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THE EFFECTS OF VARYING THE TRACE INTERVAL, CS
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THE EFFECTS OF VARYING THE TRACE INTERVAL, CS DURATION,
AND INTERREINFORCEMENT INTERVAL ON
KEY PECKING IN THE PIGEON

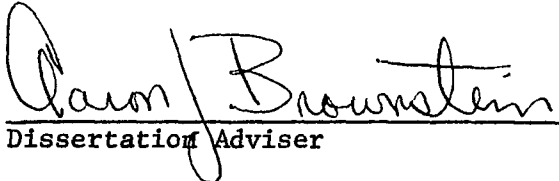
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Peter D. Balsam

A Dissertation Submitted to
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in Partial Fulfillment
of the Requirements for the Degree
Doctor of Philosophy

Greensboro
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Approved by


Dissertation Adviser

APPROVAL PAGE

This dissertation has been approved by the following committee of the Faculty of the Graduate School at the University of North Carolina at Greensboro.

Dissertation Adviser

Carol Brownstein

Committee Members

P. Scott Lawrence
Kendal Smith
William A. Power
R. L. Staley

6-30-75

Date of Acceptance by Committee

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When the illumination of a response key is followed by grain presentation, pigeons come to peck at the lighted key. Stimulus-reinforcer relationships in this procedure have been shown to exert a strong influence on the development and maintenance of responding. The control exerted by stimulus-reinforcer relationships was investigated by exposing groups of pigeons to procedures that differed according to the duration of the various intervals defined by the stimulus changes in this procedure. In the first phase of the experiment, variations in the time from keylight offset to grain onset produced an inverse relationship between several measures of the tendency to respond and the duration of the trace interval. The tendency to respond decreased as the duration of the key illumination was increased and the tendency to respond decreased as the interreinforcement interval was shortened. The effects of these three manipulations were summarized by an inverse relationship between the tendency to respond and a variable λ . This variable is formed by dividing the duration of the interstimulus interval by the duration of the interreinforcement interval and multiplying this quantity by the quotient produced by dividing the duration of the interstimulus interval by the CS duration. The within-CS response patterns indicated that subjects tended to respond soon after CS onset or not at all. Those subjects that did not respond much during the first phase of the experiment were exposed to a second procedure. The results of the second phase replicated the findings of the earlier portion of the experiment and, additionally, demonstrated that the transfer from Phase I to Phase II was related to the Phase I λ . Predictions based on

recently proposed contingency models of conditioning were not entirely consistent with the results of both phases of the experiment. A model based solely on temporal parameters was developed and the predictions based on this model were shown to be in accord with the results of the experiment.

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INTRODUCTION

Brown and Jenkins (1968) found that if grain is presented according to a Pavlovian delay procedure, that is, following the brief illumination of a response key, pigeons come to peck the illuminated disk. Key pecking is then maintained at substantial levels under conditions in which pecks produce grain immediately (Brown & Jenkins, 1968), cancel grain for that trial (Williams & Williams, 1969) or have no effect on grain presentation (Brown & Jenkins, 1968). These and other studies have indicated that the relationship between the occurrence of the conditional stimulus (key illumination) and the occurrence of the unconditional stimulus (grain presentation) is an important determinant of key pecking (Gamzu & Williams, 1971, 1972; Gamzu & Schwartz, 1973; Wasserman, Franklin, & Hearst, 1974).

One way to vary the relationship between the CS and the US is to manipulate the duration of the various intervals bounded by the stimulus changes in the conditioning situation. Variations in the temporal relationship between the CS and the US have been shown to have large effects on the acquisition and maintenance of key pecking in the pigeon.

Ricci (1973) compared the behavior controlled by a 30-sec CS with that of a 120-sec CS. A 240-sec Intertrial Interval (ITI) was employed in both procedures. He found no reliable difference between the groups in the number of trials to the first peck. Subjects did, however, make their fifth and tenth pecks significantly earlier in the 30-sec group. Newlin and LoLordo (1973) found that the median number of trials to

reach a criterion of four out of five consecutive trials with a response was 58 for a 4-sec delay group and 78 for an 8-sec group. Trials were presented on a variable time 30-sec (VT 30-sec) schedule.

In a study by Griffin (1975) a 6-sec CS was presented to different subjects after ITIs ranging from 15 to 960 seconds. The mean number of trials to the first peck was a u-shaped function of the ITI. Subjects responded after fewer trials at intermediate values of the ITI than they did at the extreme values. The median number of trials to the first peck, however, was a negatively decelerated function of the ITI. Terrace, Gibbon, Farrell, and Baldock (1975) varied the ITI from 5 to 400 seconds and also found that the median number of trials that subjects took before the first peck and to reach a criterion of pecks on three of four consecutive trials, was a negatively decelerated function of the ITI.

More extensive variation of stimulus duration has been investigated by Groves (1973) and by Baldock (1975). Groves (1973) varied the CS duration from 6 to 96 seconds while varying the inter-reinforcement interval (IRI) from 30 to 120 seconds. The ratio of trial duration to IRI varied from .2 to 1.0. The effect of these manipulations was best summarized as a direct relationship between ratio size and the mean and median number of trials to the first peck. Baldock (1974) exposed subjects to CS durations ranging from 1 to 32 seconds and to ITI values ranging from 6 to 768 seconds. The ratio of CS to ITI duration ranged from 1.5 to 0.01. The mean and median number of trials to reach the previously mentioned Terrace et al. criterion was described by a power function of the decreasing ratio with a negative exponent.

The previously cited studies that found effects of singly varying either the CS duration or ITI values may be considered to be special cases of a more general relationship. In most instances, acquisition of key pecking is adequately described as a negatively decelerated function of the ratio of CS duration to ITI or of the ratio of CS duration to IRI.

The effects of varying the temporal relationship between the CS and US on "steady state" performance are less consistently documented than the acquisition effects are. Terrace et al. (1975) found that after 275 trials, the mean terminal running rate was a power function of the ITI. The ITI effect was statistically significant, although there was considerable overlap between groups and the rates of responding in the groups exposed to the longer ITIs were declining toward the end of training. There was no significant difference between subjects exposed to different ITI values in respect to the proportion of trials with at least one response. Griffin (1975) exposed subjects to various ITIs for 600 trials and found that during the last 150 trials both the mean and median number of trial responses and trials with at least one response were inverted u-shaped functions of ITI duration. Baldock (1974) exposed subjects to various combinations of CS durations and ITIs for 15 days after reaching the Terrace et al. criterion. Terminal overall rates of responding were therefore computed after different numbers of trials for the different groups. The median number of trials ranged from about 280 to 525 for the different groups. These data showed no consistent relationship between the ratio of CS duration to ITI and response rates. For a given CS duration, the function relating

ITI values to response rates was inconsistent in form. For three of the four CS durations for which there was enough data, however, response rates were highest for subjects exposed to intermediate ITI values. There was also a slight trend in the data suggesting that response rates might decrease as the CS duration increases.

The major temporal parameter that seems to affect "steady state" performance, therefore, appears to be the absolute size of the ITI. Consistent with this effect of absolute ITI duration, Groves (1973) found that for a given ratio of CS duration to IRI response rates decreased as the absolute size of the IRI increased. The use of a negative response contingency in this study does not permit direct comparison with the previously mentioned studies. It should be noted, however, that Griffin (1975) found no difference in the shape of the functions relating ITI to response rate between subjects exposed to a delay procedure and those exposed to an identical procedure except for pecks cancelling grain for the trials on which they occurred.

In summary, it might tentatively be suggested that acquisition of key pecking is a function of the ratio of CS duration to ITI or IRI, and that terminal responding is additionally determined by the absolute stimulus durations.

This description of effects, however, is limited to a delay procedure. If the CS duration and IRI are held constant but a trace procedure is employed, there appear to be additional effects beyond those ascribable to the durations of the CS and IRI.

Newlin and LoLordo (1973) presented trials on a VT 30-sec schedule. In a trace procedure each trial consisted of a 4-sec CS presentation

followed by grain 4 seconds after CS offset. Subjects took a median of 269 trials to reach a criterion of at least one peck on five consecutive trials. This was more than three times as many trials as subjects exposed to an 8-sec delay procedure and over four times as many trials as subjects exposed to a 4-sec delay procedure took to reach the same criterion. There was no consistent difference between groups in response rates after 1500 trials.

Hemmeldinger (1974) varied the duration of the trace interval (TI) from 0 to 32 seconds. He employed a CS of 4 seconds duration and US presentations were programmed on an FT 4-min schedule. One group of subjects exposed to trace intervals of 0, 4, 8, and 16 seconds for at least 450 trials at each value, exhibited average response rates of 90, 40, 38, and 28 responses/minute, respectively. Three additional groups of subjects exposed to a 16-sec trace interval pecked at rates of about 28 to 40 responses/minute. A fifth group of subjects exposed to a 32-sec trace interval pecked at an average rate of about two or three responses/minute during the final sessions of exposure to this condition. In a second experiment, the same inverse relationship between trace interval length and response rates was obtained when subjects were exposed to each of the five different trace intervals twelve times within each session. The procedure employed in Hemmeldinger's experiments confounds increases in the trace interval with increases in the inter-stimulus interval (ISI), that is, the interval from CS onset to US onset. Thus the effects may be due to either factor or a combination of the two.

Wasserman (1974) also simultaneously varied the trace interval and ISI. In this experiment, the illumination of two response keys on opposite sides of the grain magazine were used as CSs. One of the two keys was illuminated in a random sequence every 100 seconds and grain was presented at different times following CS offset for different groups of subjects. The rates of responding, computed for the last 240 of the experiment's 840 trials, were inversely related to the mean trace interval for each of the groups. Trace intervals ranging from 10 to 19 seconds controlled only very low average rates of responding and trace intervals greater than 19 seconds engendered almost no responding. In addition, those subjects exposed to trace intervals greater than 19 seconds tended to withdraw from the side of the experimental chamber in which the CS was presented.

In summary, the temporal relationship between CS and US is an important determinant of key pecking in the pigeon. Variations in the IRI or ITI and variations in the ISI in both delay and trace procedures exert strong influences on the acquisition and maintained levels of responding. There also appears to be a TI effect even when the ISI is held constant.

The purpose of the experiment reported here is to further explore the effects of varying the temporal relationship between CS and US by assessing the effects of different trace intervals at various ISIs and IRIs on the acquisition and maintenance of key pecking in different groups of pigeons.

METHOD

Subjects

Eighty experimentally naive white Carneaux hens, 6 to 9 months old at the start of the experiment, served as experimental subjects. Birds were maintained at 80% of their free-feeding body weight throughout the course of the study.

Apparatus

The apparatus consisted of two standard pigeon chambers painted flat black measuring 30 cm X 36 cm X 45 cm.

In one of the boxes, two response keys were located 35 cm above the floor and 14 cm from the respective sides. The key on the right side of the response panel remained covered by tape at all times. In this chamber, general illumination was provided by three unshielded GE #1829 bulbs. Two of these bulbs were located in the upper right corner of the response panel and the third bulb was located in the center of the chamber ceiling.

The second chamber contained three response keys located 35 cm above the floor. The center key, located 18 cm from the edges of the response panel, was employed as the manipulandum. The two side keys were covered with tape during the experiment. General illumination in this chamber was provided for by 12 Sylvania 28PSB bulbs mounted behind a translucent plate above the center key.

In both boxes, the house lights remained on at all times during the session except during feeder operation. The functional response

keys were illuminated by two GE #1829 bulbs in series with 65 Ω of fixed resistance and a 28-volt power source. White noise remained on at all times in order to mask extraneous sounds. Standard electro-mechanical programming equipment was used to control the experiment and record data.

Procedure

All subjects were trained to eat from the hopper in the following manner. The hopper remained raised until each bird had eaten for 15 seconds. The hopper was then lowered and subsequent hopper presentations occurred on a VT 15-sec schedule. The hopper duration was set at 15 seconds until each subject ate twice when the hopper was raised. The feeder duration was then reduced to 8 seconds until each bird ate twice and was then further reduced to 4.5 seconds for all subsequent presentations. Each hopper-training session lasted for 125 brief feeder presentations or until a subject reached a criterion of inserting its head in the feeder aperture on 15 consecutive presentations. Sixty-seven of the subjects reached this criterion within the first session. Ten subjects achieved the criterion performance in the second session, and one subject did so in the third session. The average number of presentations before reaching criterion across these subjects was 37.18. The remaining two subjects never hopper-trained and were dropped from the study.

Subjects were then randomly assigned to one of the 20 experimental groups. The experimental groups differed according to the durations of the various stimuli that were presented during each session. The specific values that each of the subjects in each group experienced are presented in the left-hand column of Table 1. The groups are identified by three hyphenated numbers. The first number represents the duration

TABLE 1
 EXPERIMENTAL CONDITIONS AND NUMBER OF TRIALS OF EXPOSURE
 TO EACH CONDITION FOR INDIVIDUAL SUBJECTS

Phase I Condition	Subject	No. of Phase I Trials	Phase II Condition	No. of Phase II Trials
16-8-8	111	450	80-12-4	675
	112	450	80-4-12	700
	113	450	80-12-4	625
	114	450	80-4-12	725
16-4-28	011	450	80-12-4	575
	012	450	80-4-12	400
	013	450	80-12-4	450
	014	450	80-4-12	450
92-4-0	021	435		
	022	410		
	023	400		
	024	400		
88-8-0	031	435		
	032	410		
	033	425		
	034	400		
88-4-4	041	435		
	042	410		
	043	425		
	044	425		
80-16-0	051	410		
	052	460		
	053	400		
	054	425		
80-12-4	061	450		
	062	575		
	063	650		
	064	525		
80-8-8	072	635		
	073	750		
	074	525		
80-4-12	081	450		
	082	585		
	083	450		
	084	450		

TABLE 1 (continued)

Phase I Condition	Subject	No. of Phase I Trials	Phase II Condition	No. of Phase II Trials
64-16-16	121	450	80-12-4	575
	122	450	80-4-12	825
	123	450	80-12-4	725
	124	450	80-4-12	550
48-16-32	131	450	80-12-4	450
	132	450	80-4-12	450
	133	450	80-12-4	450
	134	450	80-4-12	450
48-8-40	141	450	80-12-4	450
	142	450	80-4-12	450
	143	450	80-12-4	450
	144	450	80-4-12	450
48-4-44	151	450	80-12-4	450
	152	450	80-4-12	450
	153	450	80-12-4	450
	154	450	80-4-12	450
32-48-16	161	450		
	162	450		
	163	450		
	164	450		
32-32-32	171	410		
	172	485		
	173	450	80-4-12	450
	174	450	80-4-12	450
32-8-56	181	450	80-12-4	575
	183	450	80-12-4	675
	184	450	80-4-12	450
240-12-4	091	435		
	092	485		
	093	425		
	094	400		
240-8-8	101	435		
	102	435		
	103	425		
	104	425		
240-4-12	191	450		
	192	400		
	193	500		
	194	450		
224-4-28	201	450		
	202	500		
	203	450	80-12-4	475
	204	450	80-4-12	450

of the ITI, the second represents the duration of the CS and the third represents the duration of the TI, all in seconds. It should be noted that a variety of TIs, CS durations, ISIs and IRIs were employed in the present study. These values were chosen to permit the assessment of the effects of varying a particular parameter while the others were held constant, as well as to provide a large sample of parameter values. In all conditions, the key was dark except during CS presentations. The first two experimental sessions for half the subjects terminated after 30 grain presentations. All subsequent sessions for these subjects and all sessions for the remaining subjects terminated after 25 grain presentations.

In Phase I, subjects were exposed to the parameter values shown in Table 1 until one of two conditions was met. If subjects pecked on three of four consecutive trials within the first 450 CS presentations, they were run for an additional 15 sessions. Subjects that did not meet the criterion within 450 trials were either terminated or exposed to a second phase of the experiment. The number of trials that each subject received during the first phase of the experiment is shown in the third column of Table 1.

Those subjects that did not reach criterion and had pecked on fewer than 15 of the 450 trials were divided into two groups. One group was exposed to an 80-4-12 procedure. The remaining subjects were exposed to an 80-12-4 procedure. Subjects were assigned to Phase II groups in such a way that the different groups of Phase I were represented equally often in the groups of Phase II whenever that was appropriate and possible. The fourth column of Table 1 shows which subjects were shifted and

to which experimental treatment they were exposed during the second phase of the experiment. The fifth column of Table 1 shows the number of trials to which each subject was exposed during the second phase of the experiment.

RESULTS

The results are presented separately for the two phases of the experiment. Summary descriptions of pecking during CS presentations in early parts of Phase I are presented first. This is followed by descriptions of pecking during CS presentation across sessions in the latter part of Phase I. The within-CS patterns of responding are described next and are followed by descriptions of TI and ITI responding late in training. Phase II results are presented in an analogous fashion and are also contrasted with comparable Phase I results.

Phase I

Initial performance across CS presentations. The first response occurred on early trials for many of the subjects regardless of what treatment group they were in. The first column of Table 2 lists the trial number on which the first peck occurred for all subjects. Birds that never responded are denoted by an asterisk (*). Twelve of the subjects responded on the very first CS presentation, prior to any CS/US pairings. Thirty-six of the subjects responded within the first 10 trials. The median number of trials to the first response for all subjects was 12. Since so many subjects responded so early, the trial of the first peck did not vary systematically as a function of the temporal parameters to which a subject was exposed.

The second column of Table 2 shows the number of the trial on which the fifth response occurred for each subject. This measure seems to effectively differentiate the experimental groups in a way consistent

TABLE 2
MEAN INITIAL PERFORMANCE STATISTICS FOR PHASE I

Phase I Condition	Subject	Trial of First Response	Trial of Fifth Response	\bar{X} Latency on 1st 5 Trials w/Responses
16-8-8	111	334	*	1.0
	112	12	*	2.0
	113	*	*	*
	114	4	*	3.0
	Mean	200	450	2.0
	Median	223	450	2.0
16-4-28	011	*	*	*
	012	11	*	2.0
	013	*	*	*
	014	*	*	*
	Mean	340.3	450	2.0
	Median	450	450	2.0
92-4-0	021	22	56	2.6
	022	4	19	1.0
	023	14	25	2.2
	024	5	18	1.6
	Mean	11.3	29.5	1.9
	Median	9.5	22	1.9
88-8-0	031	17	29	2.0
	032	10	26	1.4
	033	3	49	3.2
	034	1	19	1.6
	Mean	7.8	30.8	2.1
	Median	6.5	27.5	1.8
88-4-4	041	40	60	1.6
	042	3	42	1.0
	043	1	50	1.4
	044	10	61	2.0
	Mean	13.5	53.3	1.5
	Median	6.5	55	1.5

TABLE 2 (continued)

Phase I Condition	Subject	Trial of First Response	Trial of Fifth Response	\bar{X} Latency on 1st 5 Trials w/Responses
80-16-0	051	12	33	7.0
	052	3	45	5.0
	053	15	25	5.6
	054	9	40	4.4
	Mean	9.8	35.8	5.5
	Median	10.5	36.5	5.3
80-12-4	061	324	444	2.8
	062	95	175	4.4
	063	4	244	3.2
	064	2	115	4.8
	Mean	106.3	244.5	3.8
	Median	49.5	209.5	3.8
80-8-8	072	100	229	1.2
	073	364	399	1.6
	074	91	129	2.6
	Mean	185	252.3	1.8
	Median	100	229	1.6
	80-4-12	081	166	380
082		9	168	2.0
083		*	*	*
084		3	439	1.4
Mean		157	359.3	1.5
Median		87.5	409.5	1.4
64-16-16	121	*	*	*
	122	1	*	11
	123	185	*	14
	124	52	*	5.25
	Mean	172	450	10.1
	Median	118.5	450	11.0

TABLE 2 (continued)

Phase I Condition	Subject	Trial of First Response	Trial of Fifth Response	\bar{X} Latency on 1st 5 Trials w/Responses
48-16-32	131	*	*	*
	132	396	*	1.0
	133	7	*	1.5
	134	61	197	7.8
	Mean	228	386.8	3.4
	Median	228.5	450	1.5
48-8-40	141	*	*	*
	142	1	*	0
	143	2	*	2.0
	144	8	*	2.0
	Mean	115.3	450	2.0
	Median	5	450	2.0
48-4-44	151	*	*	*
	152	*	*	*
	153	17	*	3.0
	154	146	*	1.67
	Mean	265.8	450	2.8
	Median	298	450	2.8
32-48-16	161	5	340	28.2
	162	2	302	0.8
	163	231	337	8.2
	164	49	410	13.6
	Mean	75.8	327.3	12.7
	Median	27	338.5	10.9
32-32-32	171	1	6	7.8
	172	1	57	4.6
	173	421	*	13.0
	174	1	437	17.0
	Mean	106	237.5	10.6
	Median	1.0	247	10.4

TABLE 2 (continued)

Phase I Condition	Subject	Trial of First Response	Trial of Fifth Response	\bar{X} Latency on 1st 5 Trials w/Responses
32-8-56	181	376	*	6.0
	183	4	*	1.0
	184	1	*	3.0
	Mean	127	450	3.3
	Median	4	450	3.0
240-12-4	091	1	28	5.8
	092	1	88	3.0
	093	6	32	4.6
	094	1	9	4.2
	Mean	2.3	34.3	4.4
Median	1	30	4.4	
240-8-8	101	5	44	4.4
	102	6	52	3.6
	103	33	44	3.0
	104	6	41	2.8
	Mean	12.5	45.3	3.5
Median	6	44	3.3	
240-4-12	191	4	52	1.6
	192	11	19	2.8
	193	59	83	3.0
	194	1	58	2.0
	Mean	18.8	53	2.4
Median	7.5	55	2.4	
224-4-28	201	*	*	*
	202	77	114	2.2
	203	184	*	1.0
	204	236	404	2.0
	Mean	236.8	354.5	1.7
Median	210	427	2.1	

with many measures to be presented subsequently. Figures 1a and 1b show the median number of trials to the fifth response for all the experimental groups. Several trends in the medians of these data are worth mentioning. Comparison of the three delay conditioning groups with 0 trace intervals indicates that the trial of the fifth response is an increasing function of the CS duration. Secondly, Figure 1a shows that for a given ISI, with one exception, as the trace interval increases (CS duration decreases) the number of trials to the fifth response increases rapidly. The one exception to this rule takes place at the 64-sec ISI. Group 32-32-32 took fewer trials to reach this criterion than did either group 32-48-16 or group 32-8-56. Figure 1b shows that for a given CS duration the number of trials prior to the fifth trial with a response increases rapidly as TI duration increases. It should also be noted that for a given trace interval, the number of trials to reach this criterion is generally lower for subjects exposed to shorter CS durations. Lastly, it should be noted from both parts of Figure 1 that subjects exposed to the 256-sec IRI took fewer trials until they made their fifth response than did subjects exposed to shorter IRIs with comparable ISIs, CS durations, and trace intervals.

