FOREPERIOD LENGTH, REACTION TIME AND AUTONOMIC ACTIVITY

by

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ABSTRACT

Four foreperiods, 6.1, 2.1, 1.1, and 0.6 seconds, were used to investigate the relationships between some components of the OR to a warning signal and reaction time to a stimulus. It was found that reaction time was slowest with the longest foreperiod and fastest with the shortest foreperiod. Duration of the components of the OR correlated negatively with reaction time, and no correlation was found between reaction time and heart-rate deceleration. Heart-rate deceleration was found to be maximal during the 6.1 second foreperiod and to correlate positively with the duration of the deceleration. The findings are discussed in terms of a central process, and the implications for individual learning differences are discussed in terms of the response requirements of the task.

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INTRODUCTION

Russian research has long been concerned with the investigation of the orienting reaction (OR) of an animal to novel and/or meaningful stimuli (Anokhin, 1958). Usually only the peripheral components of the OR are studied; however, it is assumed that these reflect the activity of the central nervous system. Sokolov (1963), for example, believes that the OR reflects the process of matching incoming information with information that the animal has already stored. Because it is considered an holistic response (Anokhin, 1958), the OR is thought to inhibit all other activities of the organism, except when it occurs during the course of some activity which is itself connected with the stimulus which elicited the OR. In this latter case excitation of the ongoing response occurs.

Some of the peripheral components of the OR are:

1) an increase in skin conductance
2) peripheral vasoconstriction
3) cephalic vasodilation
4) pupillary dilation
5) heart-rate deceleration
6) increased muscle tension (specific and/or general).

More central components are hippocampal theta activity, and alpha-blocking (Graham and Clifton, 1966; Grastyan, et al., 1959; Lynn, 1966; Razran, 1961; Sokolov, 1963).

The OR lowers detection thresholds with the effect of amplifying weak stimuli, in direct contrast to the defensive reaction (DR) which raises detection thresholds (Lacey and Lacey, 1958, 1964; Lykken, 1968; Sokolov,

1963). Lacey and Lacey (1964, 1965) consider heart-rate deceleration as reflecting the organism's "acceptance" of the external environment, and acceleration, a defensive reaction component, as "rejection." In terms of their model, only biphasic responses would, therefore, be useful in distinguishing between an OR and a defensive reflex. Such responses would be those mediated by both parasympathetic and sympathetic activity. One such biphasic response is that of the pupil of the eye.

Research on pupil dilation suggests that the magnitude of pupillary dilation reflects task difficulty for the individual (Hess and Polt, 1964; Kahneman and Beatty, 1966; Paivio and Simpson, 1966; Simpson and Paivio, 1966). Hess and Polt (1964) presented data which showed that the pupil did not return to prestimulus level until the solution to an arithmetic task had been verbalized, although there appeared to be some constriction prior to verbalization. Holmes (1967) presented evidence that the speed of pupillary constriction, a parasympathetic response, correlated positively with the speed at which a subject learned in a verbal conditioning paradigm, and with the subject's awareness of environmental contingencies. Holmes' (1967) findings and those of Hess and Polt (1964) suggest the possibility that active inhibition of an OR might relate to an individual's "conditionability" and the speed with which he processes incoming information.

The OR and its components have often been used to operationally define anxiety. Hare (1968) has reviewed studies that suggest that, in psychopaths, in whom avoidance conditioning is impaired, the OR or conditioned galvanic skin response is of relatively short duration, i.e., it has a fast recovery time. Hare (1968) was unable to replicate these findings, although his "secondary or neurotic psychopaths" showed a slower recovery time than either

the "primary" or non-psychopathic groups. However, Hare (1968) also noted that the psychopathic group manifested slower habituation of responses with both sympathetic and parasympathetic mediation. Holmes' (1967) "rapid constrictors" were unlike Hare's psychopaths, since Holmes' subjects also manifested rapid habituation of the response. This raises some interesting questions concerning the OR.

Grastyan, et al. (1959), Sokolov (1963), and Vinogradova (1958) all mention that habituation of the OR occurs with repeated stimulation. They also speak of habituation of the OR as a conditioned reflex is formed. Gale and Ax (1968) report that habituation of the galvanic skin response and peripheral vasomotor response does not occur in a differential conditioning paradigm to either the CS^+ or CS^-. Grastyan, et al. (1959) report that the OR reoccurs during extinction, where uncertainty reoccurs. When, however, habituation does occur, such habituation is viewed by Vinogradova (1958) and Sokolov (1963) as the cortical control of the OR and presumably occurs when the incoming stimulus matches the stored information and is quickly processed. The mechanism for such rapid processing is not mentioned in Sokolov (1963); however, Lykken (1968) has proposed a theory of "preception" which involves individual differences in the ability to attenuate afferent stimulation in paradigms involving a warning signal, i.e., where predictability of a stimulus is high.

Lykken (1968) suggested that the ability to maintain "anticipatory afferent tuning" involves effort and that it may be fatiguing if it must be maintained for a long time. If the OR is seen to habituate rapidly in some subjects, it might then be viewed as reflecting the fact that such subjects

find the stimulus highly predictable. However, in view of Holmes' (1967) work and Lykken's (1965) theory it would appear that a measure other than habituation of the OR is related to an individual's "conditionability." Such a measure is the duration of one OR. It might be expected that some subjects cannot vary the duration of the OR; however, if cortical control is "normal" it is expected that the duration can be varied, within certain limits. [If, for example, a signal precedes a stimulus by too long a time interval, fatigue may interfere with vigilance (Lykken, 1968).]

Lykken (1968) and Horn and Venables (1964) have provided data on the effect of a warning signal on reaction time and the two-flash-threshold (TFT). Horn and Venables report an interval between a signal and stimulus of 300 to 600 msecs. as being optimal in the lowering of the TFT. Lykken (1968) found that a warning signal sufficiently far in advance of the stimulus to produce alpha-blocking (100-300 msecs.) also lowered reaction time. It is possible that, with longer intervals, some of the variability in simple reaction time is due to differences in the duration of the OR. This is illustrated schematically in Figure 1.

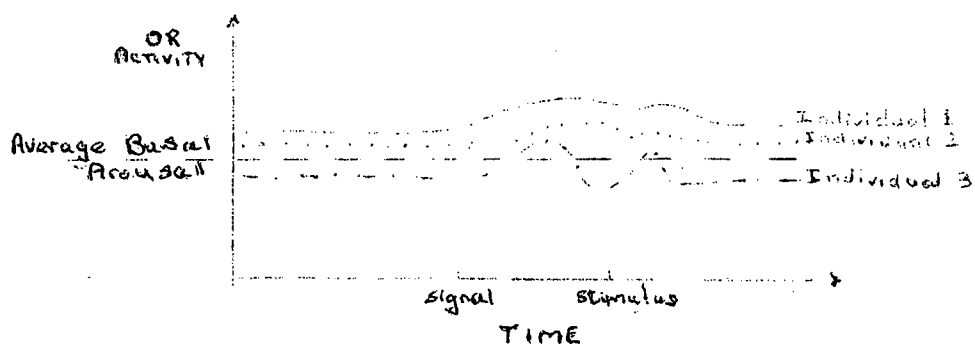

Figure 1: Hypothesized reactions to a warning signal. Individual one has a short-latency, long-lived OR. Individual two has a long-latency OR, and Individual three, a short-latency, short-lived OR.

