ABSTRACT

HEART PERIOD AND RESPIRATORY CONCOMITANTS OF ATTENTION IN NORMALS AND RETARDATES DURING A FIXED REACTION TIME TASK

By

Antoinette Krupski

This study demonstrated significant differences between retardates and normals in reaction time (RT) performance and in sec-by-sec heart period (HP) activity during preparatory intervals (PIs) of 4, 7, and 13-secs. There were no significant group differences in respiration frequency corresponding to the HP changes.

Normals were characterized as exhibiting significant HP deceleration at about the time the reaction signal occurred in all three PI conditions. Retardates, on the other hand, were characterized as showing no significant HP deceleration in the 4-sec and 7-sec PI conditions while showing a significant deceleration in the 13-sec PI condition. Group differences in HP deceleration were interpreted as indicating an inhibition deficit, or as indicating an absence of temporal conditioning in retardates. The results were also related to Lacey's theory of attention.

Heart period changes to the onset of stimulation

also differed for groups as a function of PI. Normals exhibited progressively larger accelerations as a function of PI length. Retardates, on the other hand, exhibited the same magnitude of acceleration for each PI condition. These data were interpreted as indicating an inappropriate response set, an inhibition deficit, and as reflecting a lack of integration between environmental demands and physiological responsiveness in retarded individuals.

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By

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To my dearest and best friend, Don.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF APPENDICES