Because for a given ISI, increases in trace intervals necessarily require a reduction in the duration of the CS, subjects exposed to different trace intervals at the same ISI have less opportunity to respond. In other words, perhaps the data presented in Figure 1 are a direct function of the experimental manipulation and/or the opportunity a subject has to respond. Whether or not the opportunity to respond possibly needs to be taken into account can be decided by an examination

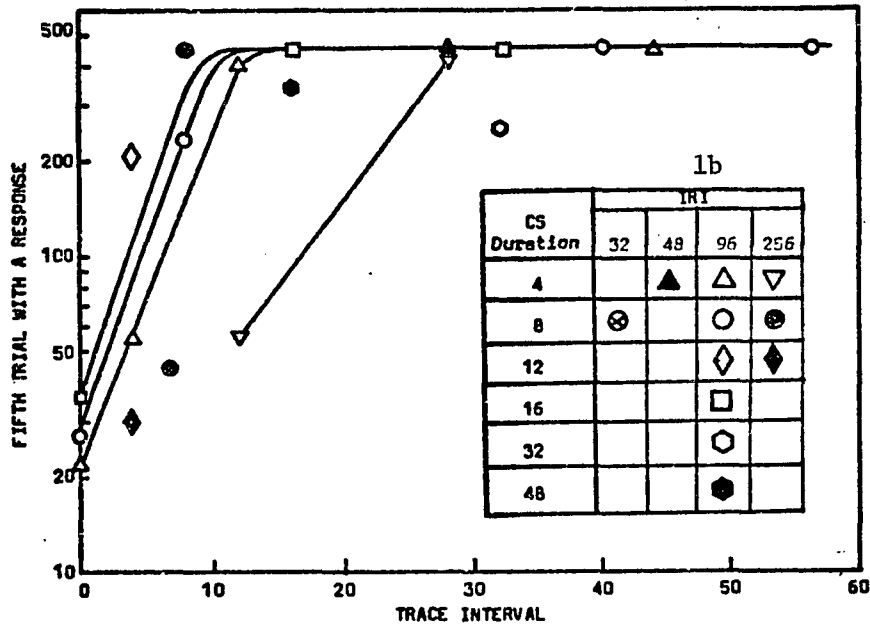
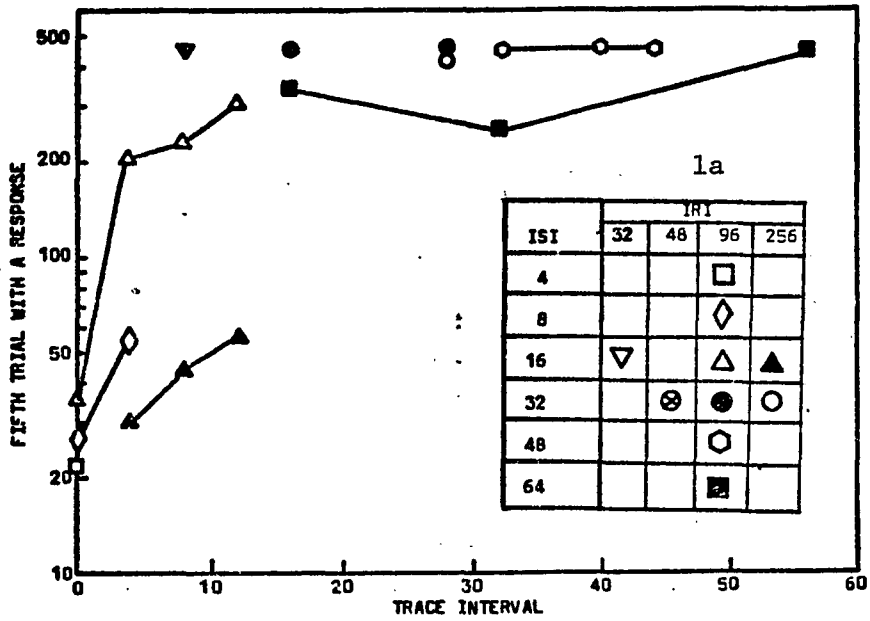


Figure 1. The median number of trials to the fifth trial with a response is shown as a function of trace interval. The upper portion of the figure shows this relation for different ISIs. The lower part of the figure shows this relation for different CS durations.

of the average latencies to the first response or the first five trials with a response. The mean latency for each subject is shown in column 3 of Table 2. The average latency for the trials in which they responded is shown for those subjects that never made responses on five trials; an asterisk indicates that a particular subject never made any responses. The relationship between the latency of these early responses and CS duration can be clearly seen in Figure 2. This figure shows that as the CS duration increases, regardless of any other parameter values, the median latencies to the first response tend to increase. This relationship may be a direct effect of CS duration or it may indicate that those subjects exposed to longer CS durations often pecked because of a greater opportunity to do so than those subjects exposed to shorter CS durations. If the latter is the case, then we might expect the latencies to get longer as the tendency to respond, as indicated by other measures such as the one depicted in Figure 1, decreases within each CS duration. The medians in Figure 2 show no such consistent ordering, suggesting that response strength coupled with increasing opportunities is not the sole determinant of the points in Figure 1. If the opportunity to respond needs to be taken into account, however, in determining the effects of the experimental manipulation, then perhaps the number of trials to the fifth response is not entirely appropriate. One measure that "weights" the number of trials by the CS duration is the number of trials prior to the fifth response with a latency less than the shortest CS duration. In this way, all experimental groups have the same statistical opportunity to respond. Figure 3 shows the median number of trials to the fifth response with a latency

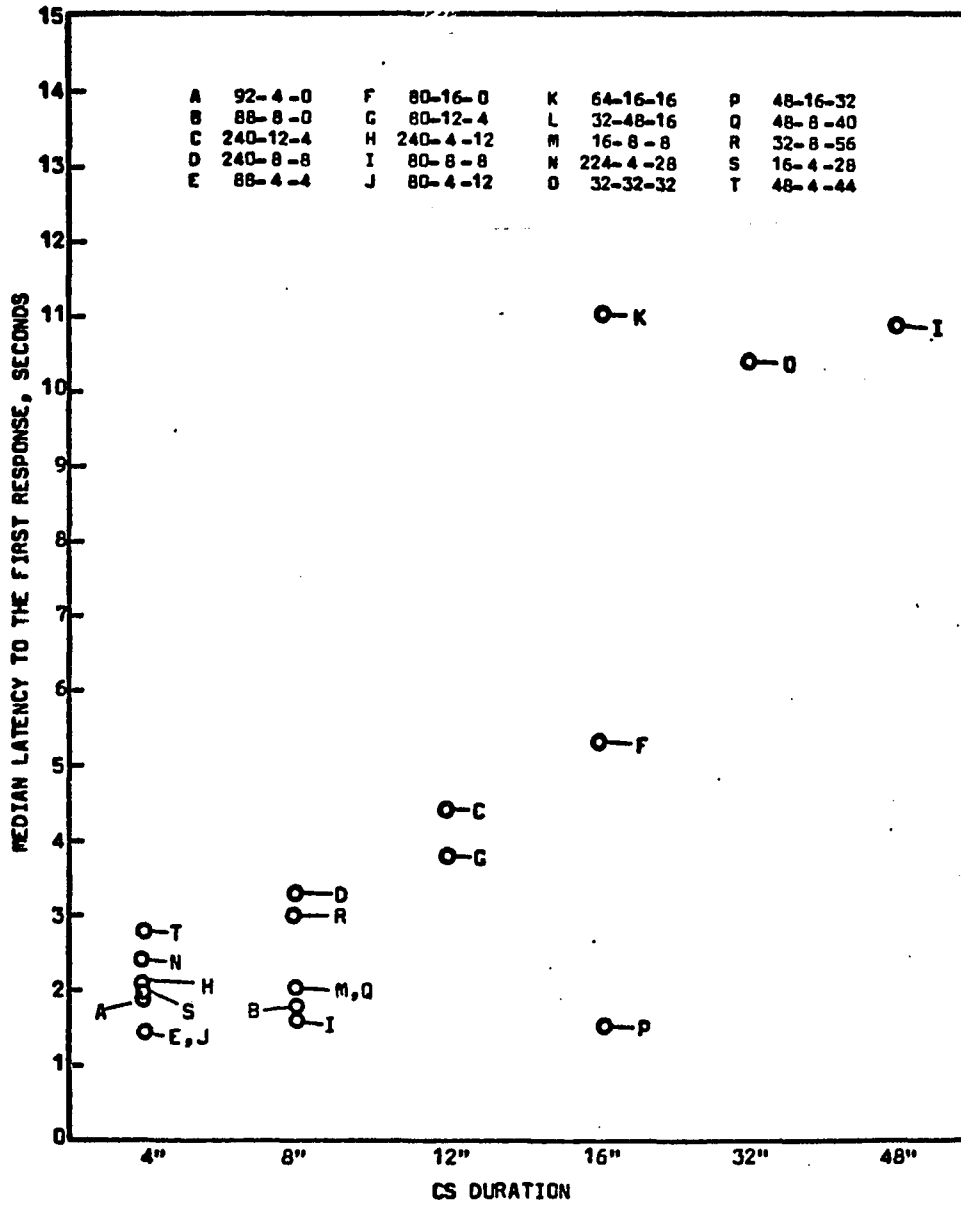


Figure 2. The median latency to the first response on the first five trials with a response as a function of CS duration is shown for the different experimental groups.

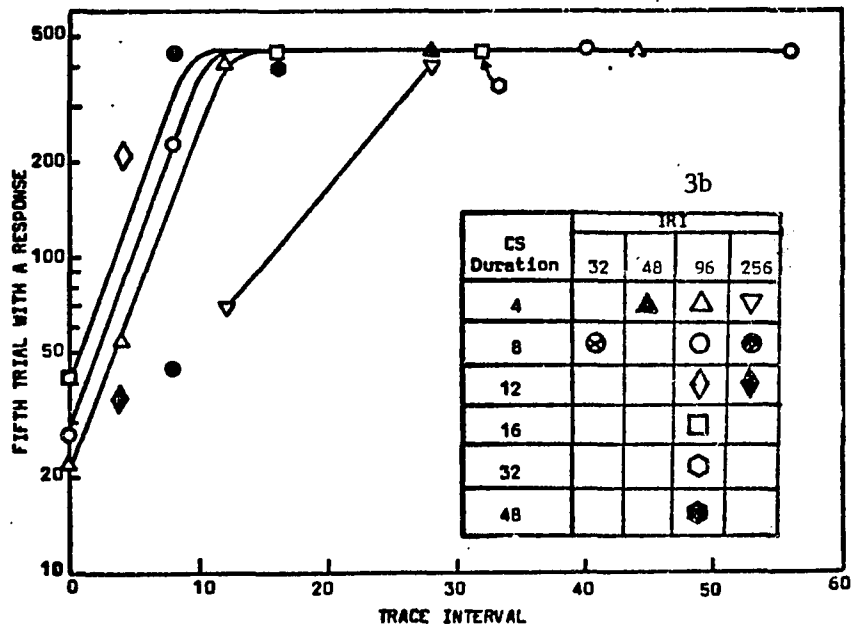
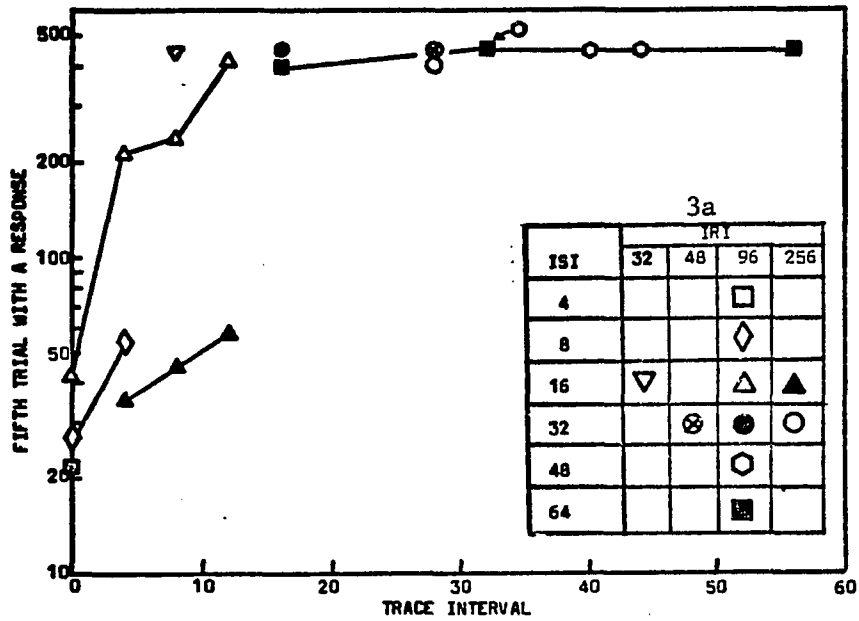


Figure 3. The median number of trials to the fifth trial with a response with a latency less than or equal to four seconds. The upper portion shows the relation between this measure and trace interval duration for different ISIs. The lower portion shows this relation for different CS durations.

less than 4 seconds for all the experimental groups. The pattern of results depicted in Figures 3a and 3b is very similar to the relationships that exist in Figure 1. The measure depicted in Figure 3 increases as CS duration increases in the three delay conditioning groups. In all instances shown in Figure 3a, for a given ISI in which subjects responded, the median number of trials to the fifth response with a latency less than 4 seconds increases rapidly as the TI increases. Figure 3b shows that for a given CS duration, the trial of the fifth peck increases rapidly as a function of the TI. Lastly, the data in Figure 3 show that subjects exposed to the 256-sec IRI took fewer trials to reach the fifth trial with a short latency response than did subjects exposed to comparable CS durations and trace intervals presented with shorter IRIs.

Three other acquisition measures were examined. They were the number of trials that the subjects in each experimental group took to reach a criterion of pecks on three of four consecutive trials, the number of trials until each subject reached the trial of the tenth peck, and the total number of trials with a response in the first 200 trials. All of these measures of the early tendency to respond yield a similar pattern of results to the acquisition measures presented earlier.

In summary, all of the acquisition statistics indicate the same general pattern of results: (a) For a given ISI or CS duration increasing the TI increases the number of trials to reach the acquisition criteria, (b) increasing the CS duration when the trace interval is equal to zero retards acquisition, and (c) increasing the IRI facilitates acquisition when all other parameters are held constant.

Figure 4 shows the development of key pecking during the first 15 sessions. The number of trials with at least one response is shown as a function of session number for individual subjects in all of the groups. Figure 4 shows that most subjects reached their asymptotic level of performance within three or four sessions. Reaching asymptote appears to be somewhat retarded, however, in subjects exposed to trace procedures with a 16-sec ISI and a 96-sec IRI (groups 80-4-12, 80-8-8, and 80-12-4) and in those subjects that responded which were exposed to the longer trace intervals at the 256-sec IRI (groups 240-4-12 and 224-4-28).

The terminal level of performance depicted in Figure 4 also appears to vary with the experimental condition. The differences can be more clearly seen in the group descriptions of terminal performance.

Terminal performance across CS presentations. Terminal performance measures were computed for each subject on data collected during each subject's last five sessions (125 trials) of exposure to an experimental treatment. These data were collected during trials 325 to 450 for those subjects that never responded on three of four consecutive trials and on the 250th to 375th trial following the day on which the remaining subjects met this criterion.

The median number of trials with a response during the last five days is shown as a function of experimental group in Figure 5 and the individual data on which these statistics are based appear in column 1 of Table 3. There appear to be only two relationships between these measures and the temporal parameters. First, for a given CS duration there is a decreasing tendency to respond with increasing trace intervals

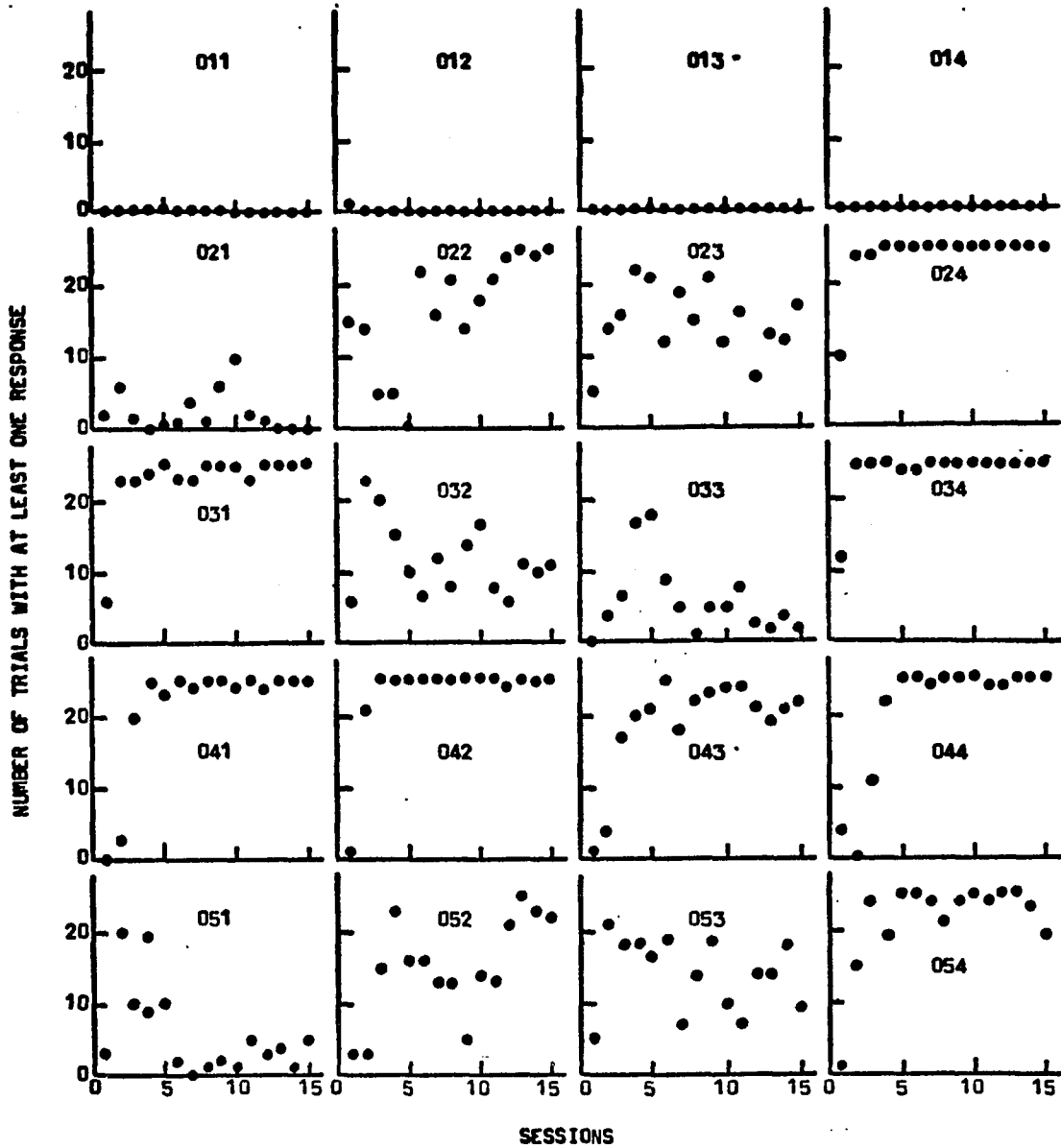


Figure 4. The number of trials with at least one response during the first 15 sessions of the experiment. Each row of figures depicts the performance of subjects exposed to different experimental treatments. The specific treatment of each group can be found in Table 1.