According to Anokhin's (1958) postulate that the OR is holistic, Individual 1 (Figure 1) would have a longer reaction time than Individual 3. There are only two possible situations in which this would not be the case. The first situation would be that in which the motor response had already started when the OR occurred, as might be expected in short signal-stimulus intervals, and the second, that in which the signal has already been conditioned as the CS in an avoidance or escape learning paradigm. Given that neither of these two situations hold in a reaction time experiment with a fairly long signal-stimulus interval (foreperiod), the prediction is made that reaction time is faster when the duration of the OR is short, and its magnitude is small.

Coquery and Lacey (1966) and Lacey and Lacey (1965) investigated the relationship between signal-initiated cardiac deceleration and reaction time to a stimulus. Their theory suggested that reaction time should be faster when the stimulus, to which the response must be made, is facilitated by heart-rate deceleration. Thus, they predicted a positive correlation between reaction time and heart-rate deceleration. Most of their data appeared to support their prediction. However, Chase, Graham, and Graham (1968) disagreed with the Lacey's (1965) interpretations. Lacey and Lacey (1965) had reported a polyphasic cardiac response consisting of an early deceleration, followed by acceleration, followed by deceleration which peaked at the time of the stimulus. They noted that the pattern of the response changed with foreperiod length, with the early acceleratory limb sacrificed during the shorter foreperiods. Chase et al. (1968) point out that both their own findings of a second declerative phase, and the ones reported by Lacey and Lacey (1965) should be viewed as conditioned anticipatory responses and not

ORs, since they were clearly not extensions of the deceleration following the signal. If this is so, then the first deceleration noted by both Chase et al. (1968) and Lacey and Lacey (1965) might be viewed as an OR which varied in length depending on the length of the foreperiod.

Using a non-continuous signal, it was decided to test the hypothesis that the duration of an OR could be varied by varying foreperiods. Some specific predictions were

1) Duration, as defined by the length of time from start to finish of the cardiovascular response, would be longest with the longest foreperiod, and shortest with the shortest. (If two responses appeared in the interval, only the first was to be scored.)

2) Maximal heart-rate deceleration would occur during the longest foreperiod, given that it was related to response duration.

3) Reaction time would be longest in the longest foreperiod, and fastest during the shortest one.

4) Reaction time would be longer the longer the duration of the response.

5) Increased skin conductance at the time of the stimulus, as compared with that prior to the signal, would occur when reaction times were slow.

METHOD

Subjects

The subjects (Ss) were nineteen male undergraduates and graduates at the University of British Columbia. However, due to equipment failures only fourteen were included in the analyses. The mean age of the Ss was twenty-four years. All were right-handed volunteers.

Apparatus

The stimuli used were two tones, 48 Hz. and 78 Hz. of .6 seconds duration. The tones were generated by two sine wave generators, and presented to the subject through stereo earphones. The loudness of each tone was judged to be subjectively equal and of moderate intensity by five observers and the intensity remained the same for each subject.

The response key was attached to a board on the right hand side of the armchair in which the Ss sat. The board could be adjusted so that each subject could comfortably reach the response key with his right index finger, and still have his arm supported. Reaction time (R.T.) was recorded by a timer accurate to .01 seconds. The timer was activated only by the high tone.

An Offner Type R Dynograph was used to measure respiration, heart-rate, palmar skin resistance, and cephalic and digital vasomotor responses. Skin resistance was determined by passing a 9 $\mu amp/cm^2$ current through Beckman Biopotential Electrodes attached to the second phalange of the first and third fingers of the left hand. The vasomotor responses were monitored by photocell transducers on the forehead, and on the palmar surface of the first phalange of the thumb on the left hand. Room light was prevented from interfering with the photocells by covering them with black cotton cloth after they

were attached to the subject. Respiration was measured by a strain gauge around the lower part of the S's rib cage. Heart-rate was recorded by two active electrodes placed on the midline of the chest, one above the heart and one on the sternum. A cardiotachometer converted the EKG signal to heart-rate (beats per minute). The S was grounded by an electrode on the left side of the rib cage under the left arm.

A Gerbrand's three-channel tape programmer, running at a speed of 4.75 mm/sec., was used to trigger the tones, the reaction timer, and a stimulus marker on the polygraph.

Procedure

The subject was seated in an armchair in a dimly lit, shielded, air-conditioned room. The temperature of the room averaged 71° and the humidity, 64%. The electrodes were attached to the subject, and the task was explained.

The Ss were told that they would hear a low tone (signal) followed by a high tone (stimulus). The signal was to be a warning that the high tone was going to come on. It was explained that the task was to press the response key as rapidly as possible, without anticipating, whenever the high tone came on. The Ss were also told that the interval between the low tone and high tone would vary, but, that, for each block of ten trials, the interval would remain constant. This last instruction, plus the use of a constant ten second intertrial interval and no "catch" trials was an attempt to eliminate any uncertainty due to stimulus conditions and its resultant effects on R.T. (Chase, et al., 1968; Klemmer, 1956).

After the instructions were given, the S was asked to relax while the experimenter calibrated the polygraph. The S was told that the first ten trials would be practice trials and that the experimenter would give him a

one minute warning before the trials began. About fifteen minutes after the electrodes had been attached the one minute warning was given.

Four foreperiods were used: .6 seconds, 1.1 seconds, 2.1 seconds, and 6.1 seconds. They were chosen because they were approximately equivalent to those used in other experiments (Lacey and Lacey, 1965; Hastings and Obrist, 1967). One-half of the Ss were run in Order 1. They had ten practice trials with the 2.1 second foreperiod, and were then tested on four blocks of ten trials each. The first block consisted of trials with a 6.1 second foreperiod, the second, of trials with a 1.1 second foreperiod, the third, of trials with a .6 second foreperiod, and the fourth, of trials with a 2.1 second foreperiod. The other half of the Ss were run in Order 2. They practiced with the 1.1 second foreperiod, and were tested with the trial blocks of foreperiods ordered .6, 2.1, 6.1, and 1.1 seconds. The experimental paradigm is described in Figure 2.

Order 1	Foreperiods			
Trials	6.1 secs. 1-10	1.1 secs. 1-10	.6 secs. 1-10	2.1 secs. 1-10
Ss 1 . . . 7				

Order 2	Foreperiods			
Trials	.6 secs. 1-10	2.1 secs. 1-10	6.1 secs. 1-10	1.1 secs. 1-10
Ss 8 . . . 14				

Figure 2: Experimental paradigm with length of foreperiods used, and the orders in which the trial blocks were presented.

Scoring Procedures

During the minute preceding the one minute warning, basal levels of autonomic activity were scored. These basal data included the mean heart-rate (HR) and its standard deviation; the respiration rate per minute and the variability, as estimated by ratings, of the respiration; the rated variability of the vasomotor responses; the mean skin conductance (SC) in μmhos; and the number of non-specific galvanic skin responses (NSP's) occurring in that minute.

