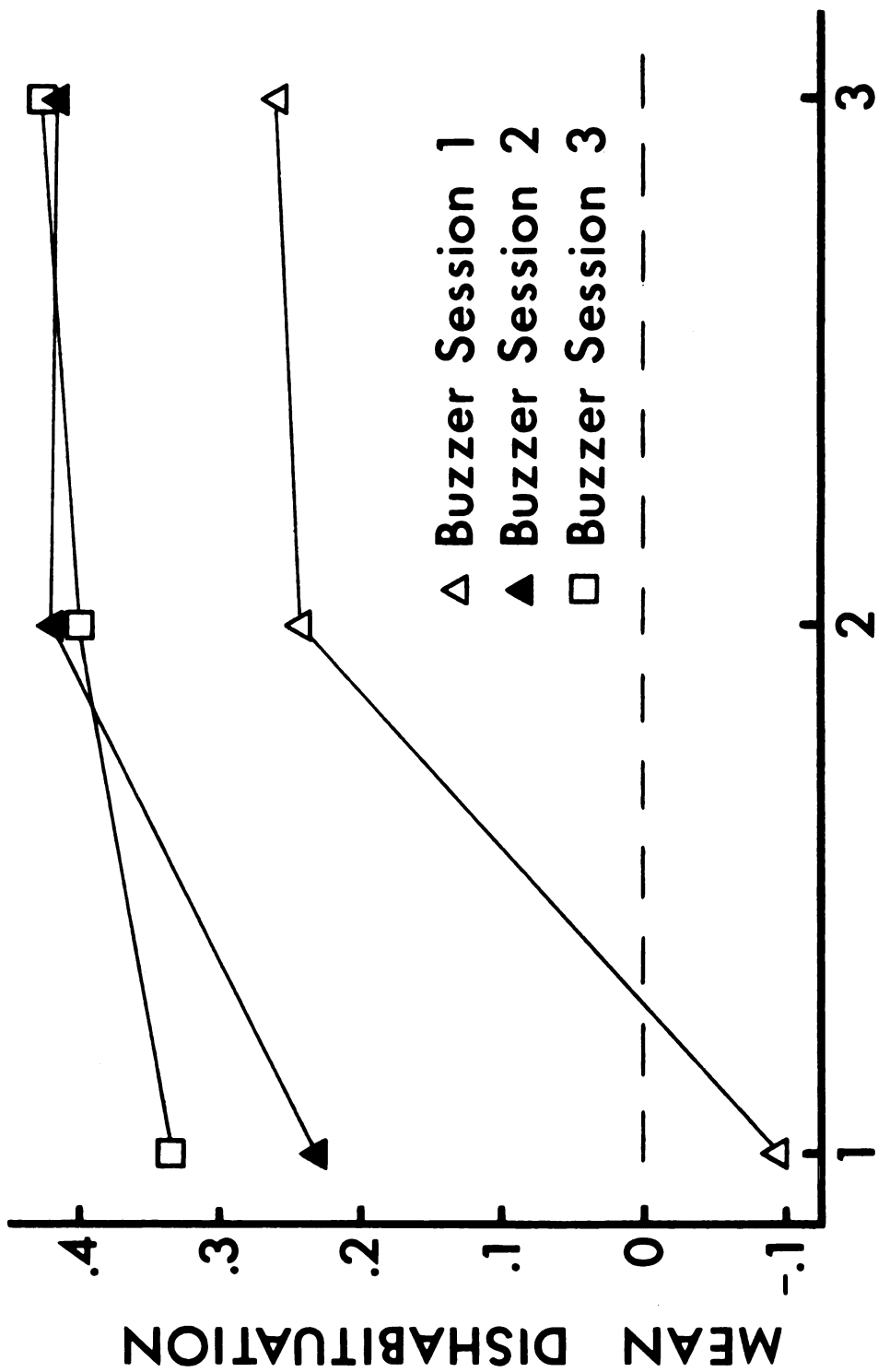

Figure 5. Mean amount of dishabituation as a function of 3-trial blocks for each buzzer session (within-trial experiment).

do produce significant dishabituation. This initial failure to produce dishabituation does not appear after the first buzzer session.

While the rate of re-habituation decreased significantly across trials during control sessions (F = 6.76, df = 2/58, p < .005), no significant decrease occurred during buzzer sessions (see Table 5). This indicates that presentation of the buzzer prevents the rate of post-buzzer re-habituation from becoming faster across trials.

While the latency of the first post-buzzer response increased significantly across trial blocks during control sessions (F = 6.27, df = 2/58, p < .005), no significant increase occurred during buzzer sessions (see Table 5). However, it is difficult to interpret this finding, since the general increase in latency during dishabituation sessions (as compared with control sessions) was opposite to the expected direction of change.