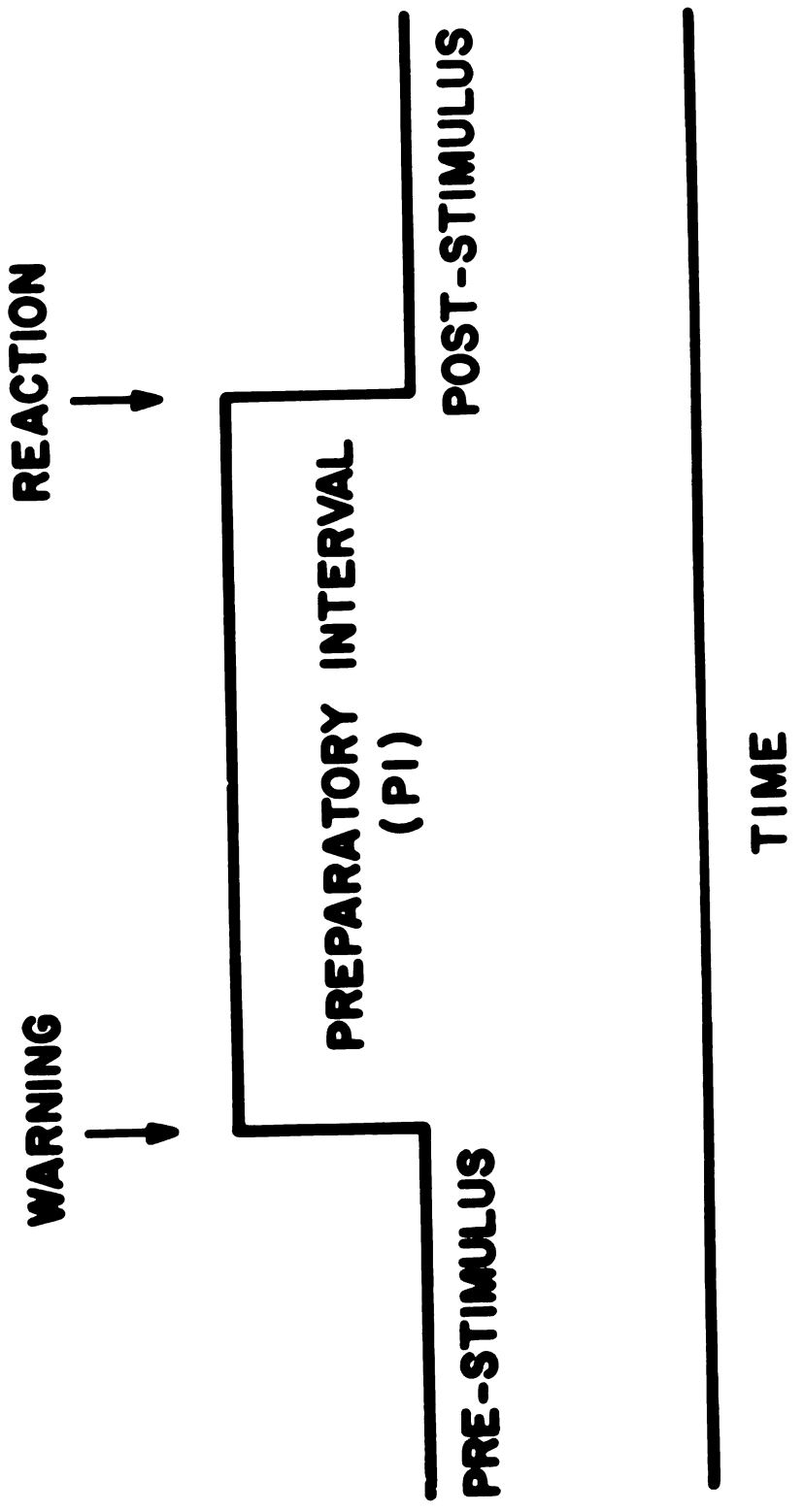
INTRODUCTION

When compared to normals, retardates typically perform significantly slower on reaction time (RT) tasks (Baumeister & Kellas, 1968). Several investigators have suggested that the retardate's slower RT is due to an inability to maintain the level of attention that is required for a fast RT (Baumeister & Kellas, 1968; Denny, 1964). The specific purpose of the present study was to examine heart rate (HR) changes that occur in both retarded and non-retarded individuals during a RT task in an attempt to evaluate the alleged "attention deficit" frequently attributed to retardates. Heart rate was chosen for this assessment because recent theoretical analyses suggest that the direction of response, that is HR acceleration or HR deceleration, is related to environmental demands (Coquery & Lacey, 1966; Lacey, 1967; Lacey & Lacey, 1966; Obrist, Sutterer, & Howard, 1969a; Obrist, Webb, & Sutterer, 1969b). The directional HR response has the additional advantage of being a discrete response that is amenable to precise quantification (Brener, 1967).

The typical RT design is depicted in Figure 1. As depicted in Figure 1, a simple RT situation typically involves the successive presentation of a warning signal, a preparatory interval (PI), and a reaction signal. For

FIGURE 1

A typical fixed reaction time paradigm.


WARNING
REACTION
PRE-STIMULUS
PREPARATORY INTERVAL
(PI)
POST-STIMULUS
TIME

example, the subject (S) is told to press a button as quickly as he can when the green light goes off--onset of the green light being the warning signal and offset of the green light being the reaction signal. Reaction time score is the elapsed time between onset of reaction signal and occurrence of a motor response. The PI is the time between onset of warning and reaction signals, i.e., the time during which S can prepare to respond to the reaction signal. When the PI is the same duration throughout a block of trials, the task is called a fixed RT task. It is generally assumed that S is attending or concentrating during the PI if he makes a fast response.

A growing body of literature relates directional HR changes to RT performance, and theoretically to attention. The majority of these studies have been done with nonretarded Ss and have typically employed a fixed RT task where the PI is 4-secs or longer (Chase, Graham, & Graham, 1968; Coquery & Lacey, 1966; Lacey & Lacey, 1966; Obrist, et al., 1969a, 1969b; Obrist, Webb, Sutterer, & Howard, 1970; Webb & Obrist, 1970). The resulting HR pattern during the PI is usually triphasic in nature: sometimes a brief deceleration is reported to occur after the warning signal, followed by an acceleratory response, and finally a deceleratory response in anticipation of the reaction signal. Studies employing a classical conditioning paradigm with a fixed CS-UCS interval greater than 4-secs in length have yielded similar HR patterns (Hastings & Obrist,

1967; Headrick & Graham, 1969; Wilson, 1969; Wood & Obrist, 1964; Zeaman & Smith, 1965). Research in this area has typically focused upon the HR deceleration which occurs prior to the reaction signal in the fixed RT studies, and prior to the appearance of the UCS in the classical conditioning studies. This focus on HR deceleration probably stems from the finding that magnitude of anticipatory HR deceleration has been found to be positively correlated to RT performance; that is, the greater the HR deceleration, the faster the RT (Obrist et al., 1969a, 1969b; Lacey, 1967; Lacey & Lacey, 1966; Coquery & Lacey, 1966).

Lacey (1967) interprets the HR deceleration immediately preceding a reaction signal in the fixed RT situation as a state of enhanced sensitivity to external stimulation and hence, greater attention. Consistent with Lacey's interpretation, Graham & Clifton (1966) suggest that cardiac deceleration is a component of the orienting response (OR) which is also presumed to reflect a state of enhanced sensitivity. It follows from both of these approaches that a large HR deceleration occurring at about the time a signal stimulus is to occur would result in enhanced sensitivity to external stimuli, enhanced attention to environmental demands, and therefore, a faster RT.