The scoring of the responses during the experiment proper was done as follows. The proportion change in the size of the blood volume pulse was calculated for both the digital and cephalic vasomotor responses. This was done by comparing the size of the pulses during the five seconds preceding the signal with the size of the pulses during the five seconds after the signal. The presence of digital vasoconstriction after the signal was determined visually (see Figure 3) by an upward deflection of the pen and/or a reduction in pulse size. The opposite phenomena were used to determine the presence of cephalic vasodilation. The latency (time from signal to response beginning) and duration (time from beginning of the response to its end) were then measured. Heart-rate deceleration was calculated by subtracting the mean of the three lowest beats during the 7 seconds prior to the signal from the mean of the three lowest beats during the 7 seconds after the signal. The latency and duration of the cardiac deceleration was determined after visual inspection revealed a decrease in heart-rate following the signal, and duration was here defined as the time from the beginning of deceleration to the beginning of acceleration. The heart-rate at the time of the stimulus was scored, as was the skin conductance. The mean SC

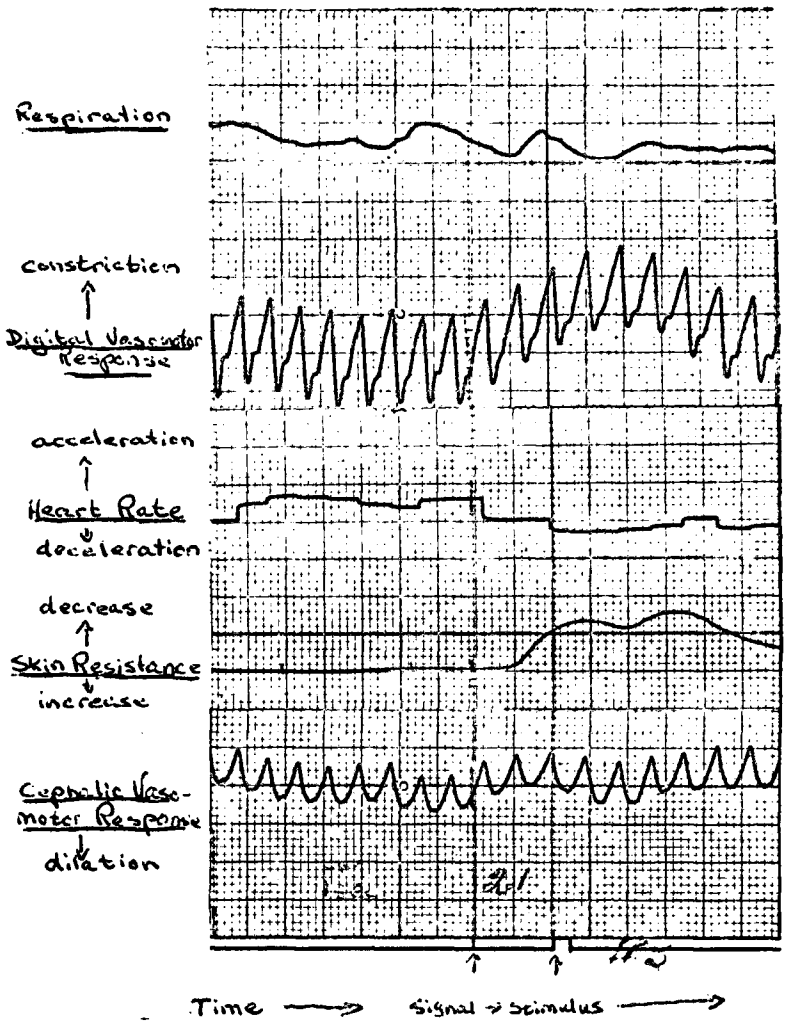

Figure 3: Components of the OR during the 2.1 second foreperiod (Trial 3). The respiration strain gauge was not functioning properly with this subject, however, digital vasoconstriction, heart-rate deceleration, a G.S.R., and what appears to be cephalic vasoconstriction followed by vasodilation are shown.

for the 5 seconds prior to the signal was also scored.

RESULTS

Preliminary analyses of variance on the basal data indicated no significant differences between Ss assigned to Order 1 and those assigned to Order 2. The mean HR for all Ss was 78.3 and the mean SC, 12.08 μmhos. Correlations on the basal data indicated significant correlations between responses reflecting sympathetic activity (see Table 1).

Basal Variables	1	2	3	4	5	6	7
1 Respiration Rate/minute	1.00						
2 Respiration Variability	-.72*	1.00					
3 Digital Vasomotor Variability	-.23	.49	1.00				
4 Heart-rate/minute	-.03	.06	.57*	1.00			
5 Cardiac Variability	-.33	.66*	.31	-.05	1.00		
6 Skin Conductance	-.41	.14	.20	.23	-.27	1.00	
7 Nonspecific GSR's	-.42	.48	.17	.19	.07	.35	1.00
8 Cephalic Vasomotor Variability	-.28	.28	.30	.42	.25	.56*	.54*

*$p \leq .05$, N=15

Table 1: Correlation Matrix on Basal Data

From Table 1 it may be seen that the higher the heart rate, the larger was the variability of the digital blood volume pulse, and the higher the SC, the higher the number of NSP's. It was noted further, that the more irregular the respiration, the larger the size of the standard deviation of the HR (cardiac variability), and the slower the respiration rate. No further analysis was conducted on the basal data.

Figure 4 shows reaction time as a function of length of foreperiod. As predicted, the longest foreperiod resulted in the longest reaction time, while the shortest foreperiod produced the shortest reaction time. The results of an analysis of variance on this data are presented in Table 2. (In order to

normalize the distribution of reaction time scores, the raw data were converted to Log_{10} for this analysis.) Neuman-Keuls tests indicated that R.T. was shortest during the .6 second foreperiod, next shortest during the 2.1

Source	DF	SS	MS	F	Prob.
Between Ss	13	5.6759	.4366	-	-
Order	1	.0849	.0849	n.s.	-
$Error_{(between)}$	12	5.5909	.4659	-	-
Within Ss	546	-	-	-	-
Foreperiods	3	2.7385	.9128	18.68*	.0000
Foreperiods x Order	3	.1733	.0578	n.s.	-
$Error_{F)}$	36	1.7596	.0489	-	-
Trials	9	.6079	.0675	3.60*	.0006
Trials x Order	9	.3013	.0335	n.s.	-
$Error_{(T)}$	108	2.0276	.0188	-	-
Foreperiods x Trials	27	1.3316	.0493	2.54*	.0001
Foreperiods x Trials x Order	27	.5573	.0206	n.s.	-
$Error_{(FT)}$	324	6.2868	.0194	-	-
Total	559	21.4600	-	-	-

Table 2: Analysis of Variance on the Converted Reaction Time Data

second foreperiod, and longest during the 1.1 and 6.1 second foreperiods. The differences were significant at the .05 level. Trials also had a significant effect, with reaction time shorter on the last trial than on the first, as might be expected from practice. A significant interaction between foreperiods and trials was more difficult to interpret. It would appear, however, that practice effects were small when the foreperiod was long and the variability, large (see Table 3).

Trials	.6 secs.	1.1 secs.	2.1 secs.	6.1 secs.
1	2.23	2.57	2.41	2.39
2	2.18	2.27	2.33	2.27
3	2.28	2.34	2.38	2.35
4	2.18	2.39	2.37	2.45
5	2.20	2.43	2.37	2.47
6	2.24	2.39	2.35	2.38
7	2.21	2.38	2.38	2.36
8	2.26	2.31	2.40	2.42
9	2.14	2.38	2.32	2.47
10	2.30	2.41	2.23	2.40

Table 3: Interaction Between Foreperiods, Trials and Log_{10} Reaction Time.