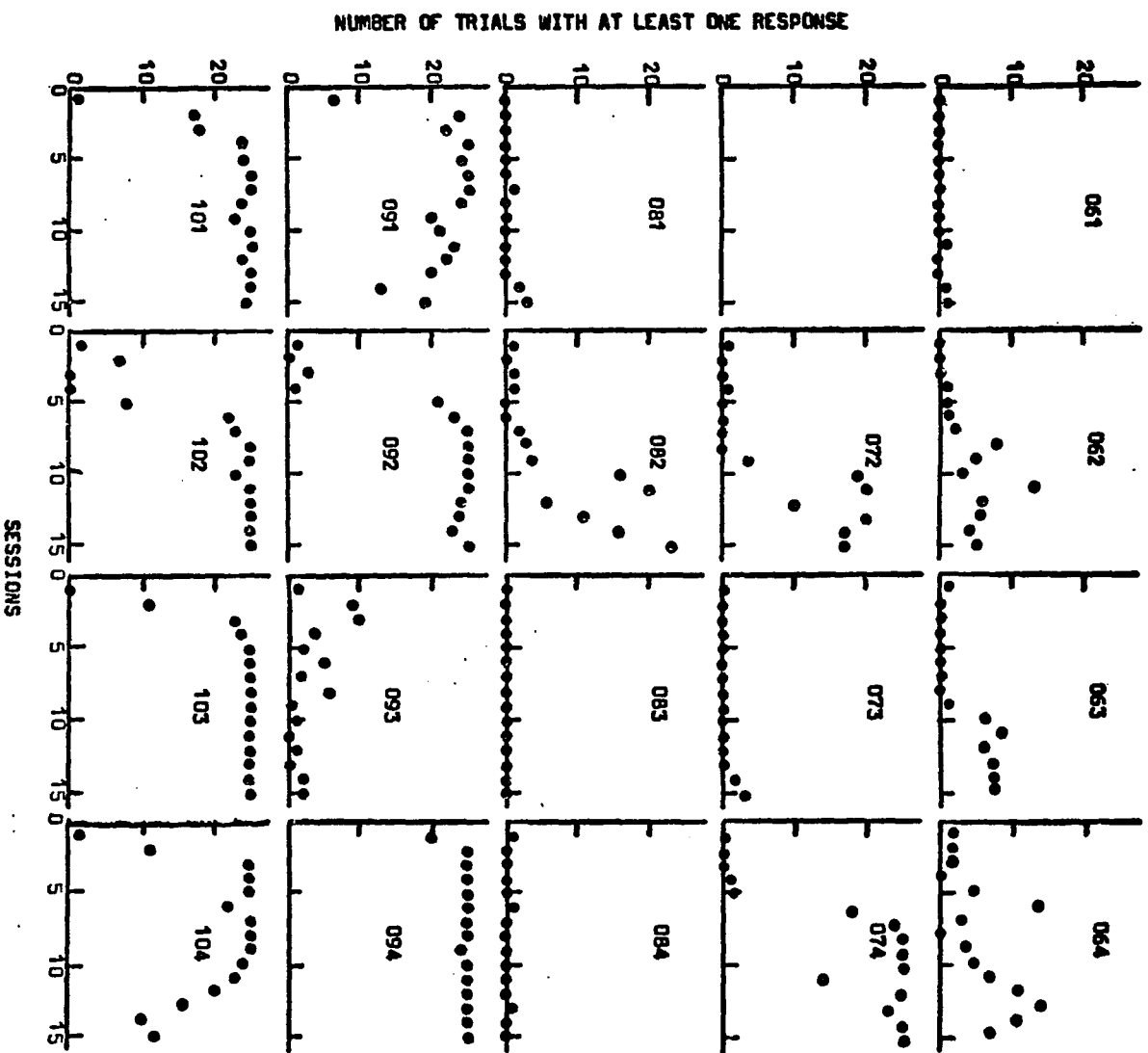


Figure 4. (continued)

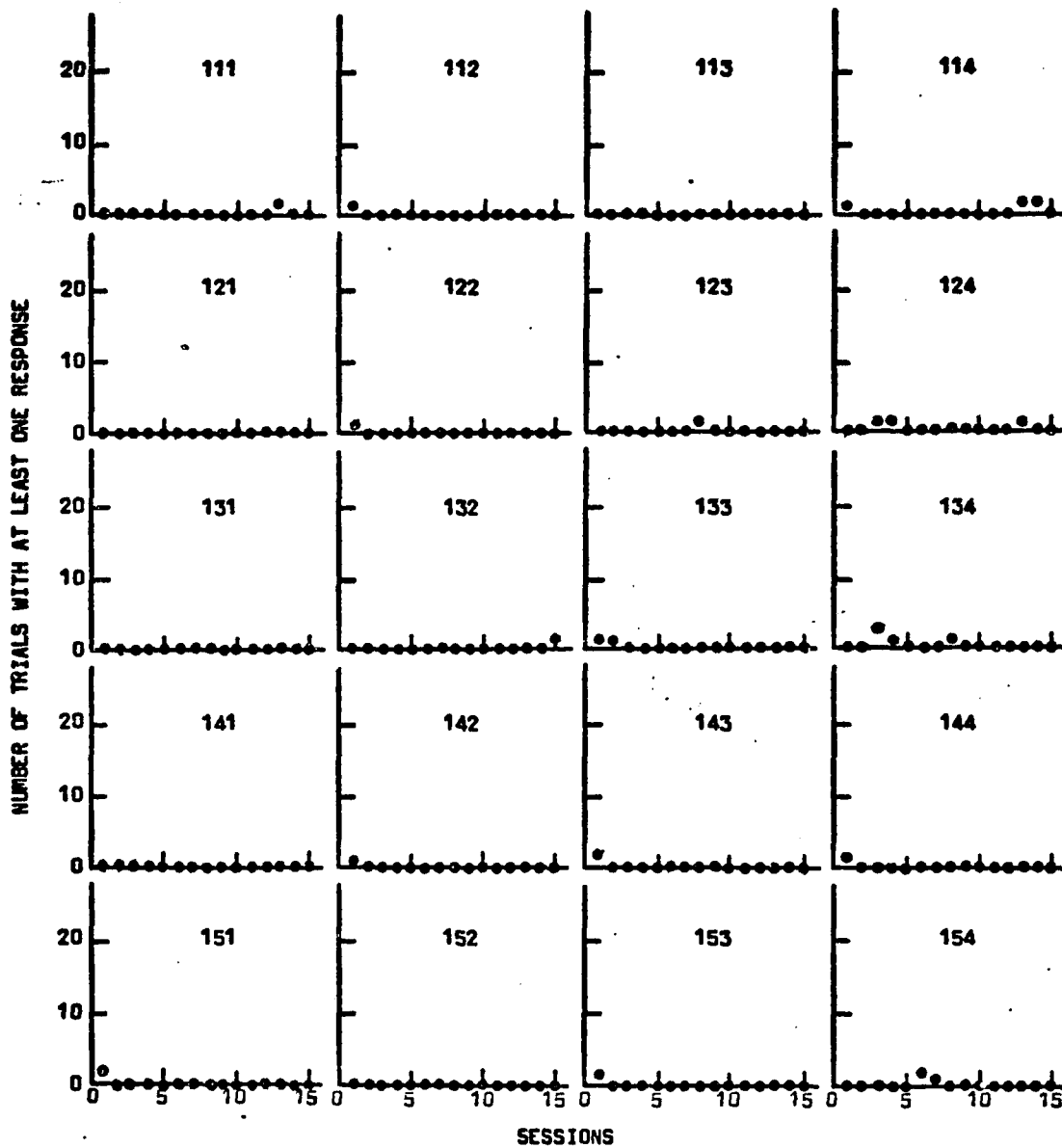


Figure 4. (continued)

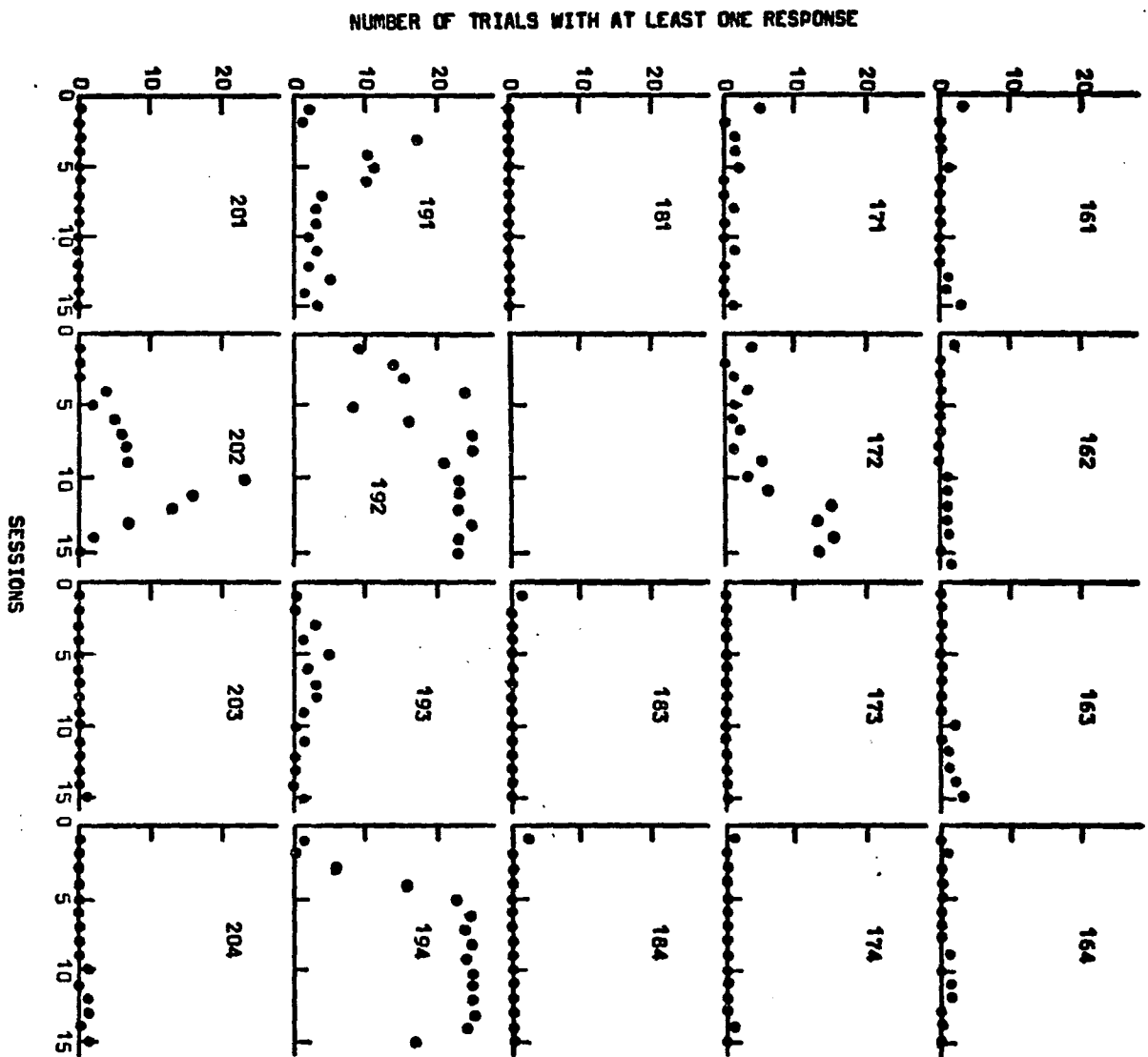


Figure 4. (continued)

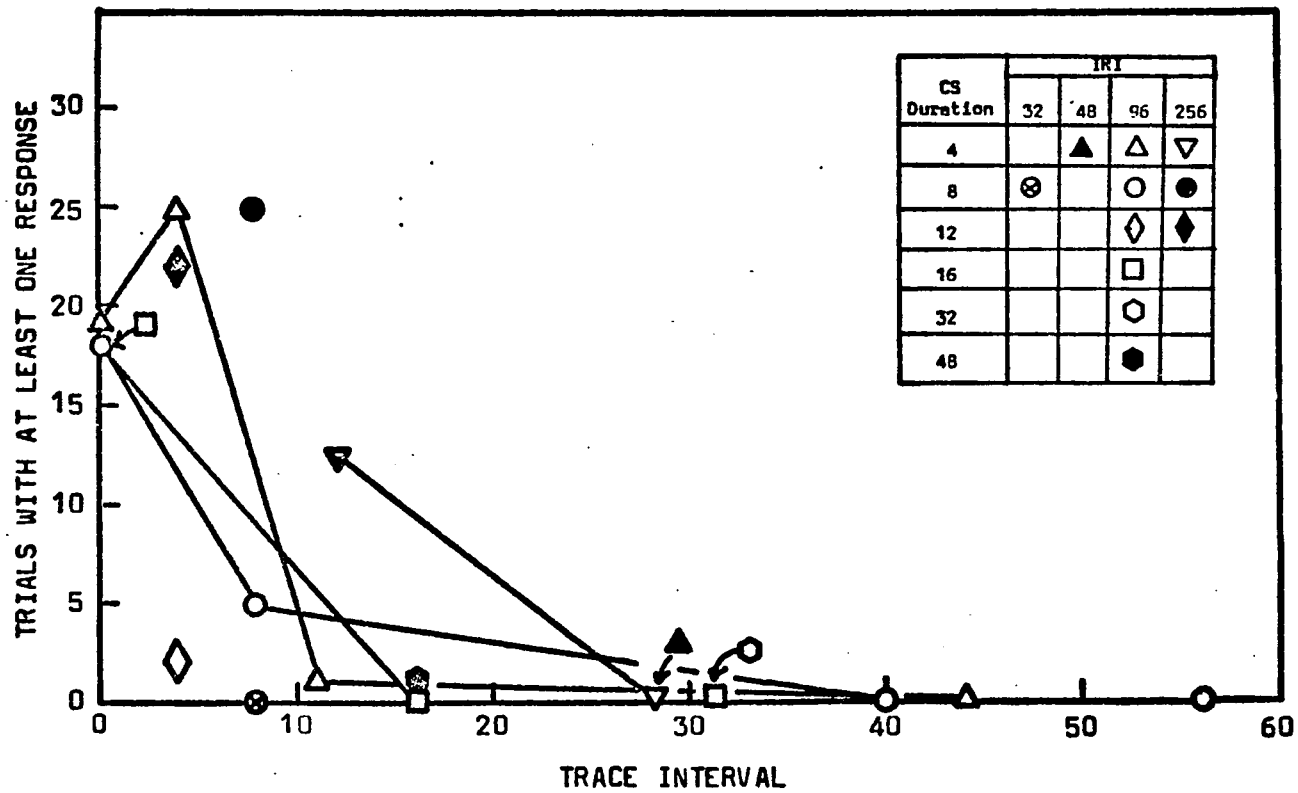


Figure 5. The median number of trials with at least one response during the last 125 trials of phase I is shown as a function of the trace interval for different CS durations.

TABLE 3

MEAN TERMINAL PERFORMANCE STATISTICS FOR PHASE I

Phase I Condition	Subject	\bar{X} No. Trials w/Response	\bar{X} Overall Response Rate	\bar{X} Latency to First Response	\bar{X} Running Rate	\bar{X} TI Response Rate	\bar{X} ITI Response Rate
16-8-8	111	0.0	0.0	*	0.0	0.0	0.0
	112	0.0	0.0	*	0.0	0.0	0.0
	113	0.0	0.0	*	0.0	0.0	0.0
	114	0.4	0.1	1.4	7.7	0.0	0.3
	Mean	0.1	0.0	1.4	1.9	0.0	0.1
	Median	0.0	0.0	1.4	0.0	0.0	0.0
16-4-28	011	0.0	0.0	*	0.0	0.0	0.0
	012	0.0	0.0	*	0.0	0.0	0.0
	013	0.0	0.0	*	0.0	0.0	0.0
	014	0.0	0.0	*	0.0	0.0	0.0
	Mean	0.0	0.0	*	0.0	0.0	0.0
	Median	0.0	0.0	*	0.0	0.0	0.0
92-4-0	021	0.0	0.0	*	0.0	0.0	0.2
	022	24.4	92.6	0.55	110.2	0.0	0.0
	023	13.4	37.7	0.86	89.9	0.0	0.0
	024	25.0	207.0	0.27	221.9	0.0	28.9
	Mean	15.7	84.3		105.5	0.0	7.3
	Median	19.4	65.2	0.70	100.1	0.0	0.1

TABLE 3 (continued)

Phase I Condition	Subject	\bar{X} No. Trials w/Response	\bar{X} Overall Response Rate	\bar{X} Latency to First Response	\bar{X} Running Rate	\bar{X} TI Response Rate	\bar{X} ITI Response Rate
88-8-0	031	25.0	42.8	0.82	47.7	0.0	0.1
	032	10.2	3.1	0.54	8.2	0.0	0.0
	033	1.8	0.5	1.88	9.0	0.0	0.0
	034	25.0	63.2	0.18	64.6	0.0	0.1
	Mean	15.5	27.4	0.76	32.4	0.0	0.0
	Median	17.6	24.9	0.68	28.4	0.0	0.0
88-4-4	041	25.0	143.2	0.17	149.9	5.9	0.1
	042	25.0	144.1	0.54	166.6	4.9	0.0
	043	19.0	21.5	0.49	32.6	0.0	9.2
	044	25.0	83.0	0.17	86.6	0.0	0.0
	Mean	23.5	97.9	0.34	108.9	2.7	2.3
	Median	25.0	113.1	0.33	118.3	2.5	0.1
80-16-0	051	9.0	5.9	1.03	13.8	0.0	1.0
	052	23.4	17.3	4.71	25.9	0.0	0.0
	053	23.4	3.3	1.56	6.8	0.0	0.0
	054	12.4	21.9	3.09	28.7	0.0	1.1
	Mean	17.1	12.1	2.60	18.8	0.0	0.5
	Median	18.4	11.6	2.33	19.9	0.0	0.5

TABLE 3 (continued)

Phase I Condition	Subject	\bar{X} No. Trials w/Response	\bar{X} Overall Response Rate	\bar{X} Latency to First Response	\bar{X} Running Rate	\bar{X} TI Response Rate	\bar{X} ITI Response Rate
80-12-4	061	1.4	0.5	2.20	6.7	0.0	0.1
	062	0.4	0.1	0.80	4.0	0.0	0.0
	063	5.4	1.5	1.34	7.8	0.0	0.0
	064	4.6	1.7	1.66	9.9	0.0	0.0
	Mean	2.9	0.9	1.50	7.1	0.0	0.0
	Median	3.0	1.0	1.50	7.3	0.0	0.0
80-8-8	072	5.8	3.2	1.43	14.9	1.3	0.2
	073	0.4	0.1	0.40	3.4	0.0	0.0
	074	25.0	110.3	0.25	113.9	0.6	4.0
	Mean	10.4	37.9	0.69	44.1	0.6	1.4
	Median	5.8	3.2	0.40	14.9	0.6	0.2
80-4-12	081	1.6	0.9	0.90	14.8	0.0	0.0
	082	15.2	31.8	0.68	61.1	0.5	0.0
	083	0.0	0.0	*	0.0	0.0	0.0
	084	0.4	0.2	0.20	8.0	0.0	0.2
	Mean	4.3	8.3		20.9	0.1	0.1
	Median	1.0	0.6	0.68	11.2	0.0	0.0

TABLE 3 (continued)

Phase I Condition	Subject	\bar{X} No. Trials w/Response	\bar{X} Overall Response Rate	\bar{X} Latency to First Response	\bar{X} Running Rate	\bar{X} TI Response Rate	\bar{X} ITI Response Rate
64-16-16	121	0.0	0.0	*	0.0	0.0	0.0
	122	0.0	0.0	*	0.0	0.0	0.0
	123	0.0	0.0	*	0.0	0.0	0.0
	124	0.2	0.0	2.4	3.0	0.2	0.2
	Mean	0.1	0.0	2.4	0.8	0.1	
	Median	0.0	0.0	2.4	0.0	0.0	0.0
48-16-32	131	0.0	0.0	*	0.0	0.0	0.0
	132	0.2	0.0	0.20	0.8	0.0	0.0
	133	0.0	0.0	*	0.0	0.0	0.0
	134	0.0	0.0	*	0.0	0.0	0.0
	Mean	0.1	0.0	0.2	0.2	0.0	0.0
	Median	0.0	0.0	0.2	0.0	0.0	0.0
48-8-40	141	0.0	0.0	*	0.0	0.0	0.0
	142	0.0	0.0	*	0.0	0.0	0.0
	143	0.0	0.0	*	0.0	0.0	0.0
	144	0.0	0.0	*	0.0	0.0	0.0
	Mean	0.0	0.0	*	0.0	0.0	0.0
	Median	0.0	0.0	*	0.0	0.0	0.0

TABLE 3 (continued)

Phase I Condition	Subject	\bar{X} No. Trials w/Response	\bar{X} Overall Response Rate	\bar{X} Latency to First Response	\bar{X} Running Rate	\bar{X} TI Response Rate	\bar{X} ITI Response Rate
48-4-44	151	0.0	0.0	*	0.0	0.0	0.0
	152	0.0	0.0	*	0.0	0.0	0.0
	153	0.0	0.0	*	0.0	0.0	0.1
	154	0.0	0.0	*	0.0	0.0	0.1
	Mean	0.0	0.0	*	0.0	0.0	0.1
	Median	0.0	0.0	*	0.0	0.0	0.1
32-48-16	161	1.4	0.1	16.27	1.9	0.2	0.1
	162	0.6	0.1	5.80	2.7	14.8	4.2
	163	3.2	0.3	14.82	4.4	20.0	4.4
	164	1.2	0.1	20.93	3.5	4.8	0.3
	Mean	1.6	0.1	14.5	3.1	9.9	2.2
	Median	1.3	0.1	15.5	3.1	9.8	2.3
32-32-32	171	0.6	0.0	6.80	12.4	0.0	0.1
	172	9.6	2.8	13.84	12.7	2.6	0.3
	173	0.2	0.0	3.00	0.7	0.2	0.2
	174	0.6	0.1	11.00	8.3	0.2	0.2
	Mean	2.8	0.7	8.7	8.5	0.8	0.2
	Median	0.6	0.1	8.9	10.4	0.2	0.2

TABLE 3 (continued)

Phase I Condition	Subject	\bar{X} No. Trials w/Response	\bar{X} Overall Response Rate	\bar{X} Latency to First Response	\bar{X} Running Rate	\bar{X} TI Response Rate	\bar{X} ITI Response Rate
32-8-56	181	0.2	0.1	1.20	12.0	0.0	0.0
	183	0.0	0.0	*	0.0	0.0	0.0
	184	0.0	0.0	*	0.0	0.0	0.0
	Mean	0.1	0.0	1.20	4.0	0.0	0.0
	Median	0.0	0.0	1.20	0.0	0.0	0.0
240-12-4	091	18.6	14.5	0.83	20.7	0.6	0.9
	092	24.4	18.8	1.02	21.5	0.0	0.1
	093	0.8	0.2	0.60	2.3	0.0	0.0
	094	25.0	147.6	0.52	154.2	23.3	0.2
	Mean	17.2	45.3	0.74	49.7	5.9	0.3
Median	21.5	16.7	0.72	21.1	0.3	0.2	
240-8-8	101	24.8	77.1	0.85	86.9	1.2	0.2
	102	25.0	79.7	0.91	90.3	0.8	0.1
	103	25.0	108.2	0.90	122.1	17.9	0.6
	104	14.0	16.7	1.54	35.7	0.1	0.0
	Mean	22.2	70.5	1.05	83.8	5.0	0.2
Median	24.9	78.4	0.91	88.6	1.0	0.2	

TABLE 3 (continued)

Phase I Condition	Subject	\bar{X} No. Trials w/Response	\bar{X} Overall Response Rate	\bar{X} Latency to First Response	\bar{X} Running Rate	\bar{X} TI Response Rate	\bar{X} ITI Response Rate
240-4-12	191	1.0	0.7	1.07	19.5	0.0	0.0
	192	24.8	67.6	1.23	103.2	0.4	0.1
	193	0	0.0	*	0.0	0.0	0.0
	194	18.2	90.0	0.73	121.5	0.8	0.0
	Mean	11.0	39.6	1.01	61.1	0.3	0.0
Median		9.6	34.2	1.07	61.4	0.2	0.0
224-4-28	201	0.0	0.0	*	0.0	0.0	0.0
	202	2.4	2.2	1.65	34.3	0.0	0.4
	203	0.0	0.0	*	0.0	0.0	0.0
	204	0.6	1.6	0.90	12.3	0.0	0.1
	Mean	0.8	0.9	1.28	11.6	0.0	0.1
Median		0.3	0.8	1.28	6.2	0.0	0.1

with one exception: The 4-sec trace group responded more often than the 0-sec trace group with a 4-sec CS at the 96-sec IRI. Secondly, those subjects exposed to the 256 IRI tend to respond more often than those subjects exposed to shorter IRIs.