The number of HSRs in the first post-buzzer 2.5-sec. time period did not change significantly across trial blocks for either control sessions or dishabituation sessions (see Table 5).

Summary of results on effects of trials. Data for amount of dishabituation revealed an initial failure of the buzzer to produce dishabituation, which disappeared across trials and across sessions. In addition to the

immediate increase in responding, the buzzer also retarded across-trial decrement in the total number of post-buzzer HSRs and prevented the rate of post-buzzer re-habituation from decreasing across trials as it did during control sessions. No evidence for across-trial dishabituation effects was provided by the number of HSRs in the first post-buzzer time period or the latency of the first post-buzzer response.

Effects of buzzer positions. It will be recalled that the buzzer was presented during presentations of the air stimulus at points 5, 15, and 25 sec. following the onset of the trial. The means for the five major dependent variables appear in Table 6 as a function of these three Buzzer Positions. Data have been combined for the three dishabituation sessions and for the two control sessions. Data from the dishabituation sessions were analyzed together, while data from the control sessions were analyzed in a separate AOV.

There was no significant effect of Buzzer Positions during dishabituation sessions for any of the five major dependent variables. The effect of Buzzer Positions during control sessions was significant only for the number of post-buzzer HSRs per trial and the post-buzzer rate of re-habituation (see Table 6). Significant effects of Buzzer Positions during control sessions (when no buzzer was presented) were due to the fact that

TABLE 6.--Means, Fs, and p-values for 5 Dependent Variables as a Function of Within-Trial Buzzer Positions, with Means for Control Sessions in Parentheses.

Dependent variable	Buzzer Position (sec. following trial onset) 5"	15"	25"		
# Post-buzzer HSRs per trial	2.86 (3.67)	2.71 (3.26)	2.77 (3.02)	1.01 16.76	NS .001
# HSRs, 1st post-buzzer period	.52 (.36)	.50 (.28)	.52 (.21)	<1 4.35	NS .025
Latency, first post-buzzer HSR	7.92 (6.02)	7.78 (7.26)	6.63 (7.51)	1.96 4.79	NS .025
Post-buzzer rate of re-habituation	1.53 (1.61)	1.55 (1.42)	1.54 (1.29)	<1 7.90	NS .001
Amount of dishabituation	.22 (-.14)	.31 (-.09)	.34 (-.11)	2.14 <1	NS NS

buzzer points for the different positions were scheduled after differing amounts of within-trial habituation had taken place. However, the lack of a significant effect of Buzzer Positions on the number of HSRs in the first post-buzzer time period during control sessions (see Table 6) indicated that response strength did not actually vary greatly for the different buzzer positions.

The post-buzzer response patterns can be seen in Figure 6, which presents the mean numbers of HSRs per 2.5-sec. period as a function of buzzer positions. Data for all five sessions are included, and it is important to note that only the last 13 periods of each trial are represented. Means for the first 2.5-sec. period of each trial are also included (solid circles) to indicate the initial response level. Time period "P" represents the period immediately preceding the buzzer presentation, and arrows mark the point at which the buzzer was presented. A progressive decline in responding occurred throughout the latter portion of each trial during the control sessions. During dishabituation sessions, however, only a brief period of post-buzzer response decrement occurred, with responding tending to increase during the latter 15 sec. of the post-buzzer interval. This increasing tendency during the terminal portion of the trial is similar to the pattern found in Experiment 1. In order to test the significance of this increasing trend, HSRs