Analysis of variance on presignal SC revealed significant foreperiods and trials effects (see Table 4). The presignal SC was higher during the block of trials using the longest foreperiod. If one considers this measure to be indicative of arousal, then it would appear that the longest foreperiod produced a high level of arousal in subjects. Neuman-Keuls tests indicated that the highest level of SC occurred during the block of trials with the 1.1 second foreperiod, next during the block of trials with the 6.1 second foreperiod, and lowest during the 2.1 and .6 second foreperiods ($p \leq .05$).

Source	DF	SS	MS	F	Prob.
Between Ss	13	222.43	1711.0	-	-
Order	1	103.25	103.25	n.s.	-
$Error_{(between)}$	12	221.40	1845.0	-	-
Within Ss	546	-	-	-	-
Foreperiods	3	548.92	182.97	5.74*	.0027
Foreperiods x Order	3	55.495	18.50	n.s.	-
$Error_{(F)}$	36	1147.6	31.88	-	-
Trials	9	23.116	2.57	2.22*	.0257
Trials x Order	9	19.104	2.12	n.s.	
$Error_{(T)}$	108	124.79	1.16	-	-
Foreperiods x Trials	27	42.831	1.59	n.s.	-
Foreperiods x Trials x Order	27	46.496	1.72	n.s.	-
$Error_{(FT)}$	324	857.39	2.65	-	-
Total	559	25.109	-	-	-

Table 4: Analysis of Variance on Presignal Skin Conductance

The SC at the time of the stimulus was also significantly affected by foreperiod as indicated by Table 5. Neuman-Keuls tests indicated that the highest level of SC at the time of the signal was during the 1.1 and 6.1 second foreperiods, with no difference between the two, and lowest during the 2.1 and .6 second foreperiods ($p \leq .05$). (Very few subjects gave a galvanic skin response (GSR) to the signal as well as to the stimulus, so it was decided to omit this usual measure and to look only at the more tonic changes in skin resistance.)

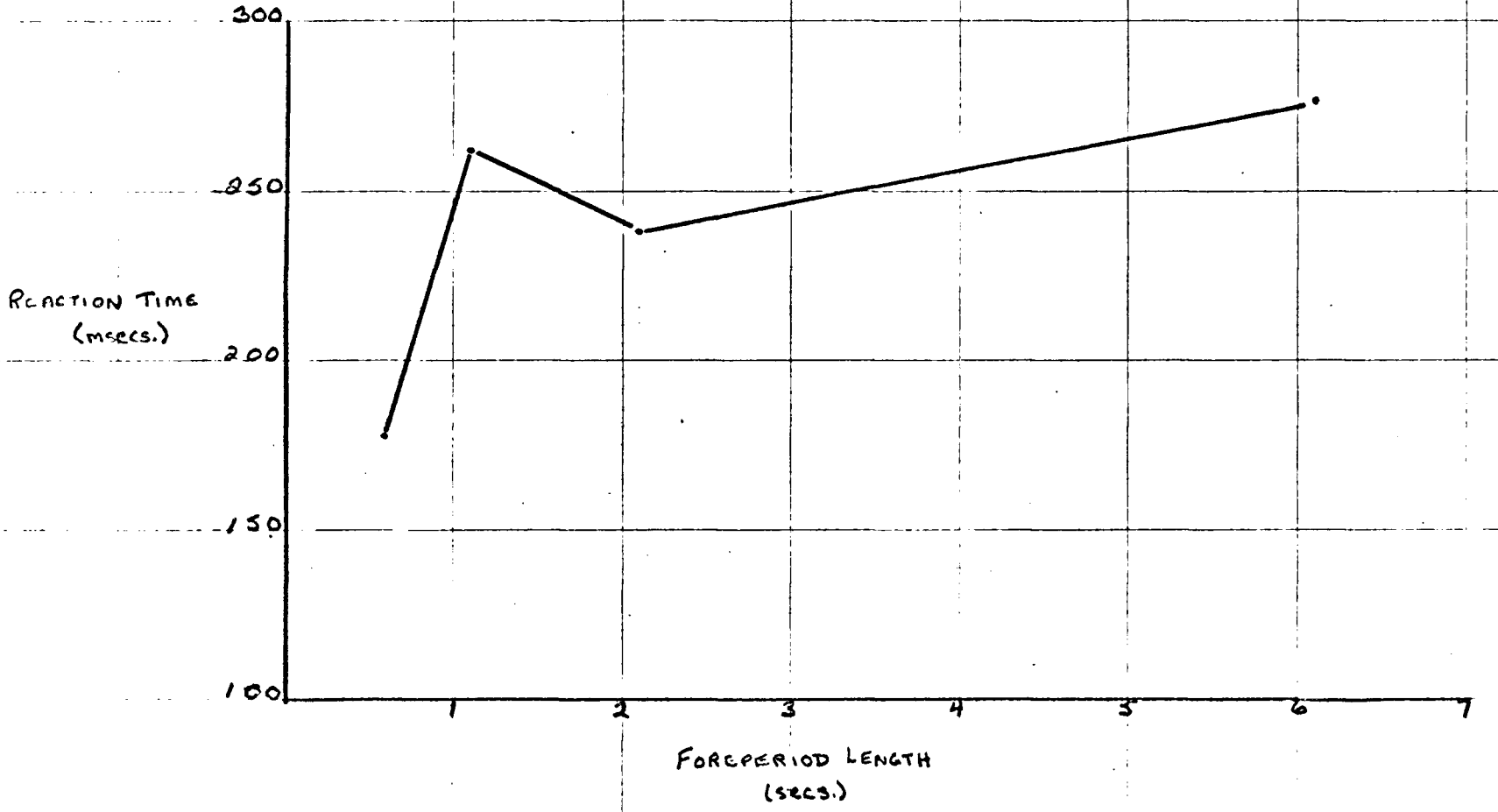

FIG. 4: Reaction time as a function of foreperiod length

Source	DF	SS	MS	F	Prob.
Between Ss	13	22638	1741.4		-
Order	1	73.090	73.090	n.s.	-
Error$_{(between)}$	12	22565	1880.4	-	-
Within Ss	546	-	-	-	-
Foreperiods	3	515.74	171.91	5.82*	.0025
Foreperiods x Order	3	64.862	21.621	n.s.	-
Error$_{(F)}$	36	1063.7	29.548	-	-
Trials	9	19.398	2.1554	n.s.	-
Trials x Order	9	14.427	1.6030	n.s.	-
Error$_{(T)}$	108	142.12	1.3159	-	-
Foreperiods x Trials	27	46.894	1.7368	n.s.	-
Foreperiods x Trials x Order	27	36.086	1.3365	n.s.	-
Error$_{(FT)}$	324	932.17	2.8771	-	-
Total	559	25473.	-	-	-

Table 5: Analysis of Variance on Skin Conductance at the Time of the Stimulus

Table 6 summarizes the effects of foreperiod on Log_{10} R.T., presignal SC, and stimulus SC.

Measure	.6 secs.	1.1 secs.	2.1 secs.	6.1 secs.
$\bar{X}$ Log_{10} R.T.	2.2219	2.3881	2.3539	2.3953
Presignal SC	13.8301	15.9744	13.7113	15.4536
Stimulus SC	13.7619	15.8027	13.7896	15.5726

Table 6: The Effects of Foreperiods on Log_{10} Reaction Time, Presignal and Stimulus Skin Conductance

It would appear that the presence of minor activity in the direction of higher SC (foreperiods 2.1 and 6.1 seconds) was related to a longer R.T. when the background level of arousal was high, i.e., during the 6.1 second foreperiod, but to a shorter R.T. when the background level of arousal was

was low, i.e., during the 2.1 second foreperiod. This gives tentative support to Lykken's (1968) hypothesis that Ss under high arousal will perform poorly with a warning, whereas, those under low arousal conditions will perform well with a warning, c.f., the .6 and 2.1 second foreperiods versus the 1.1 and 6.1 second foreperiods.