A similar pattern of results is found in the median overall rates of responding which are presented in Figure 6. In addition, one other relationship appears in this figure that was not evident in the preceding one. The overall rate of responding in the three groups with zero trace intervals decreases with increases in CS duration. It should be noted that this overall measure is computed by dividing total responses by total CS time and thus weights the total number of responses by the different opportunities to respond in different experimental groups. The data for individual subjects appear in column 2 of Table 3.

It is unlikely that differing opportunities to respond influence the terminal measures appreciably: When subjects do respond the mean and median latencies to the first response are in all but two groups less than the 4-sec minimum CS duration. The average latencies of individual subjects, which are presented in column 3 of Table 3, are consistent with the group statistics, showing only 8 of the 78 subjects responding with average latencies greater than the minimum CS duration. Figure 7 shows the median latencies to the first response on trials with a response. Although the latencies decrease as a function of trace interval within an ISI, Figure 10 shows that this effect seems attributable primarily to the relationship between CS duration and latency. At a given trace interval the latency increases with increasing CS duration with the exception of the 16-sec CS duration at the 32-sec

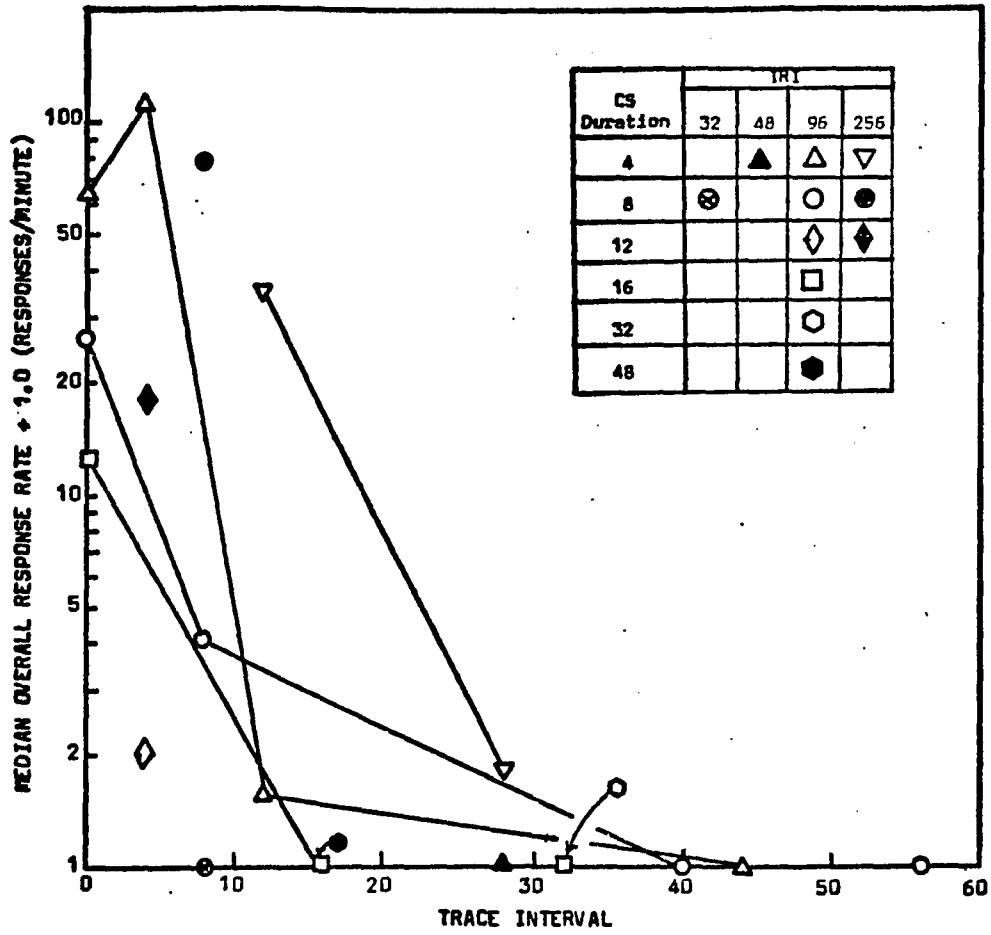


Figure 6. The median overall rate of responding during the final 125 trials of phase I is shown as a function of trace interval for different CS durations.

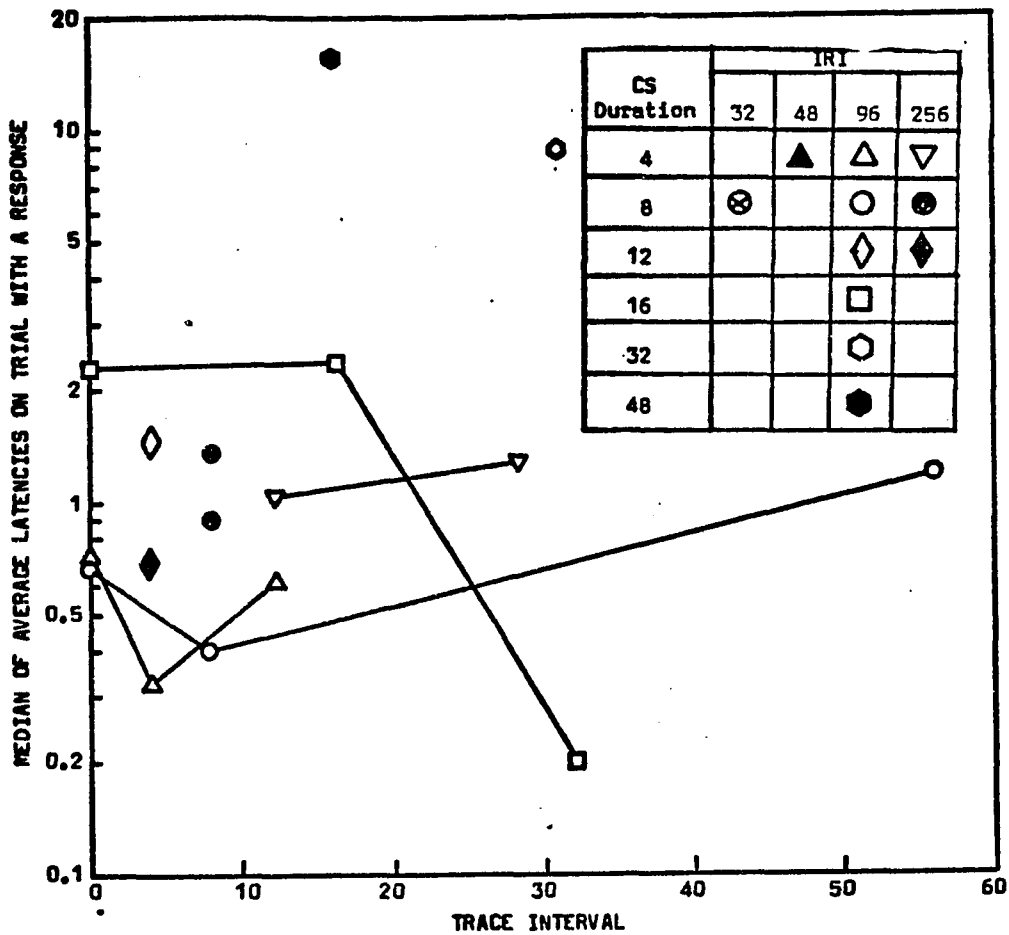


Figure 7. The median of the latency to the first response on trials with a response during the last 125 trials of phase I is shown as a function of trace interval for different CS durations.

trace interval. This one anomalous point is probably unreliable in as much as it is based on only one response of the sole subject in that group that responded during the final 125 trials. It is evident that, in terminal performance, latencies to the first response are primarily under the direct control of CS duration and are not solely attributable to differences in opportunities to respond with different CS durations.

The rate of responding, once the subject has started responding, is called the "running rate"; it is shown for individual subjects in column 4 of Table 3. The group medians plus one are plotted as a function of experimental group in Figure 8. The running rate shows a pattern of change similar to the changes exhibited by the overall rate as a function of the experimental treatment. With the one exception of group 88-4-4, the running rate decreases as a function of trace interval for all CS durations. The rates of responding are higher in groups exposed to the 256-sec IRI than in comparable groups with shorter IRIs and, lastly, the rate decreases as CS duration increases in the three delay conditioning groups.

The effects of the experimental manipulation on the terminal performance measures can best be summarized in terms of the parameters that seem to influence them the most. For a given CS duration, the number of trials with at least one response, the overall rate of responding, and the running rate all tend to decrease with increases in the trace interval. Latencies to the first peck appear to be primarily determined by CS duration, and, in the delay conditioning groups, as CS duration increases the overall and running rates decrease. Finally, the rates of responding are higher in groups exposed to the 256-sec IRI than in those groups exposed to shorter IRIs.

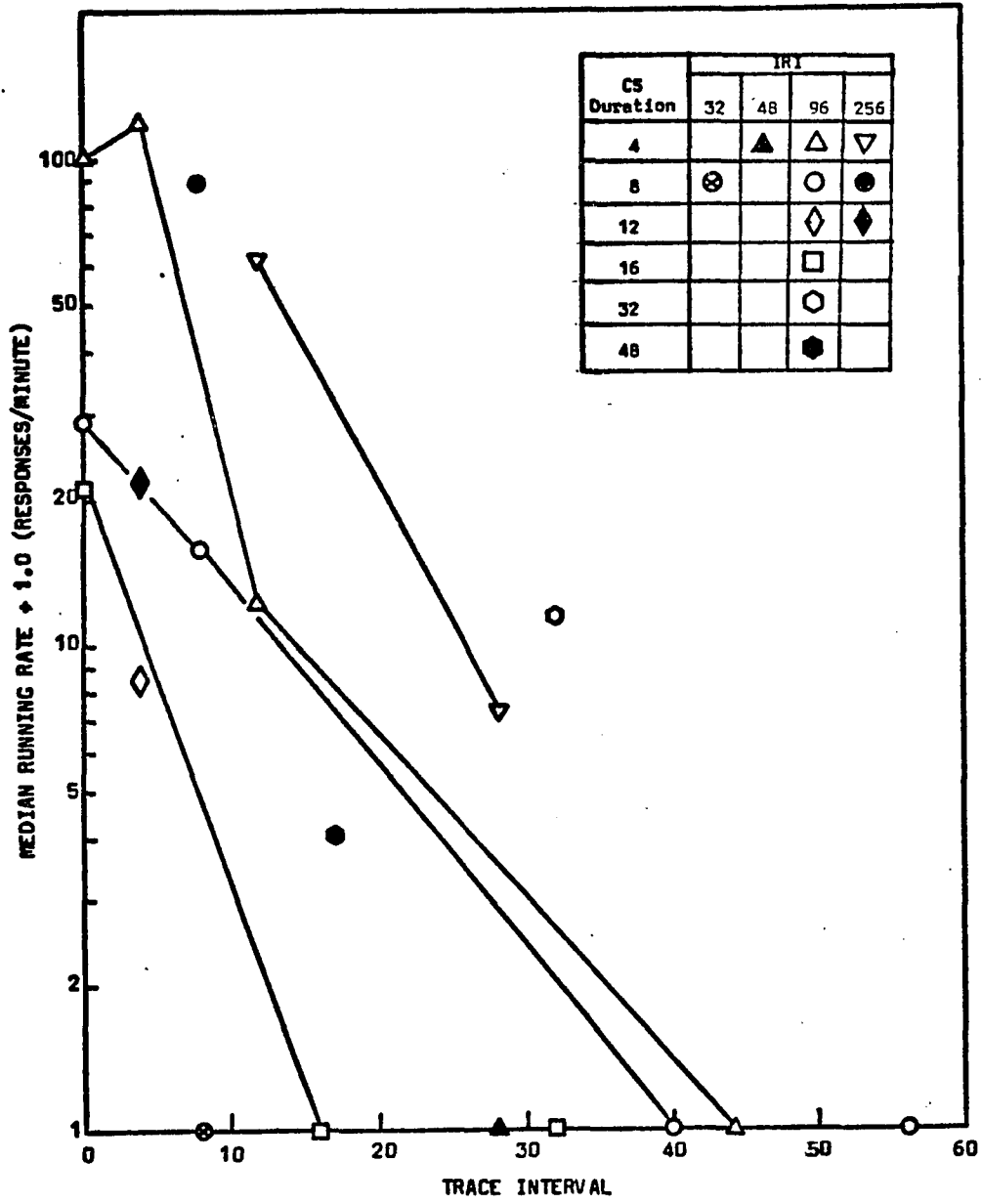


Figure 8. The median running rate of responding is shown as a function of trace interval for different CS durations.

Initial and terminal performance within CS presentations. The latency to the first response on each trial with a response was recorded for each subject during the course of the experiment. Figure 9 shows the frequency of occurrence of these latencies for all subjects that responded. The left-hand portion of each subject's graph shows the frequency of occurrence of each of the latencies during the first 100 trials following the first response. The right-hand portion of each graph is based on data collected for each subject during the final 100 trials of Phase I. The ISIs for most experimental groups were divided into eight equal class intervals. The class intervals are denoted by their upper limits on the abscissa of Figure 9. The latencies for groups 32-48-16 and 32-32-32, the groups with the longest CSs, are presented in 16 4-sec class intervals. This device is used to facilitate comparisons of within-CS responding across groups by keeping the sizes of the class intervals small.

There are several striking features of these distributions. First, the modal latency tends to fall in the first or second class interval for all subjects except those exposed to the longest CS durations. The small frequency of trial responses in the long-CS groups make their distributions hard to evaluate. Second, the modal latency either remains the same or shortens, and the number of short latencies tends to increase from the beginning of training until the end. This effect is consistent with the changes in latencies of the group statistics portrayed in Figures 2 and 7. Third, in all but the long-CS groups, very few first responses occur during the TI. Most of these TI responses occurred early in training and are all but nonexistent in the data

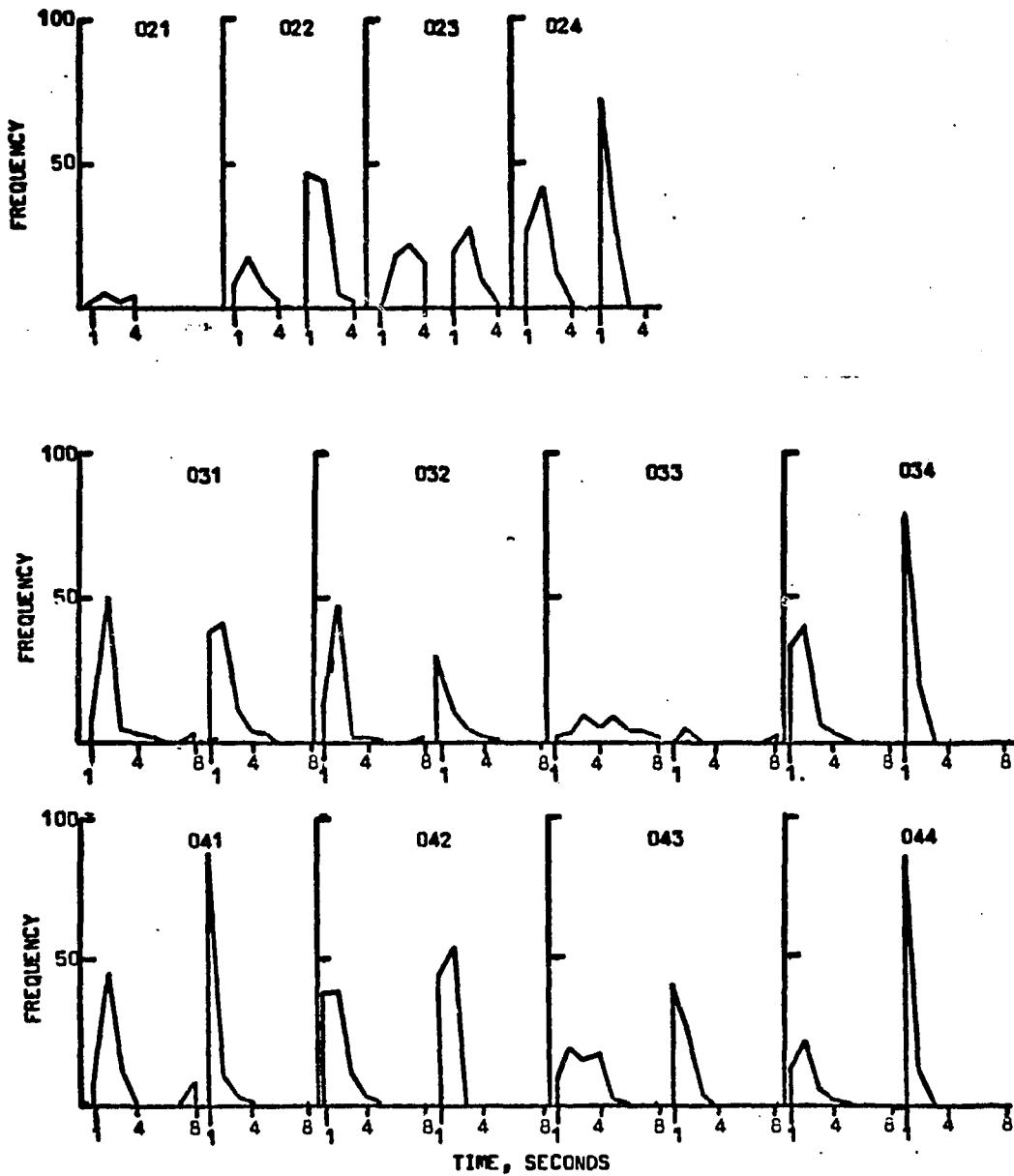


Figure 9. The frequency of occurrence of different latencies to the first response is shown for individual subjects. The left portion of each graph represents the first 100 trials after the first trial with a response. The right portion of each graph shows the last 100 trials of phase I. Every fourth class interval is denoted by its upper limit.

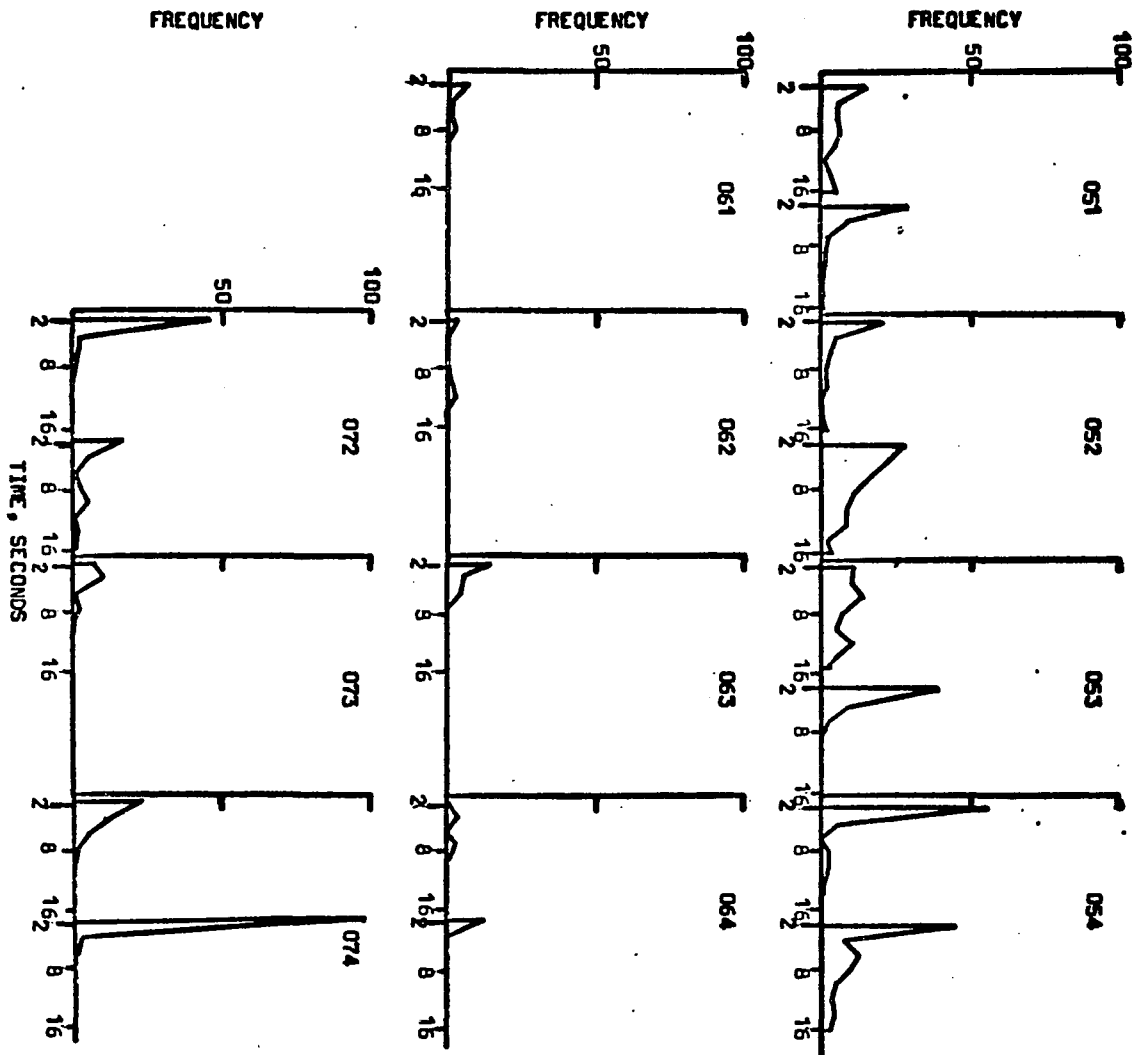


Figure 9. (continued)