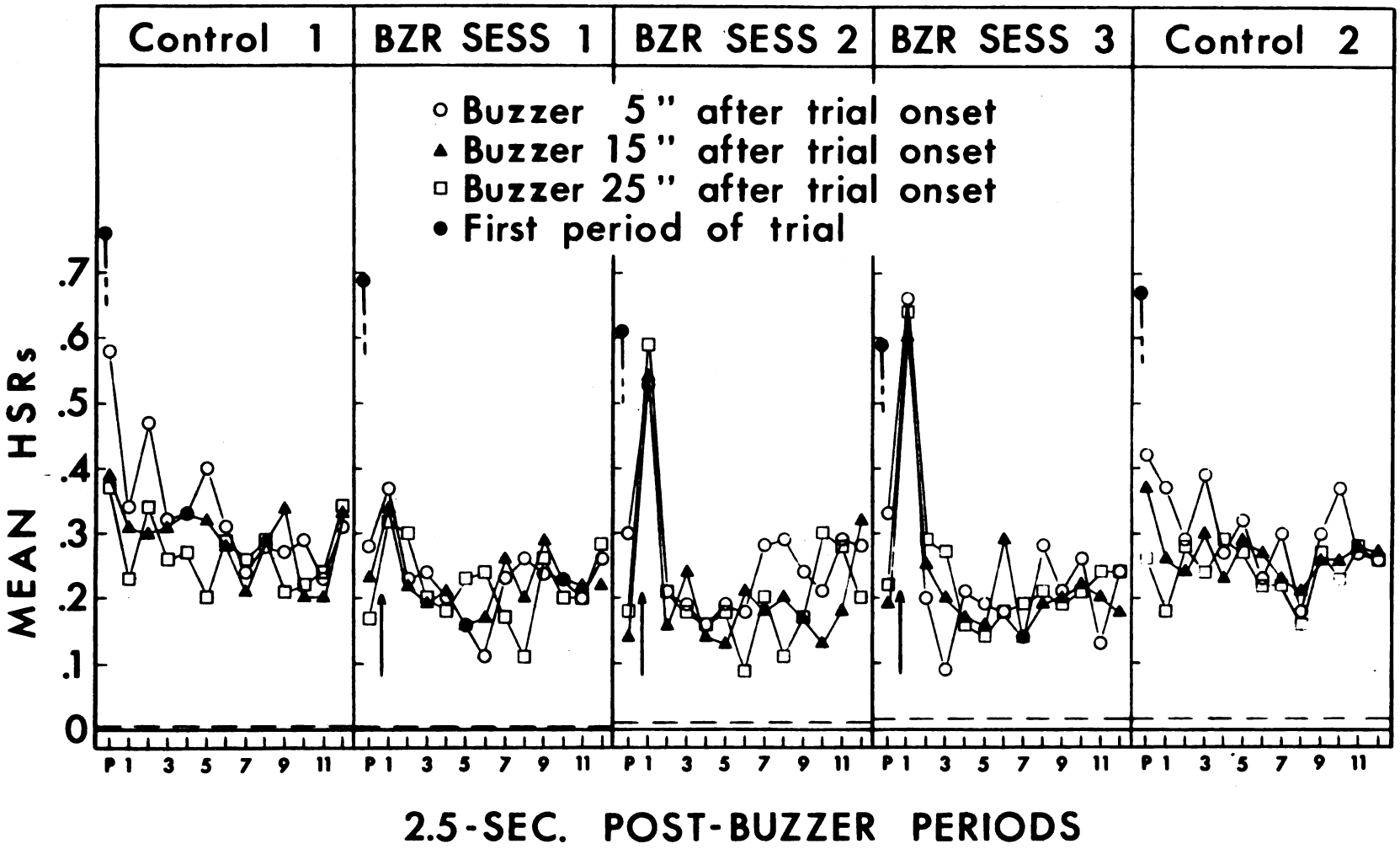

Figure 6. Mean numbers of HSRs for 2.5-sec. time periods following and immediately preceding (period "P") the buzzer as a function of buzzer positions for each session (within-trial experiment).

were summed across post-buzzer time periods 4-6 (the second quarter of the post-buzzer interval) and compared with the number of HSRs summed across post-buzzer time periods 10-12 (the final quarter of the post-buzzer interval). Since previously mentioned results showed no differences among buzzer positions on any post-buzzer index of responding, data were also summed across buzzer positions. A significant increase in responding from the second to the fourth post-buzzer quarter was shown by a significant effect of Time Period Blocks ($F = 9.13$, $df = 1/29$, $p < .01$). A significant Time Period Blocks X Sessions interaction ($F = 5.04$, $df = 2.58$, $p < .01$) indicated that response patterns changed across sessions. These changes were evaluated by means of a multiple range test, which revealed that responding increased significantly from the second to the fourth quarter during Buzzer Sessions 1 and 2, but not during the third Buzzer Session. These results show that a period of response depression followed the initial post-buzzer increase during the first two dishabituation sessions. This pattern was largely gone by the third dishabituation session.

Summary of results on effects of buzzer positions. The lack of significant differences among buzzer positions on any index of post-buzzer responding during dishabituation sessions was qualified by the lack of significant differences in response strength among the various

positions during control sessions. Fine-grain examination of the post-buzzer response patterns revealed that a period of response depression followed the initial post-buzzer increase in responding during the first two buzzer sessions but not during the third session.