Analyses of variance on the cardiovascular variables also indicated significant foreperiod effects (see Tables 7-10). Because the duration of these responses were based on the subjective estimation that the response occurred, acceptance of these measures as valid was based on two requisites. The first was that duration was to be related to magnitude of the response, and the second was that variables found to effect the objective magnitude measures were also to effect the duration measures. Thus, because a variable like foreperiod length was not found to effect the cephalic blood volume pulse, while it was found to effect the "duration" of the cephalic vasomotor response, the data on duration were considered invalid, and the results of analyses omitted. (It must be noted that the cephalic vasomotor response was not clearly manifested by many subjects, and it was expected that this data would be invalid prior to the analyses.)

Source	DF	SS	MS	F	Prob.
Between Ss	13	880.50	67.730		-
Order	1	170.42	170.42	n.s.	-
Error (between)	12	710.07	59.173	-	-
Within Ss	546	-	-	-	-
Foreperiods	3	641.04	213.68	9.88*	.0001
Foreperiods x Order	3	57.914	19.305	n.s.	-
Error ( )	36	778.33	21.620	-	-
Trials	9	312.16	34.685	2.50*	.0123
Trials x Order	9	128.20	14.244	n.s.	-
Error (T)	108	1497.2	13.863	-	-
Foreperiods x Trials	27	358.80	13.289	n.s.	-
Foreperiods x Trials x Order	27	446.51	16.538	n.s.	-
Error (FT)	324	4420.9	13.645	-	-
Total	559	9521.5	-	-	-

Table 7: Analysis of Variance on Difference in Heart-rate Between Pre- and Post-signal Seven Second Periods

Source	DF	SS	MS	F	Prob.
Between Ss	13	80.807	6.2159	-	-
Order	1	21.569	21.569	n.s.	-
Error (between)	12	59.237	4.9364	-	-
Within Ss	546	-	-	-	-
Foreperiods	3	570.77	190.26	108.43*	.0000
Foreperiods x Order	3	7.8527	2.6176	n.s.	-
Error (F)	36	63.165	1.7546	-	-
Trials	9	17.664	1.9627	1.69	.0987
Trials x Order	9	17.369	1.9299	n.s.	-
Error (T)	108	125.17	1.1589	-	-
Foreperiods x Trials	27	41.294	1.5294	n.s.	-
Foreperiods x Trials x Order	27	31.822	1.1786	n.s.	-
Error (FT)	324	406.46	1.2545	-	-
Total	559	1362.4	-	-	-

Table 8: Analysis of Variance on Duration of the Cardiac Response

Source	DF	SS	MS	F	Prob.
Between Ss	13	25.704	1.9772		-
Order	1	.3554	.3554	n.s.	-
Error (between)	12	25.348	2.1124	-	-
Within Ss	546	-	-	-	-
Foreperiods	3	18.354	6.1179	7.38*	.0006
Foreperiods x Order	3	2.8933	.9644	n.s.	-
Error (F)	36	29.826	.8285	-	-
Trials	9	11.459	1.2733	n.s.	-
Trials x Order	9	9.8576	1.0953	n.s.	-
Error (T)	108	112.87	1.0451	-	-
Foreperiods x Trials	27	23.556	.8728	n.s.	-
Foreperiods x Trials x Order	27	21.992	.8145	n.s.	-
Error (FT)	324	322.41	.9951	-	-
Total	559	578.93	-	-	-

Table 9: Analysis of Variance on the Digital Vasomotor Response

Source	DF	SS	MS	F	Prob.
Between Ss	13	256.06	19.697	-	-
Order	1	41.530	41.530	n.s.	-
Error (between)	12	214.53	17.877	-	-
Within Ss	546	-	-	-	-
Foreperiods	3	1333.1	444.38	47.54*	.0000
Foreperiods x Order	3	2.4879	.8293	n.s.	-
Error (F)	36	336.48	9.3468	-	-
Trials	9	23.429	2.6032	n.s.	-
Trials x Order	9	24.935	2.7706	n.s.	-
Error (T)	108	323.19	2.9925	-	-
Foreperiods x Trials	27	79.442	2.9423	n.s.	-
Foreperiods x Trials x Order	27	75.656	2.8021	n.s.	-
Error (FT)	324	1179.0	3.6389	-	-
Total	559	3633.8	-	-	-

Table 10: Analysis of Variance on the Duration of the Digital Vasomotor Response

As predicted, maximal HR deceleration occurred during the 6.1 second foreperiod ($p \leq .05$). The smallest deceleration occurred during the .6 second foreperiod, next smallest during the 1.1 second foreperiod, and next during the 2.1 second foreperiod; however, these differences were not significant. Trials also affected the cardiac response (see Figures 5 and 6) with the response habituating over trials. Of interest is the finding that digital vasoconstriction was greatest during the shortest foreperiod, next during the 1.1 second foreperiod, smaller during the 2.1 second foreperiod, and smallest during the 6.1 second foreperiod ($p \leq .05$). Since the duration of both the cardiac and digital responses was longest during the 6.1 second foreperiod, next during the 2.1 second foreperiod, and shortest during the .6 and 1.1 second foreperiods ($p \leq .05$), it would be expected that a low correlation would hold between the magnitude of the digital response and its duration, and a significant correlation would hold between the magnitude of the cardiac response and its duration. Table 11 indicates the correlations between the durations of the cardiac and digital responses, and between the duration of the responses and their magnitude. The negative correlation between the duration and magnitude of the cardiac response indicates that the greater the deceleration, the longer the duration of the deceleration.

	Duration of Digital Response					Magnitude of Cardiac Response
Foreperiods	.6 secs.	1.1 secs.	2.1 secs.	6.1 secs.	Overall	Overall
Duration of Cardiac Response	.24*	.18*	.02	-.05	.42**	-.14*
Magnitude of Digital Vasoconstriction - Overall	-	-	-	-	.02	-

*$p \leq .05$; **$p \leq .01$

Table 11: Correlations between the Durations of the Cardiovascular Responses, and between the Duration and Magnitude of the Responses

The results on duration are confounded by the possibility that any response to the signal in the shortest foreperiods would be attenuated by responses to the stimulus. However, such an explanation does not account for the finding of a small but significant correlation (r=+.18) between the duration of the cardiac and digital responses and R.T. If the response to the stimulus was overwhelming the signal response, a negative correlation might be expected. No significant correlation was found between HR deceleration and R.T., even during the 6.1 second foreperiod.