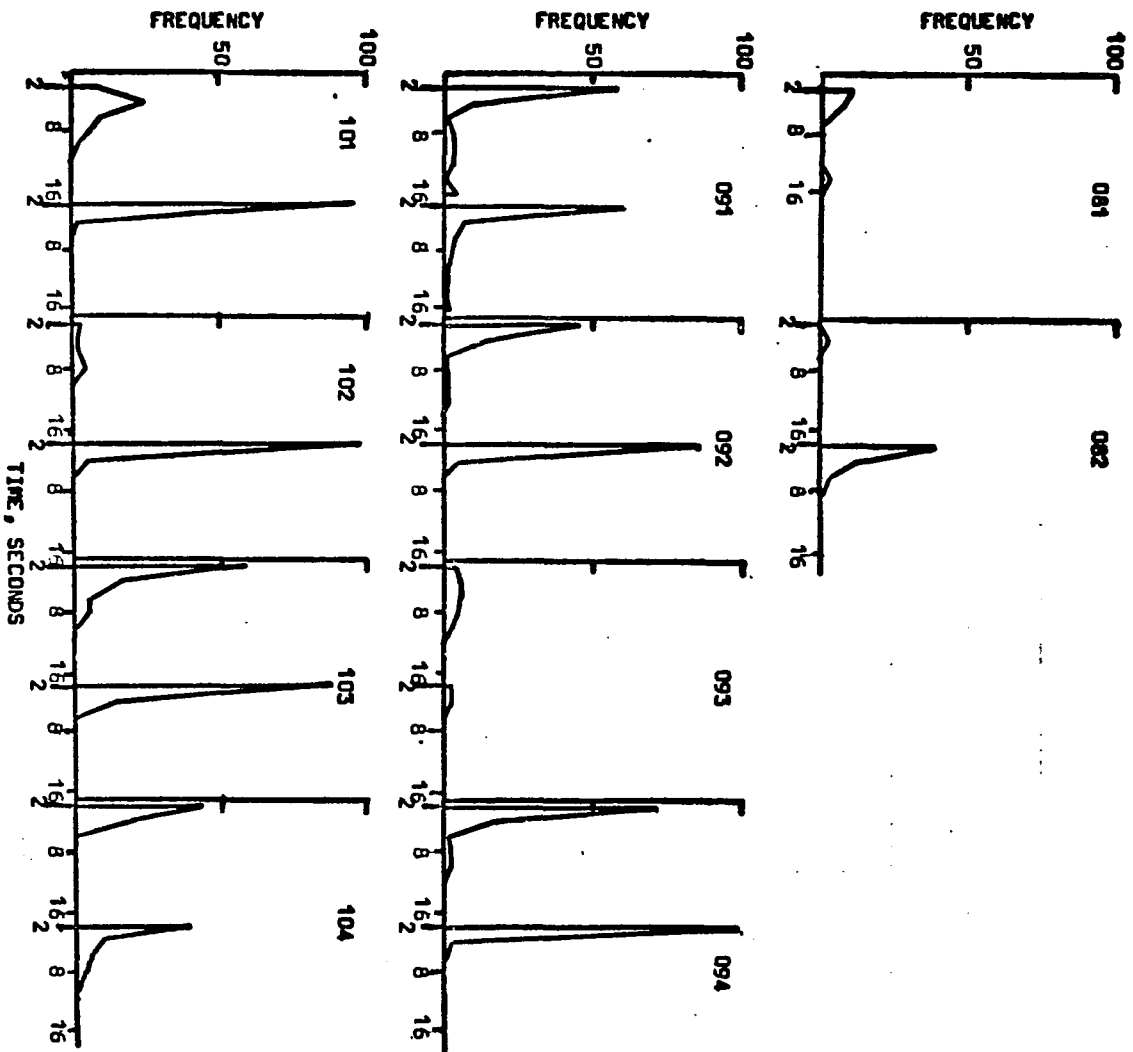


Figure 9. (continued)

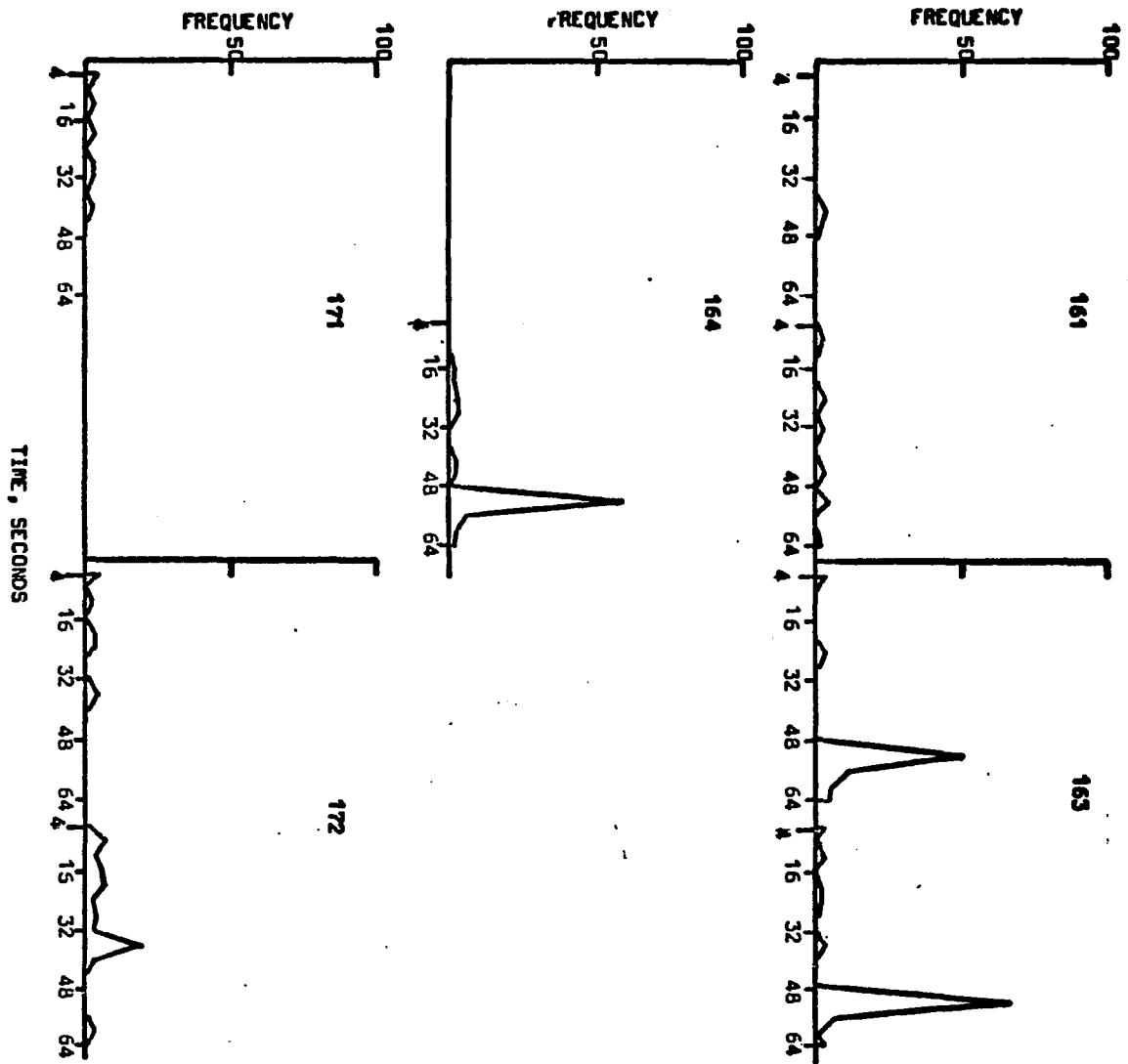


Figure 9. (continued)

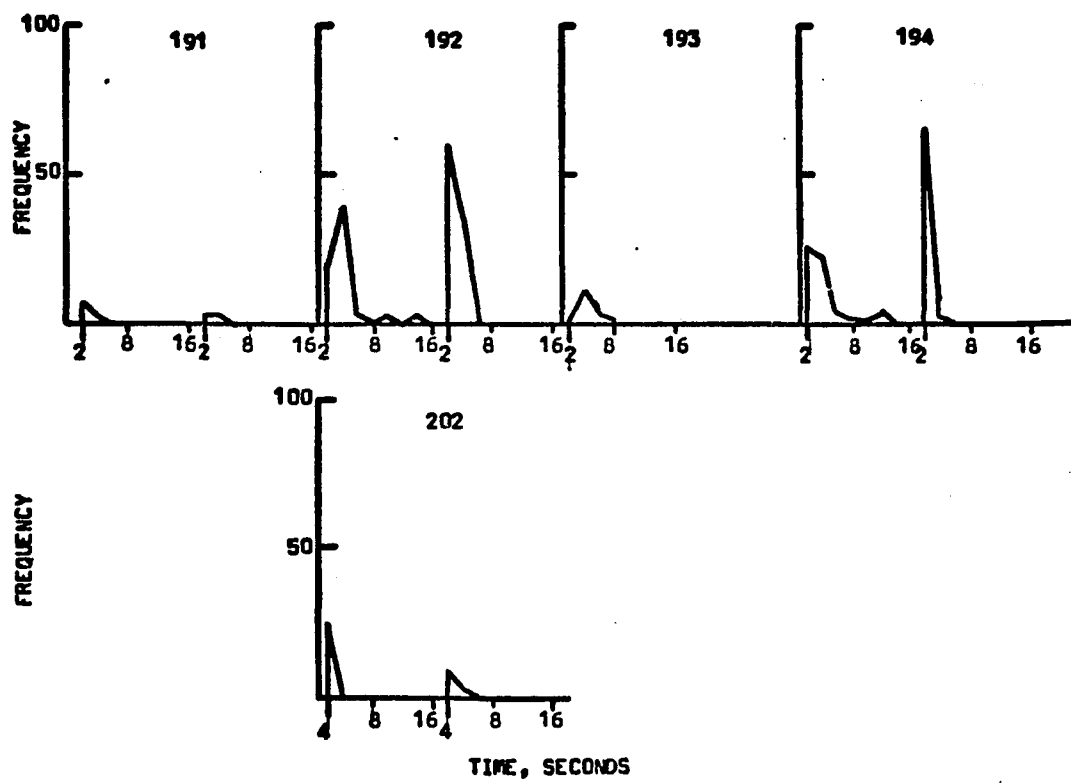


Figure 9. (continued)

collected during the last 100 trials. A different pattern of trace interval responding emerged in the groups exposed to long-CS durations. Figure 9 shows that for three subjects in group 32-48-16 and one subject in group 32-32-32, the first response often occurred during the TI.

One transformation on frequency distributions that has been proposed as an estimate of response probability in time is the response per opportunity distribution. This distribution is computed by dividing the number of times a response in a particular temporal interval occurs by the number of times the subject had waited as long or longer than that particular interval to respond. This statistic has been most widely used in the analysis of free-responding in which the time between successive responses is the datum of interest. An analogous distribution of statistics was computed for the data depicted in Figure 9. The frequency of occurrence of first responses in a particular latency class was divided by the number of CS presentations in which the subjects had not responded sooner. These data corroborate the strong control by CS onset depicted in Figure 9. The mode of these distributions was in the first or second class interval for 83% of the subjects. The mode for the remaining subjects was in the third bin with three exceptions. The mode for two subjects was in the fourth class interval and the mode for one other subject was in the fifth bin. Hence, these statistics indicated that the probability of a response was highest soon after CS onset and declined thereafter.

These data suggest that the occurrence of the first CS response is largely controlled by the early relative portions of the CS. Subjects tend to respond soon after CS onset or not at all. The next figure

shows that the control of responding by early portions of the CS is often not restricted to the first response.

Each CS duration was divided into eight equal class sizes, responses were sorted, and the frequency of occurrence of responses during each of the class intervals was recorded. Figure 10 shows the proportion of the total CS responses occurring during each eighth of the CS presentation. The solid lines represent performance early in training and the broken lines represent the pattern of responding during the final 125 trials. Two distinct patterns of CS responding can be seen in Figure 10. The first pattern tends to characterize responding in all the groups early in training. The proportion of CS responses tends to rise to a maximum by the third or fourth class interval and then remain relatively constant for the remainder of the CS presentation. The performance of some subjects early in training and many subjects at the end of training is better characterized by a different pattern of responding. The proportion of responses in each class interval for these subjects peaks within the first one or two class intervals and then declines throughout the remainder of the CS presentation. The different patterns of responding do not seem to be systematically related to the experimental manipulation.

The data collected on the time of occurrence of responding during the CS suggest that the CS onset exerts strong control over the occurrence of the first response and that for many subjects the tendency to respond is highest during the early portions of the CS. Other subjects also made their first responses early in the CS period and then responded consistently throughout the remainder of the CS. There was no evidence in any subject of accelerated responding during CS presentation.

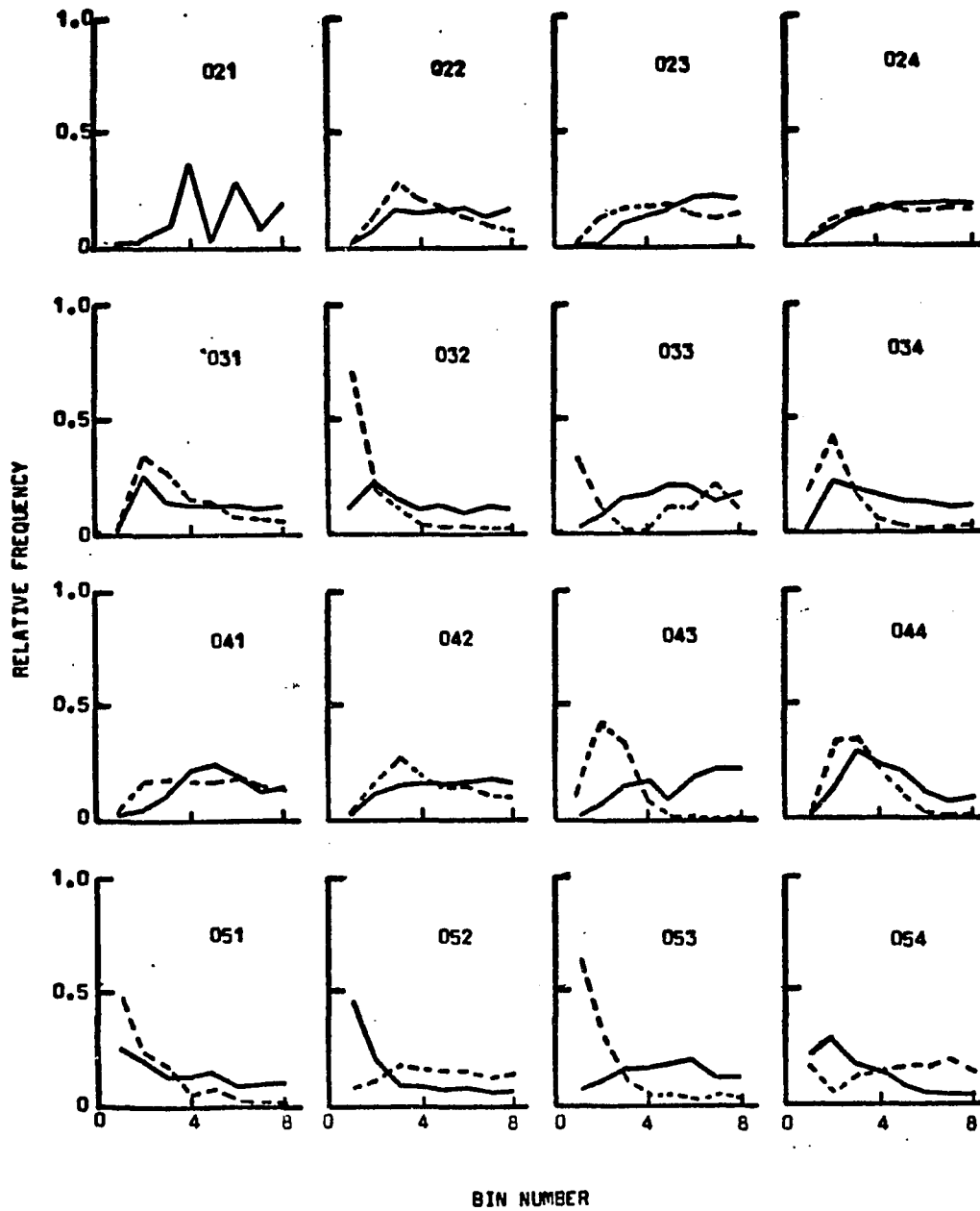


Figure 10. The proportion of total responses falling in different octiles of the CS presentation. The solid lines represent performance during the first 125 trials of phase I after the first trial with a response. The broken lines depict performance during the final 125 trials of phase I.

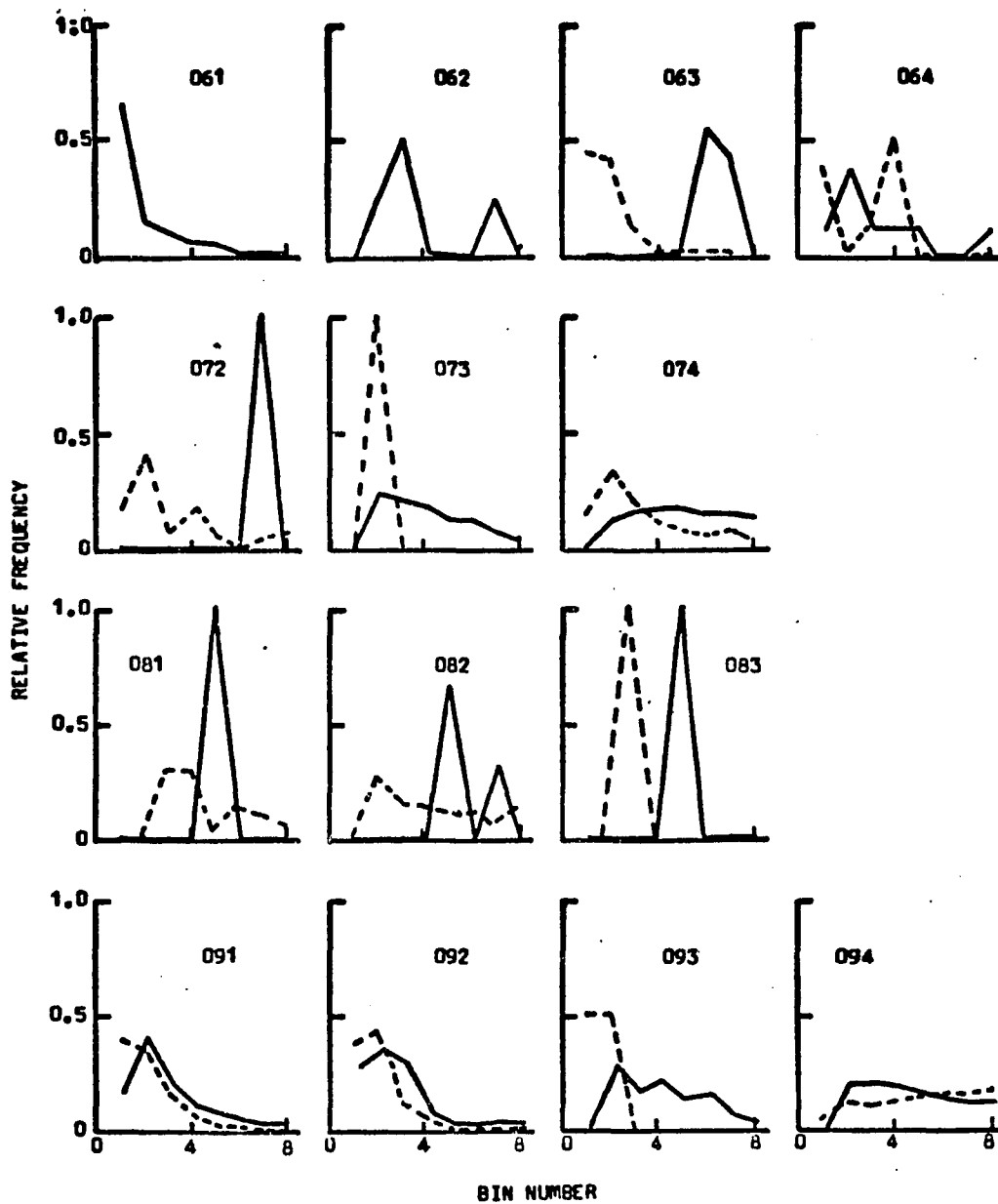


Figure 10. (continued)

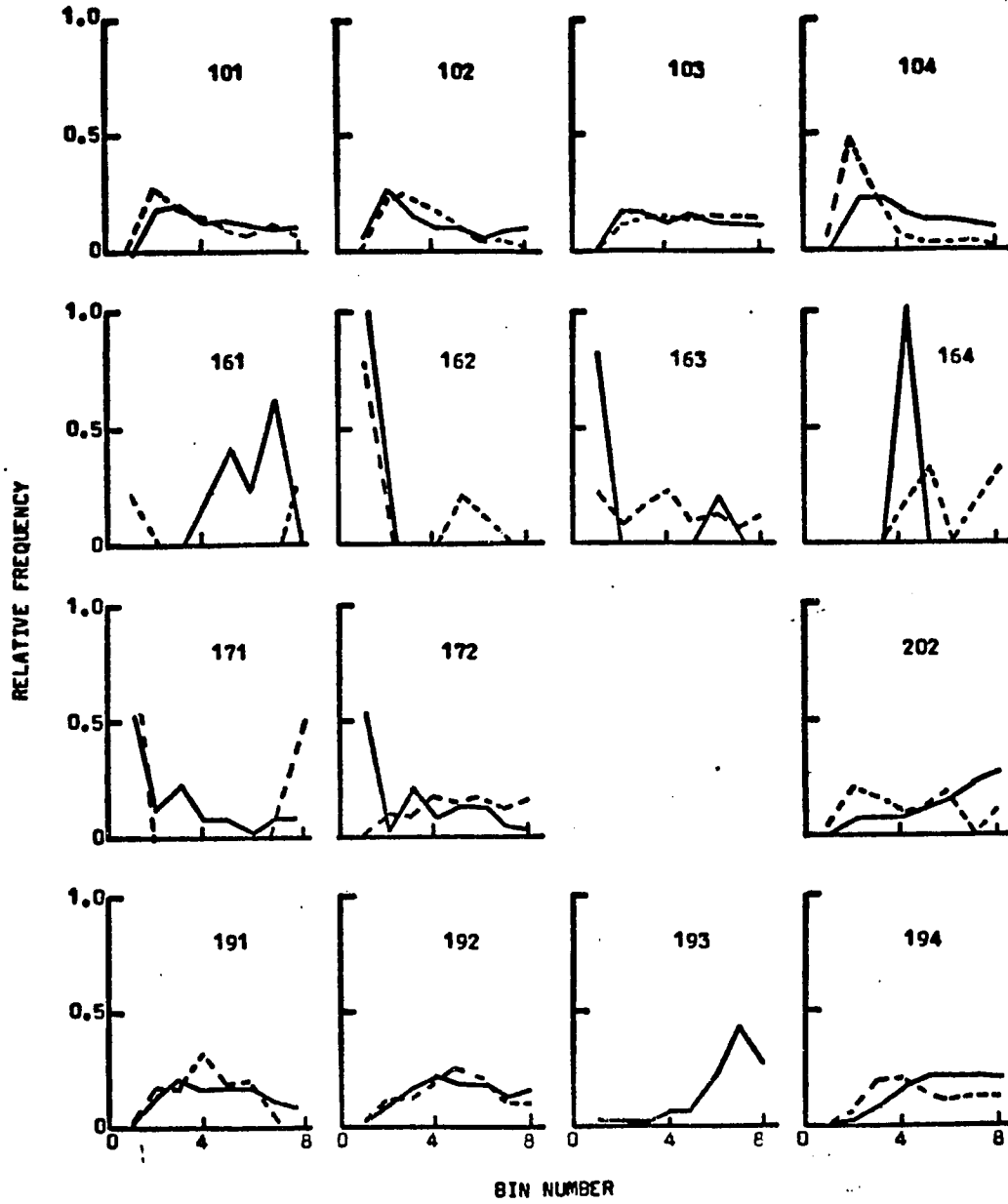


Figure 10. (continued)

Intertrial interval and trace interval responding. The intertrial interval occasioned very little, if any, responding. In all of the experimental groups except groups 32-48-16 and 32-32-32, the ITI rates were far below the rates of responding during the CS. This difference can be seen in the performance of individual subjects by comparing column 5 with column 2 of Table 3. It is also evident from the group statistics that very little responding occurred during the ITI except in group 32-48-16.