Intercorrelations. A series of product-moment intercorrelations revealed that none of several variables, including the major dependent variables plus indexes of initial and over-all response levels, proved to be a good predictor of amount of dishabituation. Test-retest reliability for amount of dishabituation was generally low (r = .01 to .41; an r of .45 or greater is required at the .01 level), with the highest correlation between Buzzer Sessions 2 and 3. It should be pointed out that multiple correlations increase the collective probability level somewhat. As in Experiment 1, Ss tended to maintain their relative response levels within and across sessions. Initial response level (number of HSRs on trial 1 of session 1) correlated significantly with number of post-buzzer HSRs (r = .56 to .66) and with total HSRs per session (r = .63 to .76). Test-retest reliability was high for number of post-buzzer HSRs (r = .67 to .92) and for total HSRs per session (r = .81 to .95). The latter two variables correlated highly on all sessions (r = .93 to .97). Finally, initial level correlated significantly with rate of

within-trial habituation on session 1 (r = .45), showing that, as in Experiment 1, high responders tended to show slower within-trial habituation.

DISCUSSION

One of the strongest, yet most unexpected, findings of the present study was the general suppression of head-shake responding produced by the buzzer in both experiments. This was reflected primarily in a reduction in the frequency of head-shake responses and a substantial increase in latency measures. That these effects were not due simply to repetition of sessions was shown by the lack of across-session changes in initial or terminal response levels (not preceded or interrupted by the buzzer), and by the lack of changes in any frequency measure during the three post-experimental no-buzzer control sessions. The significant across-session decrease in first-trial responding for the within-trial subjects is difficult to explain. However, the same subjects showed no such pattern during the three post-experimental control sessions. Therefore, the present results largely substantiate previous findings of no carry-over effects with the sessions repeated at 24 hr. intervals (Askew et al., 1969; Leibrecht and Askew, in press).

The general response suppression produced by the buzzer suggests that response inhibition, rather than dishabituation, was occurring. Similar inhibition of

responding by novel stimuli has been reported by Rushforth (1965), Hernandez-Peon (1960), Jane, Smirnov, and Jasper (1962), and Buno et al. (1966). External inhibition during classical conditioning has been observed and studied by Pavlov (1927). Such inhibition could be due to a number of factors, such as afferent neural inhibition (Pribram, 1967), sensory gating at the reticular level (Buckland et al., 1969), or competing responses. The importance of the latter, especially in habituation procedures involving concurrent stimulation, has been emphasized by Ratner and Denny (1970). Their discussion points out that an extra stimulus may facilitate or retard habituation, depending on whether the response pattern produced by the extra stimulus competes or is compatible with the response to the habituating stimulus. In the present study, the response to the buzzer usually consisted of a slight head jerk followed by freezing, with the latter component predominant and sometimes lasting for extended periods of time. It is quite feasible that freezing, which may have been accompanied by internal emotional reactions, competed with head-shaking. It is also possible that the reaction to the buzzer constituted a defensive reaction (Sokolov, 1960), which would have reduced responsiveness to external stimulation, including the air stimulus. Whatever its underlying nature, the response to the buzzer showed

habituation effects across trials and across sessions. This was reflected in an across-trial increase in amount of dishabituation on the first buzzer session and in a trend towards control levels across buzzer sessions for frequency and latency measures.

Superimposed upon this background of suppressed responding during dishabituation sessions was a temporary sensitizing, or facilitating, effect of the buzzer. The temporary nature of this process was suggested in the between-trial experiment by the fact that only when the buzzer immediately preceded a trial was there a relative increase in frequency of responding (especially during the first 2-3 sec. of the trial), a decrease in the latency of the first response, and a retardation of within-trial habituation. A similar failure to obtain facilitation when the effects of the novel stimulus are allowed to wear off has been reported by Sharpless and Jasper (1956) and Wolda (1961). The course of recovery and subsequent re-habituation in the within-trial experiment revealed that the immediate sensitizing effects of the buzzer lasted only 2-3 sec. However, evidence for a longer-term facilitation was provided in both experiments by the retardation of across-trial frequency decrement.

Following the brief period of buzzer-produced excitation was a 10-15 sec. period of response suppression,

after which responding gradually returned to control levels. This post-excitatory suppression occurred for all three buzzer positions in the within-trial experiment, and for all but the most remote buzzer position in the between-trial experiment. The phenomenon habituated across sessions so that by the third dishabituation session no significant post-excitatory suppression occurred. Possible factors accounting for this suppression phenomenon have been discussed earlier, with emphasis placed on possible competing responses produced by the buzzer.