DISCUSSION

The length of the foreperiod significantly affected most of the physiological variables as well as reaction time. Theoretically, there was no reason to expect order of the trial blocks to make a difference, and no differences were found attributable to order or to any interactions with order. Duration of the cardiovascular responses was longest during the longest foreperiod, and correlated positively with reaction time, which was also longest during the longest foreperiod. Maximal heart-rate deceleration occurred during the longest foreperiod, and correlated positively with duration of the cardiac response, but did not correlate with reaction time. The highest level of skin conductance occurred during the 1.1 and 6.1 second foreperiods, and the lowest, during the 2.1 and .6 second foreperiods, and reaction time was fastest in these latter foreperiods than in the first. An experimental artifact was probably the cause of the duration differences between the shorter foreperiods and the longer ones; however, this does not

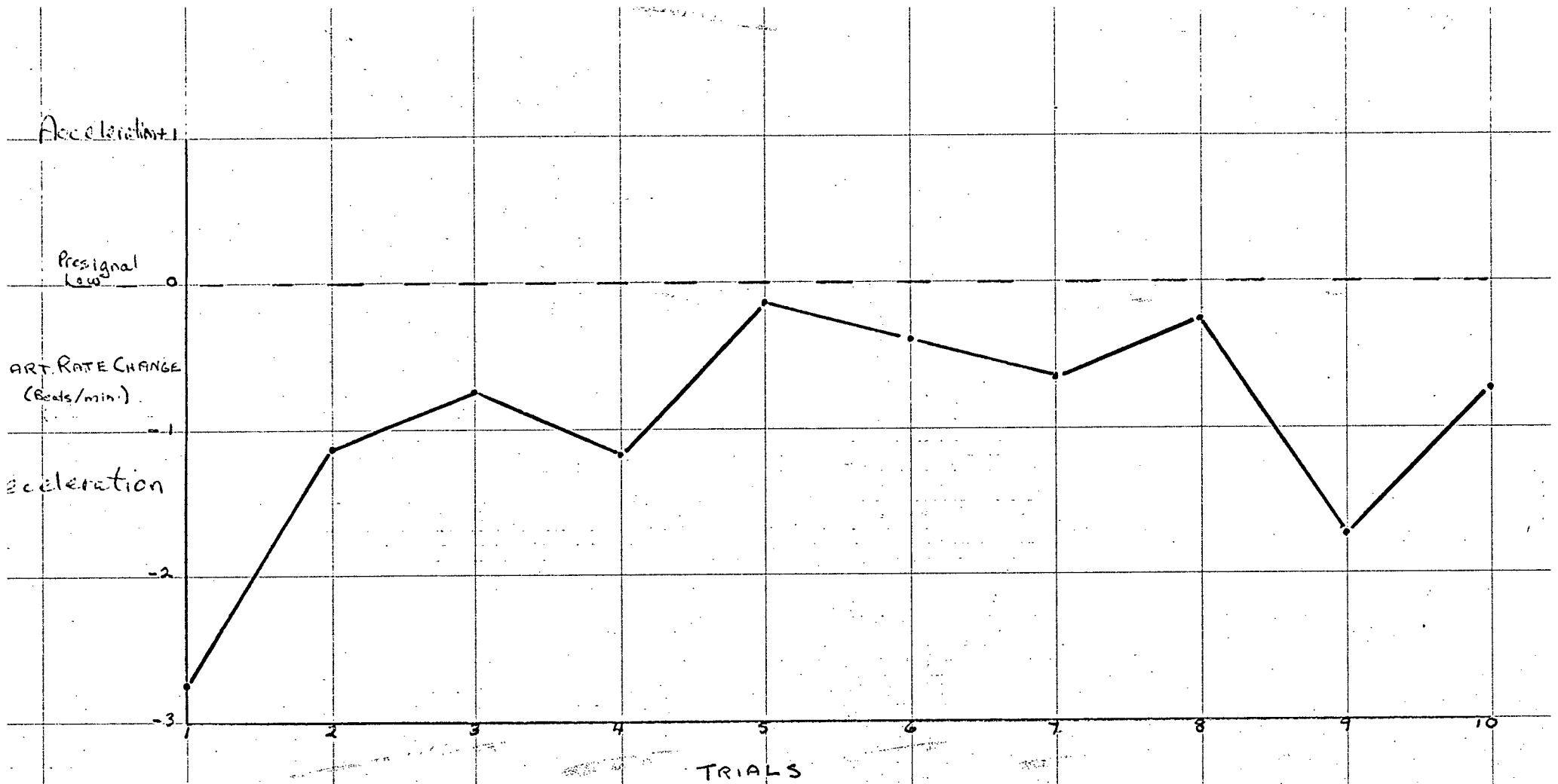

FIG. 5: Heart rate change as a function of trials

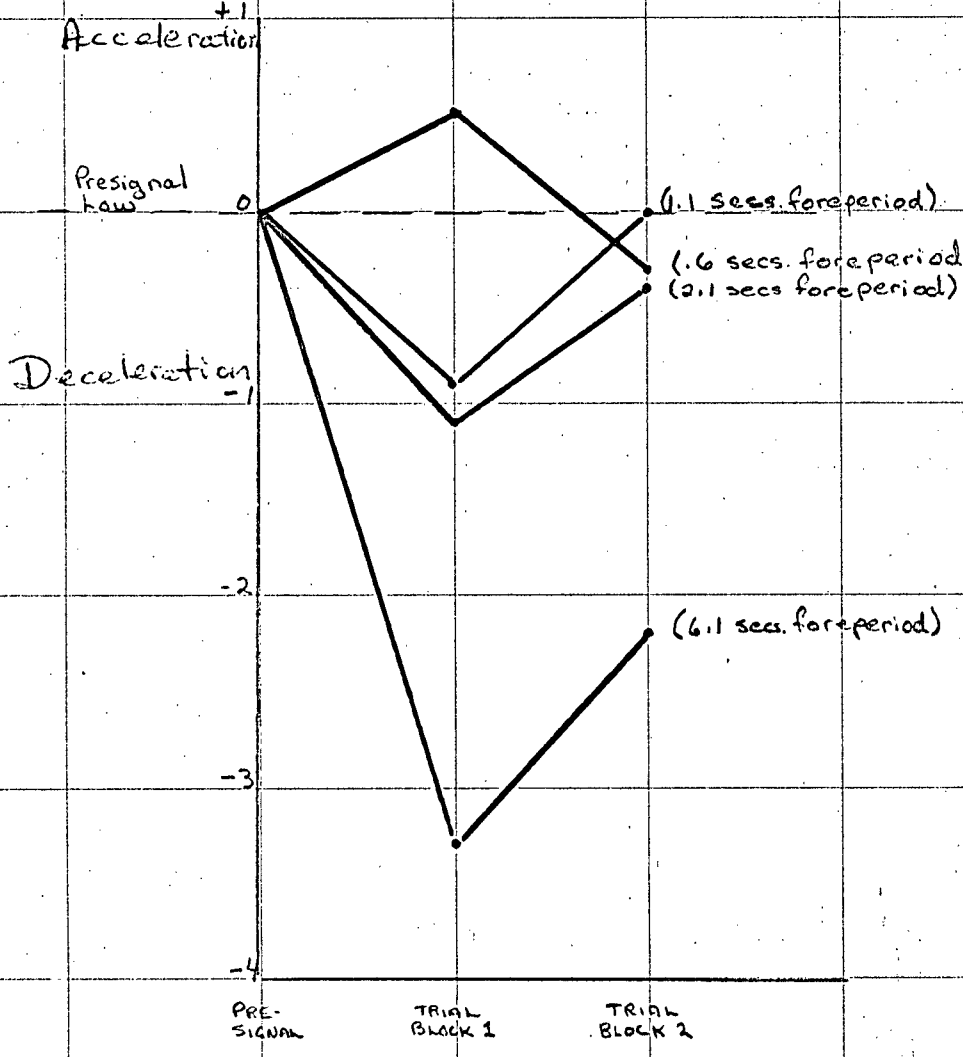

Fig. 6: Heart rate changes as a function of foreperiod and trial blocks. (Trial Block 1 = Trials 1-5; Trial Block 2 = Trials 6-10.)

explain, completely, the finding of a positive correlation between duration and reaction time.