The mean trace interval response rates for experimental groups and for individual subjects are presented in column 6 of Table 3. Both the individual and group statistics show that most subjects did not respond during the TI. The TI responding that did occur seemed to often be "run over" from CS responding in many of the groups. Event records were taken for several days toward the end of training for half the subjects in each group. Inspection of these records suggested that, for all groups but group 32-48-16 and group 32-32-32, the trace interval responses occurred when subjects did not stop pecking after CS offset. In the two exceptional groups, TI responding occurred at various times during the TI even when CS responding had not occurred.

In summary, the Phase I results indicate that the temporal relationship between the CS and US is an important determinant of the level of key pecking that will occur when the illumination of a response key is followed by grain. The primary effect is on the level of key pecking that occurs during the CS presentation. CS responding decreases with decreases in the IRI, with increases in CS duration, and with increases in the length of the trace interval.

Phase II

The second phase of the experiment consisted of exposing those subjects that responded on fewer than 15 trials during Phase I to either an 80-12-4 procedure or an 80-4-12 procedure. The results of Phase II generally replicated the differences found between groups 80-12-4 and 80-4-12 during the first phase of the experiment. The group means and medians shown in Table 4 indicate that those subjects exposed to the 80-12-4 condition generally responded earlier in training and more often later in training than those subjects exposed to the 80-4-12 procedure. Subjects in the 80-4-12 group were exposed to about twice as many trials as the 80-12-4 group before making their first response. The median trial of the fifth peck shown in column 2 of Table 4 was 255 for the former group and 450 for the latter. In both instances, this was about 50 trials more than subjects took to reach the same criterion in each of the comparable Phase I groups.

The last four columns of Table 4 show averages computed over the last 125 trials of Phase II for each of the subjects. The median number of trials with a response and overall CS response rate were higher for those subjects exposed to the 80-12-4 condition than those exposed to the 80-4-12 condition. The median number of trials with a response and overall response rate were higher for Phase II subjects exposed to the 80-12-4 condition than they were for subjects exposed to that condition in Phase I. These measures in the 80-4-12 Phase II subjects were lower than they were for subjects exposed to comparable conditions in Phase I.

A more detailed analysis of the Phase II data indicated that the specific Phase I history of the subjects influenced the Phase II results,

TABLE 4

MEAN INITIAL AND TERMINAL STATISTICS FOR PHASE II

Phase II 80-12-4 Condition

Phase I Condition	Subject	Trial of First Response	Trial of Fifth Response	\bar{X} No. Trials w/Response	\bar{X} Overall Response Rate	\bar{X} TI Response Rate	\bar{X} ITI Response Rate
16-8-8	111	17	252	12.4	4.4	0.2	0.0
	113	160	183	7.0	1.8	4.4	0.1
16-4-28	011	112	178	14.0	6.4	0.0	0.0
	013	431	450	0.0	0.0	0.0	0.0
64-16-16	121	180	188	21.2	12.0	0.0	0.0
	123	321	329	16.0	6.8	11.6	0.1
48-16-32	131	446	450	0.4	0.1	3.0	0.4
	133	450	450	0.0	0.0	0.0	0.0
48-8-40	141	1	165	0.0	0.0	0.0	0.0
	143	9	450	0.0	0.0	0.0	0.0
48-4-44	151	1	438	0.4	0.1	3.7	0.2
	153	450	450	0.0	0.0	0.0	0.2
32-8-56	181	188	196	24.2	22.2	34.8	0.4
	183	43	255	5.0	2.3	0.4	0.1
224-4-28	203	7	65	21.6	13.0	25.2	0.6
Mean		187.70	299.90	7.64	4.32	5.21	.1
Median		160	255	5.0	1.8	0.2	.1

TABLE 4 (continued)
Phase II 80-4-12 Condition

Phase I Condition	Subject	Trial of First Response	Trial of Fifth Response	\bar{X} No. Trials w/Response	\bar{X} Overall Response Rate	\bar{X} TI Response Rate	\bar{X} ITI Response Rate
16-8-8	112	228	285	16.0	31.9	0.2	0.0
	114	235	257	22.0	43.1	0.2	0.1
16-4-28	012	5	10	19.2	66.7	0.4	0.2
	014	198	450	0.0	0.0	0.0	0.0
64-16-16	122	190	256	5.0	4.3	0.2	1.1
	124	126	158	13.4	11.3	0.0	0.0
48-16-32	132	198	438	0.6	0.4	0.0	0.0
	134	450	450	0.0	0.0	0.0	0.0
48-8-40	142	450	450	0.0	0.0	0.0	0.0
	144	450	450	0.0	0.0	0.0	0.0
48-4-44	152	450	450	0.0	0.0	0.0	0.0
	154	450	450	0.0	0.0	0.0	0.0
32-8-56	184	450	450	0.0	0.0	0.0	0.0
32-32-32	173	450	450	0.0	0.0	0.0	0.0
	174	450	450	0.0	0.0	0.0	0.0
224-4-28	204	66	189	1.0	1.4	0.0	0.0
Mean		302.88	352.69	4.85	9.95	0.06	0.09
Median		343	450	0.0	0.0	0.0	0.0

although the overall pattern of results appears to be similar in both phases. Table 5 lists all the Phase II subjects by their initial condition of exposure during Phase I. The columns are labeled according to the different dependent measures. An "A" in a particular cell of the matrix indicates that a particular subject's score on that dependent measure was above the median of the comparable Phase I group. An entry of "B" indicates that the score was below the comparable Phase I median and an empty cell indicates a score equal to the Phase I median.

Table 5 indicates that some histories facilitate responding during Phase II while others seem to inhibit responding. A majority of subjects in Phase I groups 16-8-8, 64-16-16, and 224-4-28 made their fifth response earlier in Phase II than those subjects initially exposed to the comparable conditions during Phase I. The occurrence of the fifth response was retarded in the majority of subjects exposed to conditions 48-16-32, 48-8-40, 48-4-44, 32-32-32, and 32-8-56 as compared to naive subjects exposed to either the 80-4-12 or 80-12-4 conditions. The subjects in group 64-16-16 were evenly split above and below the Phase I medians. The number of trials with a response was above the Phase I median for a majority of subjects in groups 16-8-8, 16-4-28, 64-16-16, and 32-8-56 and below the Phase I medians in groups 48-16-32, 48-8-40, 48-4-44, and 32-32-32. One subject in group 224-4-28 responded on more trials and the other subject responded on as many trials as the comparable Phase I groups. Overall response rate is enhanced by prior exposure to conditions 16-8-8, 16-4-28, 64-16-16, possibly 32-8-56, and 224-4-28. The overall levels of responding are lowered with prior exposure to conditions 48-16-32, 48-8-40, 48-8-44, and 32-32-32.

TABLE 5
 THE RELATIONSHIP BETWEEN INDIVIDUAL SUBJECTS' PHASE II
 PERFORMANCE AND COMPARABLE PHASE I MEDIANS

Subject	Trial of Fifth Response	No. Trials w/Response	Overall Response Rate	TI Response Rate	ITI Response Rate
111	A	A	A	A	A
113	B	A	A	A	A
112	B	A	A	A	B
114	B	A	A	A	A
011	B	A	A		B
013	A	A	A		A
012	B	A	A	A	A
014	A	B	B	B	B
121	B	A	A		B
123	A	A	A	A	A
122	B	A	A	A	A
124	B	A	A	B	B
131	A	B	B	A	A
133	A	B	B		B
132	A	B	B	B	
134	A	B	B	B	B
141	B	B	B		B
143	A	B	B		B
142	A	B	B	B	B
144	A	B	B	B	B
151	A	B	B	A	A
153	A	B	B		A
152	A	B	B	B	B
154	A	B	B	B	B
181	B	A	A	A	A
183	A	A	A	A	A
184	A	B	B	B	B
173	A	B	B	B	B
174	A	B	B	B	B
203	B	A	A	A	A
204	B		A	B	B

The most consistent pattern of results with regard to the rate of responding during the TI has to do with the Phase II condition rather than the specific history of each subject. Subjects exposed to condition 80-12-4 during Phase II all responded at or above the Phase I median overall TI response rate for that group. On the other hand, TI response rates for 75% of the subjects exposed to the 80-4-12 condition were below the comparable Phase I median.

The ITI rates were not systematically different during the two phases of the experiment.

DISCUSSION

Acquisition and Maintenance

The current study replicates previous findings and extends the analysis of the effects of varying the temporal relationship between CS and US on the acquisition and maintenance of key pecking in the pigeon. Previous research has focused on the effects of varying the CS duration and ITI length (Terrace et al., 1975; Groves, 1974; Baldock, 1974; Griffin, 1975). The results of these studies have best been summarized in terms of functions relating the speed of acquisition and/or terminal performance levels to either the ratio of CS duration to IRI or ratio of CS duration to ITI. The present data indicate that these ratios are not a sufficient summary of all temporal parameters. The ratio of CS to IRI can be held constant when the trace interval is increased for a CS of constant duration. Obviously, the large changes in behavior that this manipulation produces are not paralleled by changes in the ratio. The ratio of CS to ITI increases with increasing CS duration but decreases with increasing trace intervals within an ISI. Both these manipulations retard acquisition and response rates. This ratio, furthermore, is equal for condition 80-4-12 and condition 240-12-4 yet the behavior in the two groups was very different. The effects of the experimental manipulation are therefore inconsistent with the concomitant changes in the ratio of CS to ITI.

The ratios described above may be viewed as special cases of a more general relationship that describes not only the effects of varying CS

and ITI duration but also includes the effects of varying the TI. Previous studies varying ratios have employed delay procedures. Thus, CS duration was equal to the ISI. The bird's sensitivity to CS and ITI manipulations is therefore equivalently described as a function of the ratio of ISI to IRI or ratio of ISI to ITI. If this ratio is weighted by the ratio of CS duration to ISI, a more general metric that takes into account trace interval durations is yielded. The variable

$$\frac{\text{ISI duration}}{\text{IRI duration}} \times \frac{\text{ISI duration}}{\text{CS duration}}$$

changes in appropriate ways as a result of the manipulation of temporal parameters. This variable will be referred to as λ (lambda). It reduces to the previously employed ratio in delay procedures and increases geometrically with increases in the trace interval. A similar variable employing the ITI instead of the IRI can be generated in an analogous fashion.

These variables do an adequate job of describing the acquisition and terminal performance functions generated during Phase I of the experiment. The ratio formed with the IRI does a slightly better job of making ordinal predictions about data and thus it is the only one presented here. It should be noted, however, that the large amount of variability in the data does not permit definitive statements about the efficacy of one ratio over another.

Figure 11 shows the median number of trials to the fifth peck as a function of λ . These medians are a monotonic increasing function of λ . Two points fall substantially below the general function. These are the medians associated with groups 32-48-16 and 32-32-32. It is

A	92-4-0	F	80-16-0	K	64-16-16	P	48-16-32
B	88-8-0	G	80-12-4	L	32-48-16	Q	48-8-40
C	240-12-4	H	240-4-12	M	16-8-8	R	32-8-56
D	240-8-8	I	80-8-8	N	224-4-28	S	16-4-28
E	88-4-4	J	80-4-12	O	32-32-32	T	48-4-44

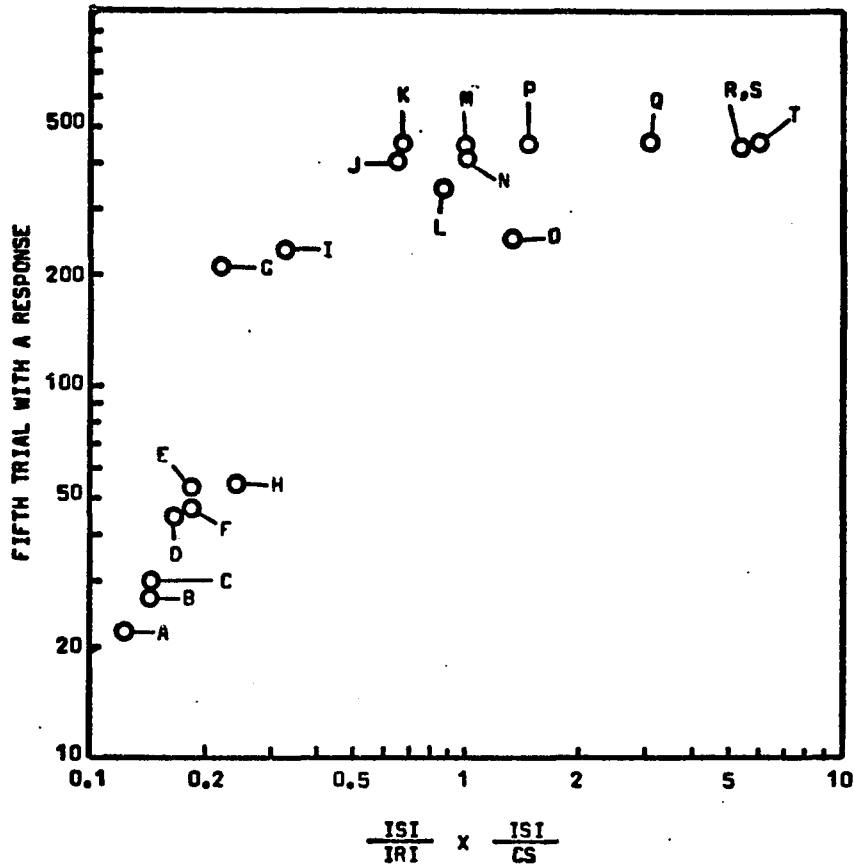


Figure 11. The fifth trial with a response is shown as a function of λ where,

$$\lambda = \frac{ISI}{IRI} \times \frac{ISI}{CS}$$

unclear why these two points lie below the others. The general function relating this measure to λ is probably an exponential one with an exponent greater than one. The points at the maximum number of trials may merely be a byproduct of terminating the experiment at 450 trials and not an indicant of the relationship between large values of λ and the trial of the fifth response. If these subjects had never made five pecks, then a more appropriate relation would be a function in the family $Y = K - 1e^{-\lambda m}$, where Y is equal to the median number of pecks, K is the asymptote and e , 1 , and m are constants.

Figure 12 shows the overall rate of responding during the last 125 trials of Phase I as a function of λ . These rates generally decrease monotonically with increases in λ . Either a power function with a negative exponent or an equation of the form $\text{Rate} = K - 1e^{-\lambda m}$ might serve to describe the relationship.

The discussion of the conceptual meaning of these relationships is deferred to a later portion of this section. For now, it is sufficient to point out that the acquisition and maintenance of responding is adequately described as some function of λ where λ is equal to the ratio of the ISI duration to IRI length multiplied by the ratio of ISI duration to CS duration.

Within-CS Responding

The primary finding of the within-CS analysis was that the first response and, for many subjects, subsequent responses occurred during early portions of the CS presentation. These data are of interest for several reasons. One view of the present study is that the primary manipulation was arranging for different CS-US contiguities. These

A	92-4-0	F	80-16-0	K	64-16-16	P	48-16-32
B	88-8-0	G	80-12-4	L	32-48-16	Q	48-8-40
C	240-12-4	H	240-4-12	M	16-8-8	R	32-8-56
D	240-8-8	I	80-8-8	N	224-4-28	S	16-4-28
E	88-4-4	J	80-4-12	O	32-32-32	T	48-4-44

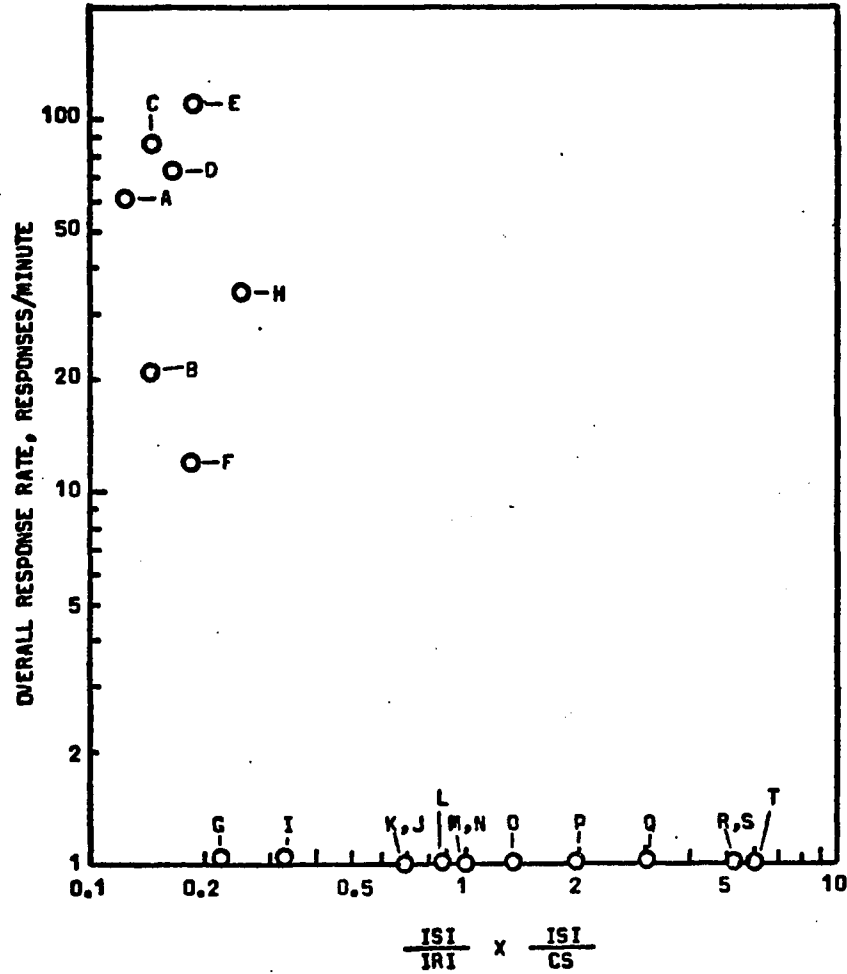


Figure 12. The overall rate of responding is shown as a function of λ .

manipulations have strong effects, yet the within-CS responding seems to indicate that a contiguity explanation of responding is not a sufficient account. The latter portions of a CS are more contiguous with grain presentation and thus it might be expected that key pecking should increase as the CS progresses and that subjects' latencies should increase substantially as CS onset becomes more and more remote from grain presentation. The data are not supportive of either of these expectations. First, early in training the latencies are unrelated to the ISI per se. The only variable consistently affecting latencies early in training is CS duration, as shown in Figure 2. Figure 11 shows that late in training even these initial differences are attenuated. The individual latency distributions also indicate no increasing tendencies to respond as the CS progresses. Secondly, there is not a single distribution of the proportion of responses in eighths of the CS which shows the scalloped response patterning that would be expected purely on the basis of CS-US contiguity.