Other investigators have suggested that anticipatory HR deceleration is a temporally conditioned response which occurs as a function of the fixed PI interval or the fixed CS-UCS interval (Chase et al., 1968; Fitzgerald &

Porges, 1970; Headrick & Graham, 1969; Porges, 1970). The temporal conditioning notion is supported by the fact that anticipatory deceleration does not become time-locked to the reaction signal or UCS when the PI or CS-UCS intervals vary from trial to trial (Obrist et al., 1969a; Porges, 1970). Other support results from trial analyses which demonstrate that the anticipatory HR deceleration does not occur on the first trial, but becomes time-locked to the signal stimulus as a function of trials (Chase et al., 1968; Hastings & Obrist, 1967; Headrick & Graham, 1969; Porges, 1970). Chase et al. (1968) propose that the conditioned deceleration which occurs in anticipation of the reaction signal corresponds to a conditioned "attention" response as described by Lacey. Consequently, their conditioning notion can be viewed as an extension of the Lacey hypothesis.

In contrast to the Lacey and Chase et al. contention, Porges (1970) asserts that the observed anticipatory HR deceleration and RT performance are not related in a causal fashion, but that significant correlations between the two reflect that temporal cardiac conditioning improves over trials as does RT. For example, Porges found that the magnitude of anticipatory HR deceleration increased as a function of trials in a RT situation. Moreover, even when the magnitude of the decelerative HR response was greatest, it was not significantly correlated to RT. Porges contends that if magnitude of HR deceleration reflects enhanced attention magnitude of HR deceleration should be strongly correlated with RT.

Although anticipatory HR deceleration prior to a reaction signal is reliably observed in a RT situation, the specific role of this deceleration is ambiguous. Lacey and Chase et al. relate these changes to an attentional process whereas Porges relates them to temporal conditioning which is coincidental to but not strongly correlated with motor performance. (A more detailed interpretation of the temporal conditioning notion will be discussed later.)

One of the purposes of the present study was to observe the HR activity of retardates in the fixed RT situation. It was felt that such observation might provide some insight into the nature of the retardate's alleged attention deficit as well as contribute to increased understanding of their poorer RT performance. Moreover, it was felt that the study of anticipatory HR deceleration in retardates would contribute to the existing theoretical frameworks concerned with directional HR changes; theoretics which have been based almost exclusively upon data obtained from normal individuals.

Heart rate acceleration, which is the second "limb" of the HR response pattern during the PI, has received less attention than anticipatory deceleration. However there are data which suggest it has important behavioral significance in that it may be related to task demands. Heart rate acceleration typically occurs after the warning signal, sometimes preceded by a brief deceleration (Chase

et al., 1968; Headrick & Graham, 1969). Coquery & Lacey (1966) reported that HR accelerations following the warning signal in a short PI (e.g., 4-secs) were attenuated when compared to accelerations in PIs of longer duration (e.g., 7-secs). Chase et al. (1968) replicated Lacey's findings in a 4-sec PI using a button-press PT task in one group of Ss. However, they also employed a second group of Ss whose RT task was leg-lifting rather than button-pressing. In this second group, significant HR acceleration followed the warning signal, presumably reflecting the more strenuous demands of the leg lift task.

A second study which relates HR acceleration to task demands was reported by Porges (1970). He found that HR accelerated to RT warning signals and decelerated to control non-signal stimuli. Control, non-signal stimuli were identical to the RT warning stimuli; the only difference between them being the instructions that E read. To summarize, both the Chase et al. and the Porges studies suggest that task demands are reflected in HR acceleration following the warning signal; the more strenuous the task, the more marked HR acceleration becomes.

Another purpose of the present study then, was to examine HR changes following the warning signal in retarded and normal individuals in order to assess retardate's acceleration responses in PIs of both short and long duration as well as to do comparative analyses between retarded and normal groups.

Purpose. The general purpose of the present study was to evaluate the "attention deficit" frequently attributed to retardates. Recent psychophysiological work done with normals suggests that this alleged deficit would be reflected in group differences in the anticipatory HR deceleration occurring prior to the reaction signal as well as in HR acceleration following the warning signal.