It was found that, whereas heart-rate deceleration was maximal during the longest foreperiod, digital vasoconstriction was maximal during the shortest. It is possible that digital vasoconstriction to the signal was accentuated by constriction to the stimulus and button-push, whereas the decelerative HR component was inhibited by acceleration to the stimulus and button-push. Chase, et al. (1968) have reported that HR accelerated in anticipation of exercise, and they also reported, as did Lacey and Lacey (1965), that HR acceleration began at the time of the stimulus in their reaction time experiments.

The use of a non-continuous signal may have played a strong role in producing results in contradiction to the Lacey's (1965). For the subject who has nothing to attend to (externally) during a long foreperiod, deceleration of the heart-rate may well reflect waiting "at attention" for the stimulus, in line with the Lacey's hypotheses; however, according to Lykken's (1968) theory, this might well be fatiguing in that the arousal state might be too high and might inhibit a motor response. It was found that reaction times were slowest in the two foreperiods with the highest level of skin conductance.

Theoretically, an OR only occurs to "meaningful" stimuli. One may argue that the signal in both the 6.1 and 2.1 second foreperiods were equally meaningful in terms of the fact that both were warnings; however, there may be some justification for regarding the 6.1 second warning signal as too far in advance of the stimulus to serve its function properly, yet insufficiently far in advance to be ignored.

Anokhin (1958) views the OR as having a facilitatory effect on either an ongoing response or a conditioned response, and an inhibitory effect on any response beginning after OR onset. The fast R.T. occurring with a 2.1 second warning might be viewed as evidence for response facilitation, whereas the motor response would be inhibited in the 6.1 second foreperiod, while the "wait" response was facilitated. Obrist (1968) presents evidence which may be viewed as supporting this hypothesis. Obrist suggests that cardio-somatic inhibition produced the results in his experiment using a 7.0 second interstimulus interval between a signal and shock. These results indicated that EMG activity (muscle tension) decreased as the anticipatory deceleration began. Using a 1.0 second interstimulus interval, however, he found that anticipatory cardiac acceleration began shortly after a decelerative phase and was concomitant with increased EMG activity. Increased EMG activity might be expected to coincide with instances of fast reaction times. In the present experiment HR acceleration began more quickly after the stimulus in the 2.1 second foreperiod than in the 6.1 second foreperiod (see Table 12). From Obrist's (1968) data, then, one might have expected that reaction times would be fastest in the 2.1 second foreperiod.

	Foreperiod	
	2.1 secs.	6.1 secs.
Time taken after stimulus to acceleration	0.87 secs.	1.17 secs.

Table 12: Time Taken After Stimulus Before Acceleration

Another of Anokhin's (1958) hypotheses concerning the OR is that the peripheral responses studied are merely components of the OR and are

merely components of the OR and are indicative of the occurrence of central processing. Certainly the correlation data would appear to reflect this. There was little evidence for the peripheral responses correlating with reaction time, although the duration of the responses was correlated with the size of the peripheral responses and with reaction time. This might be viewed as the effect of some central process governing the duration of the physiological responses, which in turn correlates with both the magnitude of the response, as well as with the speed of a motor response. Schematically, this may be depicted as in Figure 7.

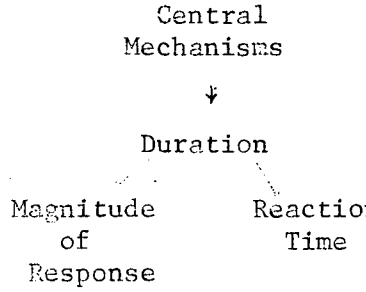

Figure 7: Postulated Relationships Between the OR and R.T.

Obrist (1968) also remarks that both the cardiac and somatic responses may be viewed as reflecting some central process, that, due to the situational requirements, may result in either cardio-somatic inhibition or facilitation.

Lykken (1968) postulated that for subjects under low arousal conditions, a warning signal would lead to reticular activation of the cortex which would result in the facilitation of a motor response as well as the facilitation of an incoming second signal. For subjects under high arousal conditions, however, a warning signal would result in cortical excitation to such an extent that it would inhibit the reticular system, and, thus, delay the perception of a second signal, and inhibit a motor response. Insofar as the SC data may be

taken as indicative of a subject's arousal state the data may also be considered as support for Lykken's hypotheses.

From this data it is possible to suggest, tentatively, certain relationships which would be expected to hold in classical and operant conditioning paradigms. In terms of passive avoidance conditioning, individuals manifesting a long duration OR should be expected to condition well; however, they might do less well if an active motor response was required of the situation. If, however, the OR of such individuals also habituated quickly, giving rise to the defensive reaction, then a motor response would be expected to be facilitated.

In an operant conditioning situation, the presence of a long-lasting OR to a signal should correlate with an individual's ability to perform well in a limited hold procedure, where he is required to inhibit a response. An individual (e.g., a child) in whom the OR was poorly developed, or in whom the OR was of short duration, might not do as well, and might be viewed as unable to avoid punishment or obtain a reward, even though the contingencies might be verbalized. The use of varying signal-stimulus intervals might, with such subjects, result in the finding of paradoxical response facilitation even though the stimulus expected is noxious, and contingent on response suppression. Because this "inhibition" of the OR would depend, in part, on the maturity of the individual, this may be what Luria(1963) is speaking of when he talks of the inhibitory effect of language on a child's motor response, i.e., it may function to inhibit the OR.

Although this study used a small number of subjects, it must be noted that Hastings and Obrist (1967) obtained similar results. In their study, maximal deceleration was found to occur with longer foreperiods (13 and 7

seconds) than with a short (.8 second) foreperiods. They also found that deceleration peaked later in the 13 second foreperiod, possibly the equivalent of the duration spoken of in this paper. Thus, the findings may not be as limited in generality as the number of subjects might suggest.

REFERENCES

Anokhin, P. K. The role of the orienting-exploratory reaction in the formation of the conditioned reflex. In Voronin, L. G., Leontiev, A.N., Luria, A. R., Sokolov, A. N., & Vinogradova, O. S. The orienting reflex and exploratory behaviour. Moscow: The Academy of Pedagogical Sciences of R.S.F.S.R., 1958. (Trans. American Institute of Biological Sciences, 1965.) Pp. 3-16.

Chase, W. G., Graham, F. K., & Graham, D. T. Components of HR response in anticipation of reaction time and exercise tasks. Journal of Experimental Psychology, 1968, 76, 642-648.

Coquery, J. M., & Lacey, J. I. The effect of foreperiod duration on the components of the cardiac response during the foreperiod of a reaction-time experiment. Paper presented at the Annual Meeting of the Society for Psychophysiological Research, Oct. 1966.

Gale, E. N., & Ax, A. F. Long-term conditioning of orienting responses. Psychophysiology, 1968, 5, 307-315.

Graham, F. K., & Clifton, R. K. Heart-rate change as a component of the orienting response. Psychological Bulletin, 1966, 65, 305-320.

Grastyan, E., Lissak, K., Madarasz, I., & Donhoffer, H. Hippocampal electrical activity during the development of conditoned reflexes. Electroencephalography and Clinical Neurophysiology, 1959, 11, 409-430.