It is apparent that the onset of the CS is the most effective stimulus controlling key pecking. This is consistent with Kamin's (1965) demonstration, in a conditioned emotional response paradigm using rats, that CS onset is the most important stimulus change in producing conditioned suppression. In the case of "fear conditioning" the behavioral changes responsible for the suppression are long lasting and the suppression is sustained throughout the CS period. In the case of a pigeon pecking, no such persistent changes occur. Thus with CS onset as the most influential stimulus change, birds peck soon after CS onset and then often stop for the remainder of the CS presentation. The

effectiveness of CS onset as a salient stimulus may arise from two factors. First, increases in stimulation make more effective CSs than sustained or terminated stimulation (Kamin, 1965). Second, if the tendency to respond is a function of the information or relative reduction in uncertainty about the time of occurrence of the US, then CS onset provides more information about US occurrence relative to the preceding stimulus than do later portions of the CS relative to CS onset. This latter explanation assumes that there is not substantial control by the time since the last reinforcement, even in a procedure with a fixed ITI such as the one employed in the present study.

The pattern of responding within CS presentations may also be attributable to the development of other behaviors than key pecking during the latter portion of the CS. Most conditioning experiments focus on the occurrence of a single response, although several and often fixed sequences of responses are conditioned (see Morrison, 1974; Farris, 1967; Thompson & Sturm, 1965). Approach and contact with the food delivery site is often reported during CS presentations (Gilbert, 1971; Farthing, 1971), as well as off-key pecking (Woodruff, 1974; Barrera, 1974). Informal observation of some of the subjects in the present study indicated that both off-key pecking and hopper-directed behavior occurred in subjects both on trials without recorded responses and on trials after responses occurred on the key. It is therefore possible that in some instances the decrease in the tendency of subjects to respond as the CS progressed was a result of other behaviors directed away from the response key being controlled by the later portions of the CS.

The latency distributions also reflect on the concept of response strength (cf. Hull, 1952). Response-strength theorists rely on the covariation of different response measures to support their theories. It is obvious that since the latencies appear to be primarily under the control of CS duration, they do not covary with other measures of response strength that change as other parameters are manipulated. The latency data suggest that CS duration is an input into both the probability of responding on a particular trial and, if the animal does respond, into when the first response will occur. Variations in the IRI and TI durations also affect the probability of responding but do not appear to exert a large influence on when the first response occurs during the CS presentation.

Transfer Effects from Phase I to Phase II

The results of Phase II generally replicated the results of Phase I. Those subjects exposed to the 80-12-4 procedure pecked sooner and at higher rates than those subjects exposed to the 80-4-12 procedure. There were large differences, however, in the performance of subjects depending on what their Phase I histories were. The subjects that had been in groups 16-8-8, 16-4-28, and 224-4-28 tended to peck sooner, on more trials, and at higher overall rates than those subjects previously exposed to conditions 32-32-32, 48-16-32, 48-8-40, 16-4-28, and 48-4-44. The subjects that had been in the former set of groups took fewer trials to the fifth response than naive subjects exposed to comparable conditions, and the subjects in the latter set of groups took more trials to reach this criterion than did naive subjects. These transfer effects are related to the previously described metric, λ .

Figure 13 shows the difference between the median number of trials to the fifth response in Phase II and Phase I as a function of the Phase I λ for all the experimental groups. Two points are shown for each group. The open points are the statistics of those subjects exposed to the 80-12-4 condition in Phase II and the closed points are the statistics associated with subjects exposed to the 80-4-12 condition. Difference scores below zero indicate facilitation of Phase II acquisition and positive difference scores indicate retardation of acquisition. Phase II responding was facilitated in all groups previously exposed to treatments which yielded λ values less than or equal to 1.00. Phase II acquisition is inhibited in groups exposed to experimental treatments that yield λ values greater than or equal to 1.33.

Figure 14 shows the difference between median number of trials with at least one response in comparable Phase II and Phase I groups as a function of Phase I λ for all the Phase II groups. Facilitation of Phase II responding is evidenced by positive difference scores in many of the experimental groups. In general, facilitation is evident in all groups exposed to Phase I procedures associated with λ values less than or equal to 1.0. Additionally, performance in groups 32-8-56 and 16-4-28 is facilitated. It appears coincidental that both these groups have λ values equal to 5.33. There is substantial variability in this measure not accounted for by the manipulation of temporal parameters in the Phase II results, as was the case during Phase I. The low level of trials with at least one response in groups 80-12-4 and 80-4-12 during Phase I makes Phase II negative transfer effects difficult to assess.

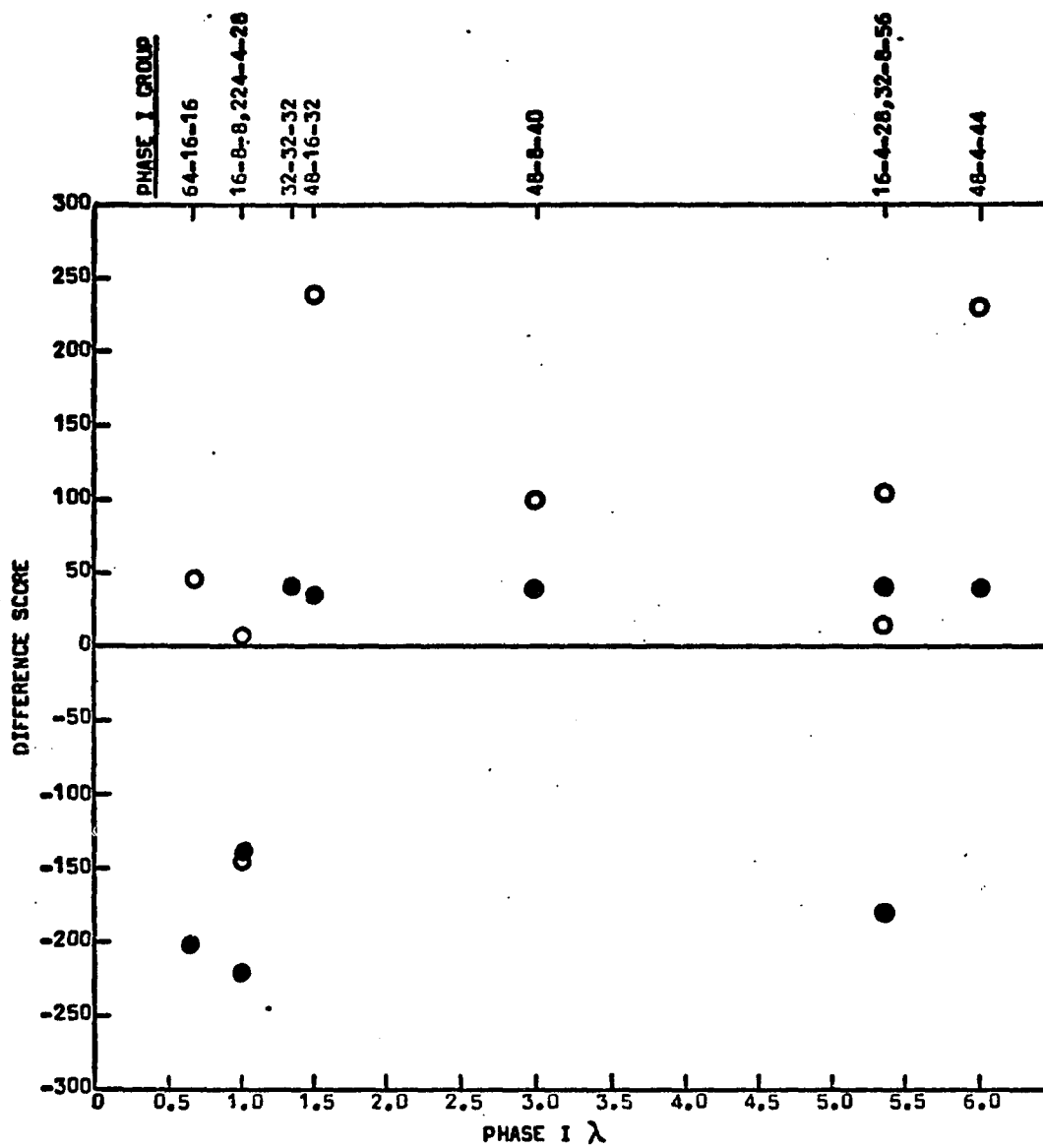


Figure 13. The difference between the phase II median number of trials to fifth response and the comparable phase I median. These difference scores are plotted as a function of the phase I λ .

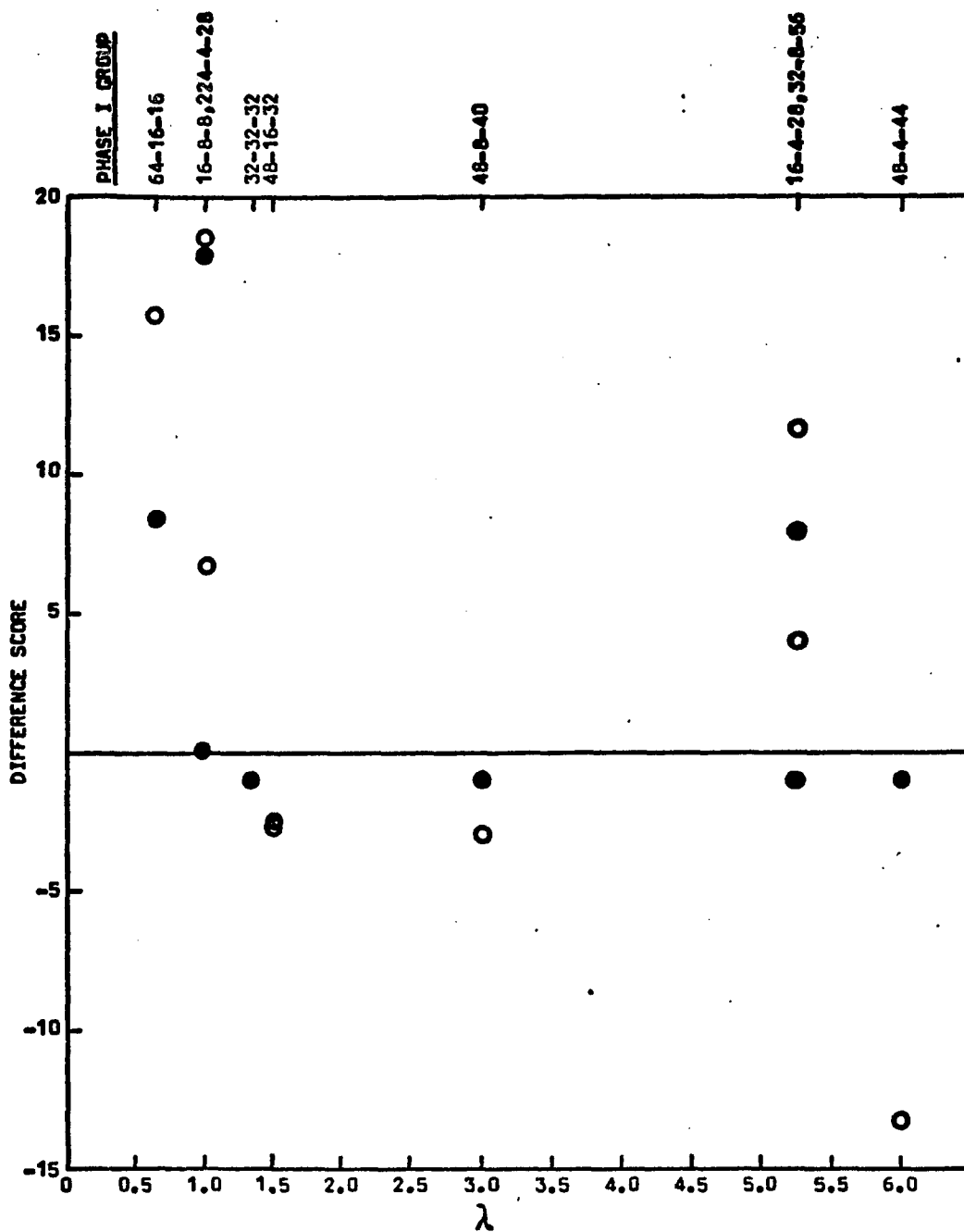


Figure 14. The difference between the median number of trials with at least one response during the final 125 trials of phase II and the comparable phase I median. These difference scores are shown as a function of phase I λ .

Figure 15 depicts the difference between the median overall rate of responding during the last 125 trials of Phase II and Phase I as a function of Phase I λ . These data show a pattern of results similar to that seen in the preceding figure. The response rate of all those subjects exposed to Phase I procedures associated with λ values less than or equal to 1 are above those of the comparable Phase I subjects. The performance of subjects in groups 16-4-28 and 32-8-56 is also above the comparable Phase I medians.

These data are not entirely consistent with several recently proposed models of conditioning. Rescorla (1967) has suggested that it is the contingency between the CS and US that is the necessary relationship between the two stimuli that determines the sort of control the CS exerts over responding. If the conditional probability of the US, given the CS, is greater than the conditional probability of the US in the absence of the CS, then the CS should become a positive conditioned stimulus. If the probability of the US is greater in the absence of the CS than in its presence, the CS should become a conditioned inhibitor. This definition implies that all trace procedures are formally inhibitory procedures. It would, therefore, be expected that, for all of the subjects exposed to Phase II procedures in the current experiment, the CS should have been a conditioned inhibitor of responding at the end of Phase I.

If excitation and inhibition are assumed to be algebraically additive, then facilitation or retardation of the acquisition of pecking in Phase II may be taken as evidence of the prior associative control of the CS. If Phase II acquisition is retarded, then a particular Phase I

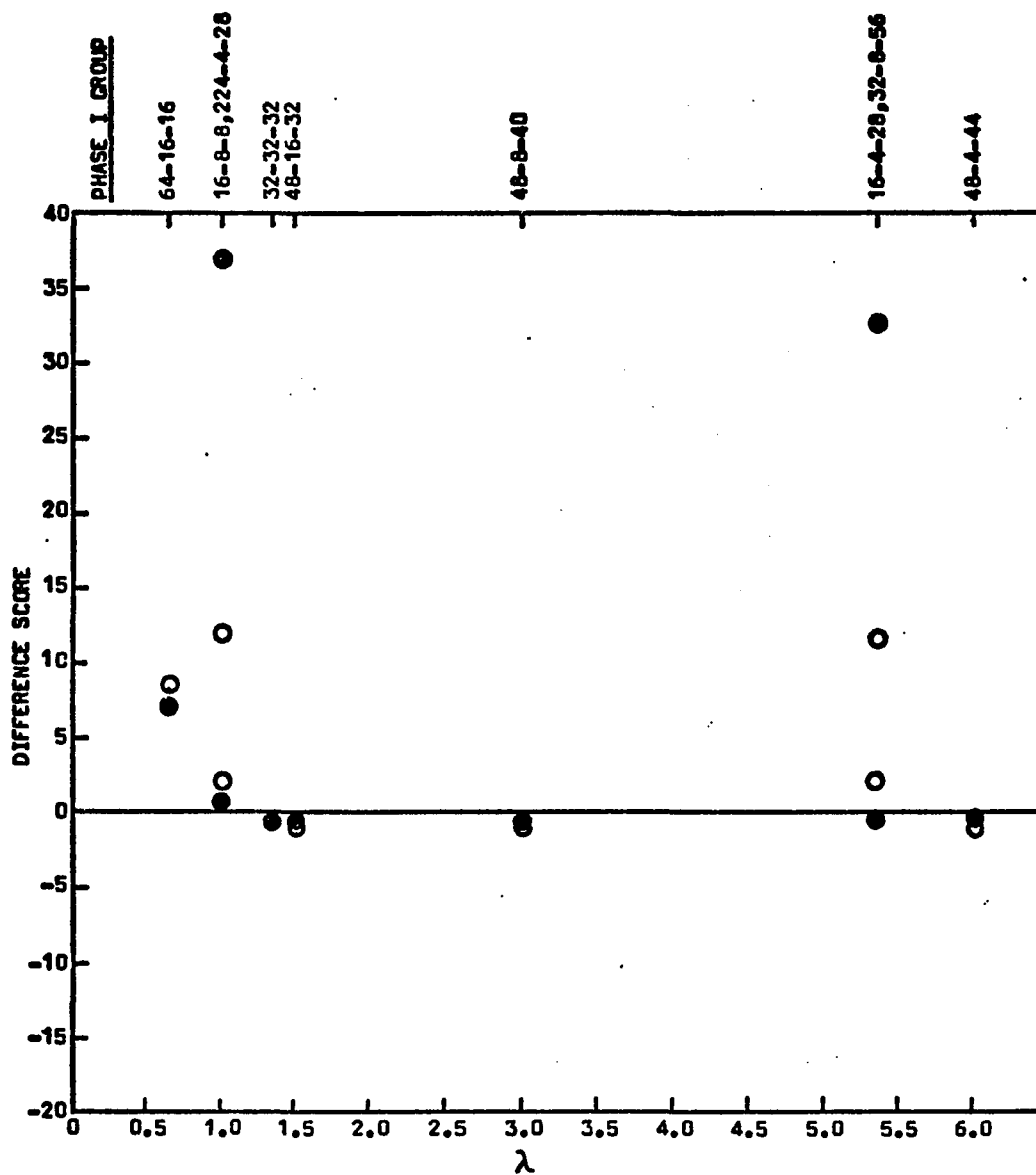


Figure 15. The difference between the median overall rate of responding during the last 125 trials of phase II and the comparable phase I median. The difference scores are shown as a function of phase I λ .

history must have established the CS as a conditioned inhibitor. If Phase II acquisition is facilitated, then the Phase I CS must have been a positive conditioned stimulus.

Obviously, not all of the subjects in Phase II behaved as though the CS had become an inhibitory stimulus. In fact, many subjects showed facilitation of Phase II responding. The facilitation of acquisition was, furthermore, related to all three temporal parameters manipulated in the current study. This fact is indicated by the relationship between the λ values that were associated with Phase I procedures and subsequent Phase II performance. Those subjects exposed to Phase I procedures with small λ values generally pecked sooner during Phase II than those subjects exposed to procedures associated with large λ values during the first phase of the experiment.

More recent contingency models proposed by Rescorla and Wagner (1972) and Gibbon, Berryman, and Thompson (1974) predict some of the effects of varying the duration of the different stimuli in the conditioning situation. These models predict different levels of inhibitory control established in the different Phase I procedures, but in no case do they predict the Phase II facilitation observed in some of the subjects.

Perhaps the comparison of Phase I and Phase II acquisition is not an appropriate one in assessing the associative control exerted by the Phase I CS, inasmuch as the subjects were in the experiment for different lengths of time. It is possible that there is no facilitation and that the Phase II data are evidence of different initial levels of inhibitory control. The contingency models predict decreasing inhibitory

control with smaller CS/IRI ratios, thus, within an IRI, the longer the CS duration the greater the inhibitory control and, with equal CS durations, lessening inhibitory control with longer IRIs. It may additionally be assumed for the Rescorla and Wagner model to make these predictions in a second way that subjects had not yet reached asymptotic levels of performance, since this model predicts that subjects exposed to longer CS durations should reach asymptote sooner.

Even when all these assumptions are granted, the data do not consistently confirm the predictions of the contingency models. The predicted ordering of groups from the least inhibitory to the most in Phase II groups at the 96-sec IRI with respect to the ratio of CS to IRI is $48-4-44 < 32-8-56 = 48-8-40 < 48-16-32 = 64-16-16 < 32-32-32$. The obtained ordering of groups was $64-16-16 < 32-8-56 < 48-8-40 < 48-4-44 < 48-16-32 = 32-32-32$. It is obvious that the predictions are not confirmed. The predictions made on the basis of increasing IRI are also not consistently confirmed. Although group 224-4-28 took fewer trials to criterion than any other 4-sec CS group, group 16-8-8 took fewer trials to criterion than any other 8-sec group.

In conclusion, the Phase II data are not accurately described by contingency models. Some model that is based on the temporal locus and duration of the CS during the IRI rather than the contingency between CS and US seems necessary for a description of the Phase II data.

Models of Temporal Effects

A complete model of the acquisition and maintenance of key pecking must take into account the durations of the IRI, CS, and TI. The earliest accounts of the effects of manipulating temporal parameters

were based on neural mechanisms that were hypothesized to exist within the central nervous system. Pavlov (1927) thought it essential to establish a CS with a short ISI (5-sec) before attempting to maintain one at longer ISIs. He found that subjects often responded during the TI and to account for this behavior he assumed that the effective stimulus in conditioning was the neural aftereffect of the external stimulus. At long intervals from CS offset to US onset, the neural trace would be weak and thus the speed and level of conditioning should decrease with increasing ISIs or increasing TIs within an ISI. Pavlov also did one of the earliest studies on the effects of varying the ITI. He reported that it took longer to extinguish a CR, the longer the training ITI had been (Pavlov, 1927). Gormezano and Moore (1969), however, have pointed out that there is little difference between the various ITI conditions in the total trials to extinction. In any event, Pavlov did not suggest the underlying reason for an IRI effect, and it was not until much later that other theoretical accounts attempted to deal with these effects.

Some of the earliest accounts of the effects of varying temporal parameters rely on time dependent processes to account for the facilitated acquisition with increasing IRIs. Hull (1952) assumed that reactive inhibition dissipated with longer IRIs and thus accounted for the facilitated performance. It would have to be assumed, though, that reactive inhibition would play a major role over the range of IRIs from 10 to 960 seconds employed in the current study or those of previous studies (Groves, 1974; Baldock, 1974; Terrace, Gibbon, Farrell, and Baldock, 1975; Griffin, 1975).

Estes (1959) assumed an increasing negative correlation between the stimulus elements sampled on successive trials as the time between trials increases. This assumption accounts for the effect of increasing the IRI and increasing CS duration within an IRI in delay procedures. This model, however, does not predict the effects of varying the temporal parameters in trace procedures. It does not predict the decreased responding with increased TIs for a particular CS duration and, contrary to the data, it predicts decreased responding with increases in CS duration within an ISI.