The effects of buzzer stimulation on the headshake response can thus be characterized as biphasic. The first phase is a short-lived period of sensitization, or excitation, which is reflected primarily as an increase in frequency of responding. When presented within trials, the buzzer produces complete recovery in response level (except for the first buzzer session). The excitatory period lasts no longer than 2-3 sec. The second phase is a period of response suppression lasting 10-15 sec., following which responding gradually returns to control level. While brief sensitizing effects of a dishabituatory stimulus have been reported before (e.g., Thompson and Spencer, 1966; Wolda, 1961; Sharpless and Jasper, 1956), the post-excitatory response suppression observed in the present study is a

novel finding. The existing literature on dishabituation contains no report of a biphasic response pattern to the novel stimulus. Buno et al. (1966) found that, prior to habituation, a novel stimulus reduced (inhibited) evoked potentials in response to an acoustic stimulus. However, this apparent response inhibition was not accompanied by any facilitating effect; dishabituation occurred only after substantial habituation had taken place and was not apparently accompanied by any suppressive effect. It must be pointed out that most studies of dishabituation have not been designed to reveal fine-grain features, such as a post-excitatory period of suppressed responding. It is therefore possible that complex reactions to a novel stimulus, similar to that found in the present study, have occurred before but have simply gone undetected. Nevertheless, at the present time the biphasic response to the dishabituatory stimulus found in the present study cannot be considered to apply with any generality. What can be said is that response patterns to dishabituatory stimuli may well be more complex than is typically thought. Accordingly, studies of dishabituation might profitably be designed to evaluate this possibility.

Although short-term sensitizing effects of the buzzer were demonstrated, the buzzer produced no longer-term removal of accumulated inhibition, as has been

assumed in the classic view of dishabituation (Humphrey, 1933). Rather, the present findings seem to agree more with Thompson and Spencer's (1966) characterization of dishabituation as an independent process of sensitization.

Neither experiment provided any evidence for habituation of dishabituation (sensitization). In fact, dishabituation showed an increase, rather than a decrease, during the first dishabituation session in the within-trial experiment, with the buzzer failing to produce dishabituation altogether on the first few presentations. This initial failure to produce dishabituation was likely due to the elicitation of responses which strongly competed with head-shaking. This account might help explain previous failures to obtain dishabituation (e.g., Wickens et al., 1966; Fried et al., 1966) when the novel stimulus was presented only once or twice. Dishabituation also exhibited an increase across sessions in the within-trial experiment. The results thus clearly show that the sensitizing influence of the buzzer was enhanced by repeating presentations, presumably as the tendency to elicit competing responses habituated. This is a completely novel finding and is contrary to the bulk of the literature (cf. Thompson and Spencer, 1966). It is, of course, possible that extended buzzer sessions would

have eventually led to a waning of the buzzer's sensitizing effects.

While in the classic sense no habituation of dishabituation occurred, it should be pointed out that the second major component of the behavioral pattern produced by the buzzer did show habituation effects. That is, the post-excitatory response suppression decreased from session to session and had largely disappeared by the third dishabituation session. Thus the entire buzzer-produced response pattern underwent changes in topography with repeated elicitation, with the exictatory component increasing in magnitude while the post-excitatory component waned and disappeared. The characteristic of a change in response topography with repeated trials is, according to Ratner (1967), one of the identifying characteristics of habituation.

Results of the within-trial experiment showed no effect of varying the buzzer position on the amount of dishabituation produced. However, control results revealed no significant frequency differences at the various points of buzzer presentation, indicating that the positions chosen did not effectively manipulate response strength. Therefore, no statement concerning the relation between strength of the habituating response and dishabituation can be made.

Although trials occurred at regular intervals, and although visual and auditory components accompanied the air stimulus in a relatively constant manner, no conditioning effects, either classical or temporal, occurred. This agrees with earlier findings by Leibrecht (1967). The lack of temporal conditioning may have been due to the fact that the inter-trial interval was 30 sec., which is not in the optimal range for temporal conditioning (Ratner and Denny, 1970). Classical conditioning may have failed to occur because the visual and auditory components did not constitute a sufficiently salient CS. It is also possible that the head-shake response is relatively impervious to conditioning attempts.

Conclusions

Application of the extraneous stimulus in the present study produced a 2-3 sec. period of sensitization, or excitation, followed by 10-15 sec. period of response suppression. This original finding of dual effects suggests that dishabituation is more complex than is generally acknowledged. It may also help explain conflicting results of studies which have dealt with dishabituation. Dishabituation in the present instance must be characterized as a superimposed process of sensitization, very brief in duration. Although concurrent stimulation appeared to retard across-trial habituation, long-term dishabituatory effects were

largely obscured by the buzzer-produced post-excitatory effects. There was no evidence for habituation of dishabituation. That is, the excitatory effect increased across trials and across sessions, although the post-excitatory suppression effect habituated across sessions.

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