Hare, R. D. Psychopathy, autonomic functioning, and the orienting response. Journal of Abnormal Psychology, Monograph Supplement, June 1968, 73.

Hastings, S. E., & Obrist, P. A. Heart-rate during conditioning in humans: effect of varying the interstimulus (CS-UCS) interval. Journal of Experimental Psychology, 1967, 74, 431-442.

Hess, E. H., & Polt, J. M. Pupil size in relation to mental activity during simple problem-solving. Science, 1964, 143, 1190-1192.

Horn, G., & Venables, P. H. The effect of somaesthetic and acoustic stimuli on the threshold of fusion of paired light flashes in human subjects. Quarterly Journal of Experimental Psychology, 1964, 16, 289-296.

Klemmer, E. T. Time uncertainty in simple reaction time. Journal of Experimental Psychology, 1956, 51, 179-184.

Lacey, B. C., & Lacey J. I. Cardiac deceleration and simple visual reaction time in a fixed preperiod experiment. Paper presented at Society for Psychophysiological Research, Wash. D. C., Oct. 1964.

Lacey, B. C., & Lacey, J. I. Cardiovascular and respiratory correlates of reaction time. Fels Research Institute Progress Report, June, 1965.

Lacey, J. I., & Lacey, B. C. The relationship of resting autonomic activity to motor impulsivity. The Brain and Human Behaviour, Baltimore: Williams and Wilkins, 1958, 144-209.

Luria, A. R. The mentally retarded child. New York: The MacMillan Co., 1963.

Lykken, D. T. Neuropsychology and psychophysiology in personality research: Part I. In E. Borgatta and W. Lambert (Eds.), Handbook of Personality Theory and Research. Chicago: Rand McNally, 1968. Pp. 413-509.

Lynn, R. Attention, arousal and the orientation reaction. London: Pergamon Press, 1966.

Obrist, P. A. Heart-rate and somatic-motor coupling during classical aversive conditioning in humans. Journal of Experimental Psychology, 1968, 75,

Paivio, A., & Simpson, H. M. The effect of word abstractness and pleasantness on pupil size during an imagery task. Psychonomic Science, 1966, 5, 55-56.

Razran, G. The observable unconscious and the inferable conscious in current Soviet psychology: interoceptive conditioning, semantic conditioning, and the orienting reflex. Psychological Review, 1961, 68, 81-147.

Simpson, H. M., & Paivio, A. Changes in pupil size during an imagery task without motor involvement. Psychonomic Science, 1966, 12, 572-585.

Sokolov, E. N. Perception and the conditioned reflex. New York: MacMillan, 1963.

Vinogradova, O. S. On the dynamics of the orienting reflex in the course of closure of a conditioned connection, 1958. In Voronin, et al., 1965, 45-53.

Voronin, L. G., Leontiev, A. N., Luria, A. R., Sokolov, E. N., & Vinogradova, O. S. Orienting reflex and exploratory behaviour, Wash. D. C.: American Institute of Biological Sciences, translation, 1965.

Appendix A

Correlation Matrix on Ss 1-14

Table 1: Correlations between Some of the Physiological Responses and Reaction Times for Subjects 1-14.

		.6 secs.	1.1 secs.	2.1 secs.	6.1 secs.
S_1	R.T.	153.5	230.5	174.0	215.5
	Presignal SC	.60	.46	.44	-.36
	Digital Response	-.05	.59	.03	.15
	Cephalic Response	.48	.33	.63	-.31
	*Cardiac Response	-.05	.23	-.28	-.26
S_2	R.T.	219.5	284.0	251.5	228.5
	Presignal SC	-.77**	-.44	.51	-.42
	Digital Response	-.24	.10	.08	-.56
	Cephalic Response	-.46	-.35	.03	.07
	Cardiac Response	.32	.12	-.42	.28
S_3	R.T.	177.0	257.0	198.0	440.5
	Presignal SC	-.34	-.69**	.38	.22
	Digital Response	.02	-.41	-.15	.11
	Cephalic Response	-.14	.23	-.14	-.22
	Cardiac Response	-.21	.22	-.34	-.22
S_4	R.T.	177.0	266.0	277.5	262.0
	Presignal SC	-.34	.47	-.02	.19
	Digital Response	.02	-.14	.12	.34
	Cephalic Response	-.14	.40	.14	-.44
	Cardiac Response	-.21	.50	-.29	-.12
S_5	R.T.	160.5	265.5	259.0	370.0
	Presignal SC	.40	-.31	.13	-.16
	Digital Response	-.46	-.21	-.37	.40
	Cephalic Response	-.01	.19	-.33	.40
	Cardiac Response	.30	-.23	.20	.06
S_6	R.T.	128.5	156.0	185.0	185.5
	Presignal SC	.65**	-.39	-.14	.34
	Digital Response	-.15	.56	-.02	-.14
	Cephalic Response	.77**	-.02	.09	-.37
	Cardiac Response	-.63**	.38	-.44	-.24
S_7	R.T.	202.5	291.5	365.5	355.0
	Presignal SC	.22	-.33	.06	-.13
	Digital Response	.12	.09	-.05	.78**
	Cephalic Response	.16	-.10	.07	.62
	Cardiac Response	-.12	.03	-.26	.69**

Table 1 Continued

		.6 secs.	1.1 secs.	2.1 secs.	6.1 secs.
S_8	R.T.	227.5	287.0	286.5	269.0
	Presignal SC	-.08	-.29	-.00	-.24
	Digital Response	-.39	.24	.09	.64**
	Cephalic Response	.15	.29	-.02	.38
	Cardiac Response	-.02	-.12	.38	-.28
S_9	R.T.	146.0	200.5	208.5	186.5
	Presignal SC	-.36	-.54	.30	-.13
	Digital Response	-.36	.09	-.02	-.50
	Cephalic Response	.59	.04	-.69**	.04
	Cardiac Response	-.16	-.28	.34	.66**
S_{10}	R.T.	219.5	232.0	230.0	229.9
	Presignal SC	.10	-.71	-.81	.50
	Digital Response	.31	-.12	-.13	-.01
	Cephalic Response	-.13	.26	-.18	-.63**
	Cardiac Response	-.30	.14	-.56	.77**
S_{11}	R.T.	111.0	237.5	194.5	206.5
	Presignal SC	.02	-.29	-.05	-.35
	Digital Response	-.29	.48	-.30	-.39
	Cephalic Response	.34	-.08	-.72**	-.66**
	Cardiac Response	.39	-.12	.01	.18
S_{12}	R.T.	255.5	501.0	352.5	508.0
	Presignal SC	-.23	.01	.19	-.34
	Digital Response	-.18	.00	-.00	-.15
	Cephalic Response	.32	.14	-.05	-.17
	Cardiac Response	.20	.07	-.05	.24
S_{13}	R.T.	164.0	214.5	212.0	241.5
	Presignal SC	.04	.10	-.27	.45
	Digital Response	.76	-.05	.64**	.42
	Cephalic Response	.49	.07	.25	-.12
	Cardiac Response	.00	-.50	-.35	-.40
S_{14}	R.T.	141.0	203.5	187.0	173.5
	Presignal SC	.36	-.55	.07	-.61
	Digital Response	-.22	-.03	.16	.18
	Cephalic Response	-.46	-.65**	-.36	-.04
	Cardiac Response	-.41	-.13	.33	.42

*Negative correlations indicate that the faster the reaction time the less the HR deceleration.

**Significant correlations, p=.05.