More recently, contingency models of conditioning have been proposed that account for some of the effects of varying temporal parameters. The model proposed by Rescorla and Wagner (1972) predicts an inverse relationship between the speed of acquisition and the IRI duration. This model treats the ITI as a background stimulus (A) and the CS as a compound (AX) formed by the ITI stimuli plus the CS stimulus change. Increasing the IRI increases the number of nonreinforced A-trials. The more nonreinforced A-alone trials, the faster the AX compound is incremented when it is reinforced. Thus, the longer the IRI, the faster acquisition should be. This model, however, does not predict asymptotic differences as a function of IRI. According to this model, variations in CS duration in a procedure in which there is a single US presentation during each trial, such as the one employed in the current study, should not affect either the speed of acquisition or the asymptotic level of performance. In order to deal with these effects, additional assumptions about trial size must be made. For example, if trial size is taken as equal to CS duration, when the IRI

is held constant, the number of nonreinforced A-trials decreases as CS duration increases. Retarded acquisition with increasing CS durations would, therefore, be predicted. The next model discussed makes this and other additional assumptions and will be discussed later. The Rescorla and Wagner model, however, does not predict the obtained effect produced by varying CS duration with an ISI. That model, furthermore, does not predict the effects of varying the trace interval. In the trace procedure, the ITI stimulus becomes the reinforced stimulus and thus the AX compound is never reinforced. Thus decrements in acquisition are not predicted as a function of either increasing the TI within an ISI or increasing the TI for a given CS duration within or across IRIs. In general, therefore, the data obtained in the current study are not adequately handled by the contingency model proposed by Rescorla and Wagner.

Gibbon, Berryman, and Thompson (1974) have proposed a contingency model that predicts more of the effects of varying temporal parameters than does the Rescorla and Wagner model. Unlike the latter model, however, the Gibbon et al. model does not deal with acquisition; it is only a model of asymptotic performance. One might assume, however, that whatever factors are responsible for the maintained performance levels combine in similar ways during acquisition and affect behavior similarly. The contingency metric, ϕ , that is proposed in this account changes as a function of the ratio of CS to ITI duration, being inversely related to that ratio. If it is assumed that trial size is equal to the shortest stimulus duration a subject is exposed to, decreases in CS duration or increases in the IRI should result in increases in responding. This

prediction is consistent with the empirical results. There is, however, no change in ϕ as a function of increasing the time from CS offset to US onset for a given CS duration in trace procedures, and it changes in the wrong direction by becoming less negative as CS duration is decreased within a fixed ISI.

The data collected in the present study indicate that the previously discussed models are inadequate in their ability to account for the effects of varying the temporal parameters of conditioning. A model that predicts the effects of varying the IRI, CS duration, ISI, and TI is currently lacking. The effects of varying the trace interval, in particular, suggest that a successful model of conditioning must take into account either the durations or time of occurrence of the various stimuli.

The variable λ , which is formed by dividing the ratio of ISI to IRI by the ratio of CS to ISI stands in a fairly orderly relationship to the data obtained in the present and previous studies. This fact suggests that the relative time to reinforcement signalled by CS onset is one primary determinant of the level of responding that comes to be controlled by the CS. This quantity is weighted by the proximity of the time from CS onset to offset relative to the remaining time until the US presentation. Thus any model that is developed must have as its parameters the time from one US (t_0) to the next US (t_1), the time from US to CS onset (t_1), and the time from CS onset to CS offset (t_2). A model of conditioning based on these parameters can be developed with comparatively few assumptions. The first assumption of the model is that the associative strength that will accrue to a stimulus increases

exponentially as a function of time during the IRI. The second assumption is that responding will be some function of the average associative strength during the CS. The third assumption is that responding is also a function of the relative associative strengths of different stimuli during the IRI. Lastly, it must be assumed that the programmed "clock time" may not be the "phenomenal time" experienced by the experimental subjects. The basic model is developed on the basis of the first three assumptions, and the efficacy of the third assumption is demonstrated in the data.

Equation 1 embodies the above assumptions, in defining a function T:

$$T = \frac{\sum_{1}^n \frac{\int_{t_1}^{t_2} t dt / t_2 - t_1}{\int_{t_0}^{t_I} t dt / t_I - t_0}}{n} \quad (\text{EQ 1})$$

Where $t_0 = 0$, t_1 is the time from t_0 to CS onset, t_2 is the time from t_0 to CS offset, t_I is the time from t_0 to the next US, and n is the number of trials. Equation 1 reduces¹ to the computational formula shown in Equation 2.

$$T = \frac{\sum_{1}^n \frac{t_1 + t_2}{t_I}}{n} \quad (\text{EQ 2})$$

T changes in appropriate directions as a result of varying temporal parameters. It increases exponentially when t_I (IRI) is increased and

1

$$T = \frac{\frac{\int_{t_1}^{t_2} t^2 / t_2 - t_1}{t_1}}{\frac{\int_{t_0}^{t_I} t^2 / t_I - t_0}{t_0}} = \frac{t_2^2 - t_1^2 / t_2 - t_1}{t_I^2 - t_0^2 / t_I - t_0} = \frac{t_2 + t_1}{t_I}$$

$t_2 - t_1$ (CS duration) held constant. It decreases linearly as t_1 decreases (CS duration increases) and decreases linearly as a function of decreasing t_2 (increasing the TI); furthermore, T decreases more quickly as t_1 and t_2 decrease even when $t_1 - t_2$ is held constant (increasing the TI with a constant CS duration).

Figure 16 shows the median number of trials to the fifth response as a function of T . It can be seen that although the general predictions of the model are obtained when any of the manipulations discussed above are carried out, there is not a completely monotonic relationship between the trial of the fifth peck and T . The large intersubject variability makes the adequacy of the model difficult to assess but perhaps some of the failure of the model may be attributed to the use of "clock time" to compute the values of T .

It is a well-established generalization that there is not a linear relationship between "clock time" and the pigeons estimation of that time as indicated by a variety of behavioral measures. Catania (1970) has demonstrated the relation between the programmed time and the pigeon's estimation of time as measured by mean latencies on discrete trial DRL schedules. He found that the data were well described by the function $T = Kt^n$; where T is the average latency of responding, t is the scheduled DRL requirement, and K and n are constants. Catania, furthermore, found that the value of the exponent, n , increased as the ITI decreased over a range from 20 to .2 seconds. In no instance, however, did n exceed 1.0. The best fit of the pooled data yielded values for K and n of 1.6 and .8 respectively at the 20-sec ITI.

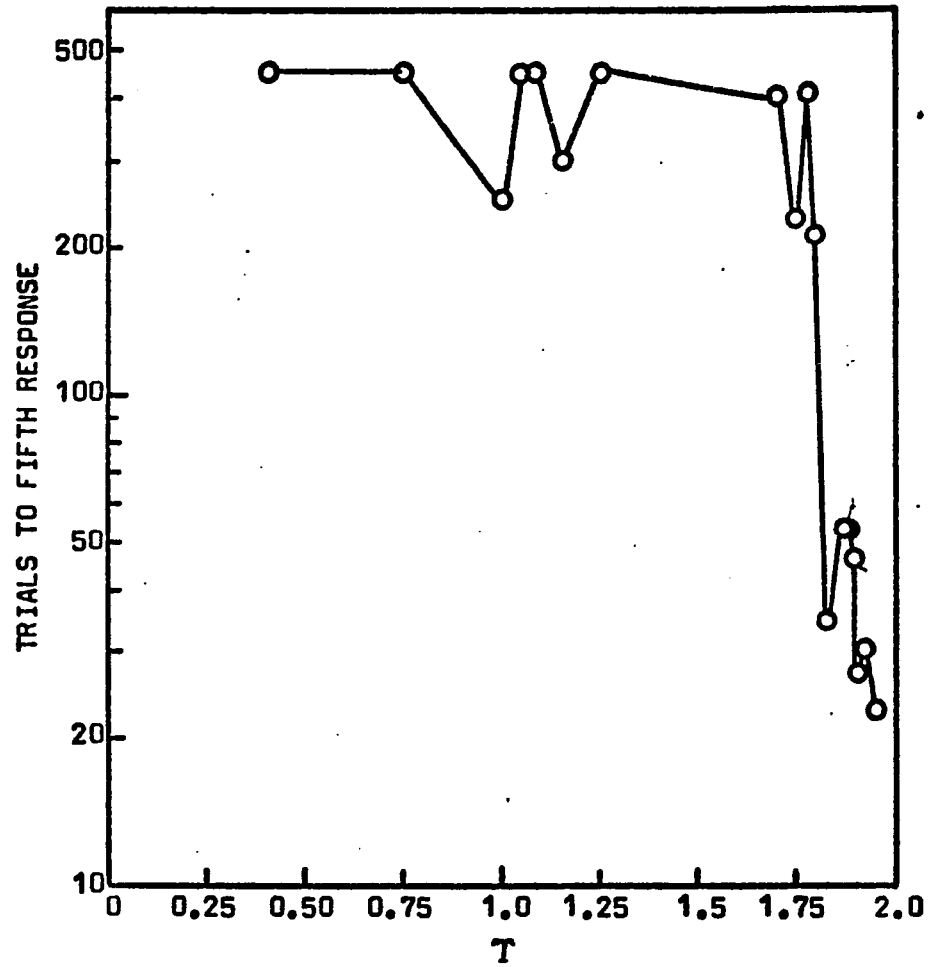


Figure 16. The median trial of the fifth response is shown as a function of T . (see text)

There is no reason to believe that the subjects in the current study "timed" any differently than did Catania's DRL subjects. Thus, it is perhaps inappropriate to try to predict a subject's behavior without doing some power function transformation on any temporal parameters that enter into a model. Such a transformation could obviously be carried out at various stages of a model. In the current case, it was decided to perform the transformation at the level at which the subject sequentially experiences the stimulus changes. The value of n was chosen according to the estimate from Catania's study. It is possible that this value overestimates the birds timing, the IRI (ITI) values employed in the present study being greater than that employed in the Catania (1970) experiments. Without the appropriate data from which to derive the exponent value, the use of .8 as the value of n seems the most justifiable thing to do at this time.

The parameter values used to compute T were translated into "bird time" by raising each "clock time" to the .8 power; T was then recomputed on the basis of the new times. Figure 17 shows the median number of trials to the fifth peck as a function of these new values of T . A relationship between T and the number of trials to the fifth peck exists in this figure that is similar to the relation depicted in the preceding one. Figure 18 shows the median overall rate of responding plus one as a function of T . The response rate rises rapidly over a small range of T values above 1.8. Thus, increasing rates generally track increases in T . The acquisition data from Phase II of the experiment have been plotted as a function of Phase I T in Figure 19. The open points represent those subjects exposed to the 80-12-4 condition and the closed

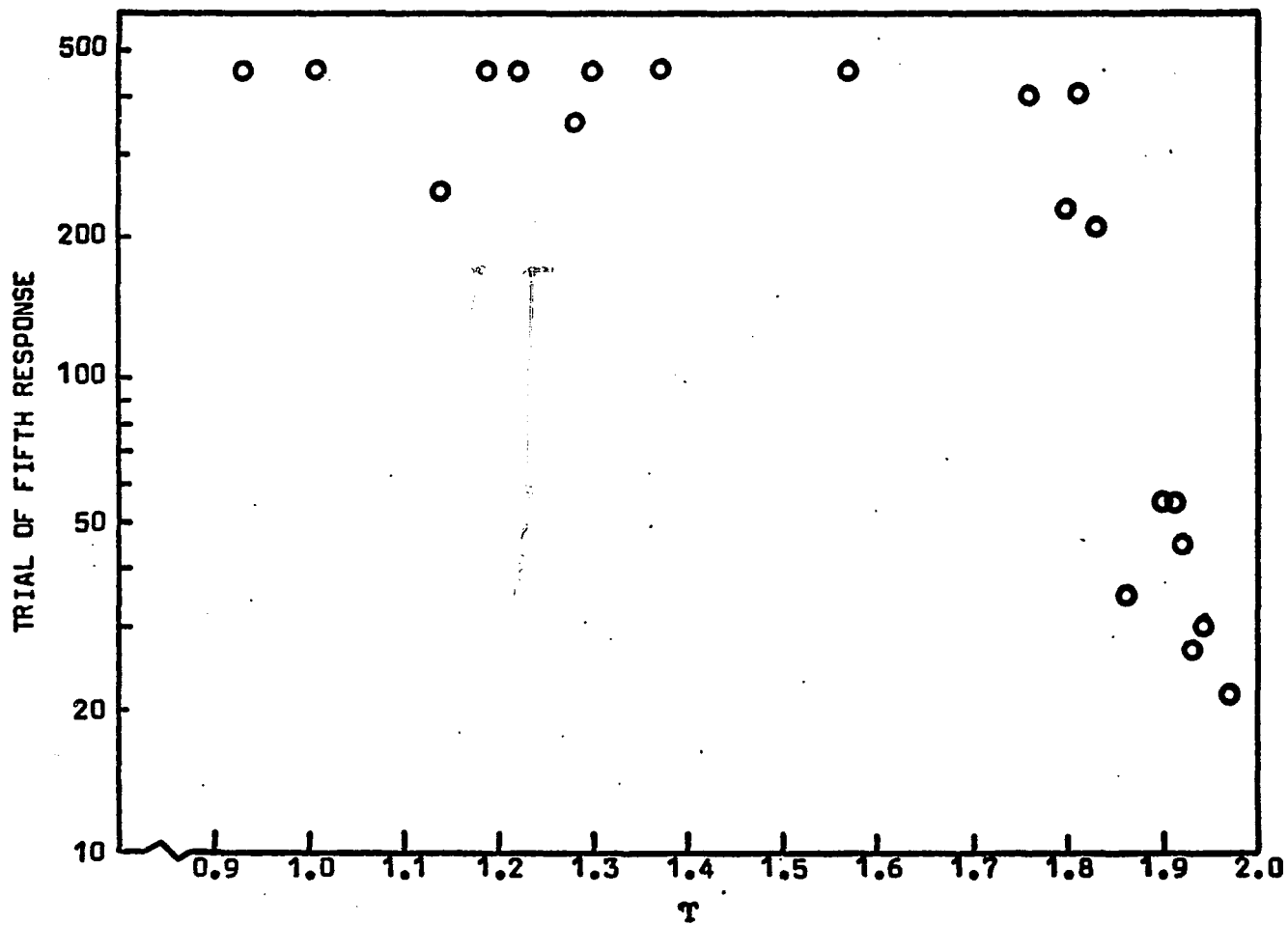


Figure 17. The median trial of the fifth response is shown as a function of T , computed using a power transformation on parameter values.

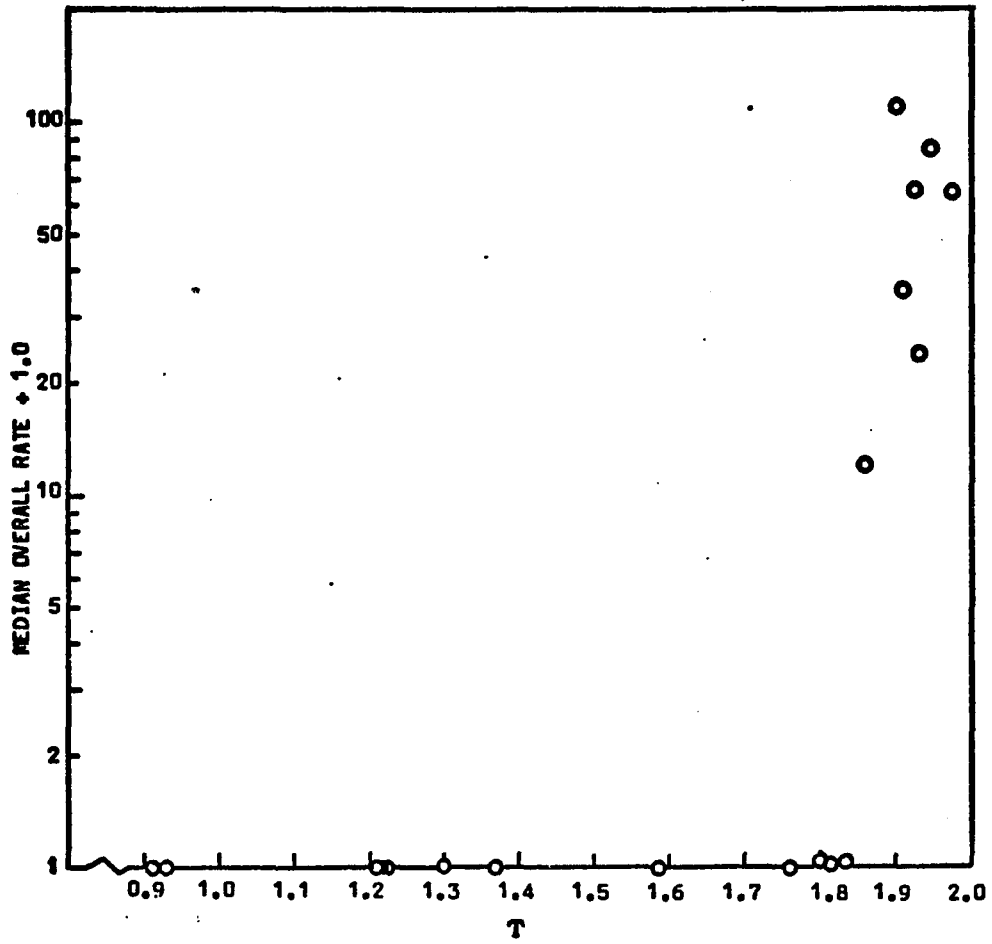


Figure 18. The overall rate of responding is shown as a function of T . The T values were computed on transformed parameter values.

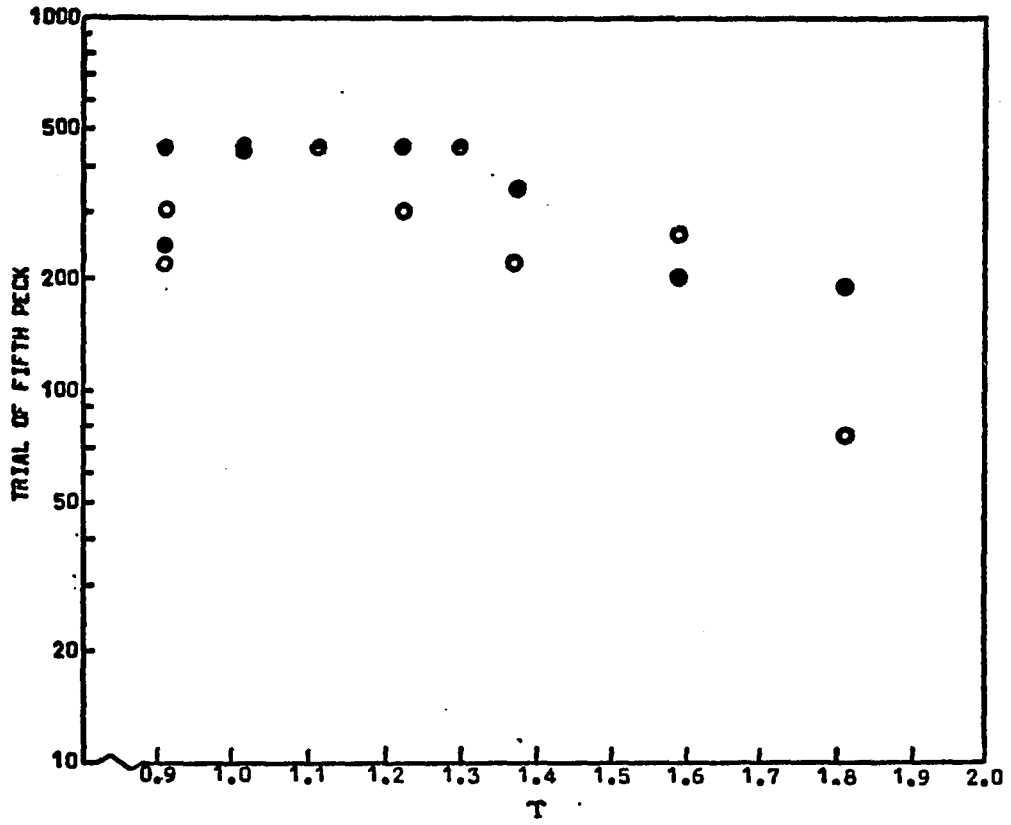


Figure 19. The fifth trial with a response during phase II is shown as a function of phase I T. The T values were computed using transformed parameter values.

points represent those subjects exposed to the 80-4-12 condition. These data show that generally the number of trials to the fifth response decreases as T increases; a finding which replicates the Phase I results. In general, those subjects exposed to Phase I procedures associated with T values greater than 1.3 show facilitation of Phase II acquisition. Those subjects exposed to Phase I procedures associated with T values of less than or equal to 1.3, generally, show evidence of inhibitory control by the CS in Phase I. Both the Phase I and Phase II results are, therefore, fairly well predicted by the model of conditioning proposed here. The stratagem of computing intervals on the basis of "phenomenal time" therefore seems justified.

The model proposed here seems to do an adequate job of summarizing the effects of varying the temporal parameters of conditioning. The exponent used to estimate the parameters that determine T would probably vary from one species to another, but the general model seems fairly effective in its predictions of the data collected in the current study. More extensive research looking at a larger number of IRIs and CS durations in pigeons as well as parametric work with other species seems warranted by the current results.

SUMMARY

When the illumination of a response key is followed by grain presentation, pigeons come to peck at the lighted key. Stimulus-reinforcer relationships in this procedure have been shown to exert a strong influence on the development and maintenance of responding. The control exerted by stimulus-reinforcer relationships was investigated by exposing groups of pigeons to procedures that differed according to the duration of the various intervals defined by the stimulus changes in this procedure. In the first phase of the experiment, variations in the time from keylight offset to grain onset produced an inverse relationship between several measures of the tendency to respond and the duration of the trace interval. The tendency to respond decreased as the duration of the key illumination was increased and the tendency to respond decreased as the interreinforcement interval was shortened. The effects of these three manipulations were summarized by an inverse relationship between the tendency to respond and a variable λ . This variable is formed by dividing the duration of the interstimulus interval by the duration of the interreinforcement interval and multiplying this quantity by the quotient produced by dividing the duration of the interstimulus interval by the CS duration. The within-CS response patterns indicated that subjects tended to respond soon after CS onset or not at all. Those subjects that did not respond much during the first phase of the experiment were exposed to a second procedure. The results of the second phase replicated the findings of the earlier portion of the

experiment and, additionally, demonstrated that the transfer from Phase I to Phase II was related to the Phase I λ . Predictions based on recently proposed contingency models of conditioning were not entirely consistent with the results of both phases of the experiment. A model based solely on temporal parameters was developed and the predictions based on this model were shown to be in accord with the results of the experiment.

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