Another interest of this study centers upon possible differences between retardates and normals in both acceleratory and decelatory phases of the HR pattern during PIs of different lengths. Although RTs have not been found to interact with PI length (Baumeister & Kellas, 1968), a time estimation study by McNutt & Melvin (1968) suggests that group differences in different PI conditions might exist. These investigators report that retardates were not significantly different from normals in their ability to estimate a 5-sec tone, but had much greater difficulty judging the length of a 13-sec tone. Thus, PIs of 4, 7, and 13-secs were used in the present study. These PIs are representative of short, moderate, and long PIs as operationally defined by previous investigators.

METHOD

Subjects. Subjects were 12 normal males and 12 non-institutionalized retarded males, mean age 20 and 21, respectively. The normal Ss, from Michigan State University, received extra course credit for their participation, while the retarded Ss, from the Lansing metropolitan area, were paid $2.00 for their participation. Retarded males had a Wechsler mean IQ of 70 (range 55 to 82). Subjects had no sensorimotor impairments. Neither had they received any drugs or medications for at least 2 weeks prior to the experiment.

Apparatus. The RT stimulus was a green 24 V. DC jewel light located about 3 feet in front of S at eye level. A white light, located 2 in. below the green light, served as the rest period stimulus and became illuminated only between blocks of trials. The presentation of these stimuli was controlled by 2 Hunter timers. The RT apparatus was a microswitch which was mounted in a wooden block. The microswitch was placed on the arm of the chair in which S was seated under Ss preferred hand. In this position, S's forearm and heel of the hand were supported by the arm of the chair and S's index finger extended above the microswitch. Reaction time was measured in milliseconds by a Standard electric clock. Subjects were tested in a

sound-attenuated room where the temperature was maintained at approximately 70° F. and the ambiant noise level was 51 db.

The physiological responses, RT stimulus, and RT responses were continuously recoreded on a 4-channel Grass P7 polygraph at a paper speed of 10mm/sec. Heart period (HP) and skin conductance (SC) recording sites were cleaned with 70% ethanol prior to the application of electrodes. Grass gold disc electrodes, filled with Grass electrode paste, were used to record HP from EKG Lead I. Heart period measures were fed into a Grass Model 7P6A EKG pre-amplifier. Zinc cup electrodes with a surface area of 3.14 sq. cm. and filled with cotton soaked in a 1% $ZnSo^4$ solution were used to record SC. Skin conductance electrodes were placed on the base of the thumb and on the inside of the forearm about 2 in. below the elbow of Ss non-preferred arm.

Changes in respiration were measured by a pneumograph that was fitted around Ss chest with a Velcro fastener. The pneumograph consisted of 2 wire strain gauges that were glued to a metal arch in a half-bridge configuration. Matching dummy resistors were used for balancing the bridge. Any changes in chest circumference resulted in changes in cord length of the metal arch and thus changes in the strain gauges. Strain gauge output was fed directly into the polygraph.

Finger vasomotor activity was measured with a

photoelectric plethysmograph. The pickup consisted of a block of black phenolic plastic in which were mounted a Clairex C1704 photocell and a General Electric 683 miniature lamp powered by a 3 V. battery. The pickup was taped flush to the tip of the finger. This arrangement held the surface of the pickup securely against S's fingertip with relatively light, constant pressure, which prevented discomfort to S and the occluding of local blood vessels. The signal from the plethysmograph bridge was recorded DC.

Procedure. All Ss were individually tested in the developmental psychophysiology laboratory at Michigan State University. After arriving at the lab, S was seated in a comfortable armchair in a sound-attenuated room. The female experimenter attached the electrodes and briefly explained their purpose. Heart rate, respiration, finger vasomotor, and SC were measured, but because of mechanical difficulties SC and vasomotor data were not scorable and will not be reported here.

After the equipment was calibrated, E read the instructions. Each S was told that a green light located 3 feet in front of him at eye level would come on periodically. Onset of the green light marked the beginning of the PI; this light remained illuminated for the entire PI. Subjects were told that their job was to press the key (i.e., the microswitch) as quickly as possible when the green light went off. A white light, located 2 in. below the green light, served as a rest period stimulus and

became illuminated only between blocks of trials.

Each S participated in a single session which consisted of 3 blocks of trials and 2 rest periods. A trial block consisted of 15 trials of either 4, 7, or 13-sec fixed PI. Only one PI value was used per trial block. For example, an S assigned to a 4-7-13-sec PI combination received the following sequence: 15 RT trials in which the PI was 4-secs, a 2-min. rest period, 15 RT trials in which the PI was 7-sec, another 2-min. rest period, and 15 RT trials in which the PI was 13-secs. Subjects were randomly assigned to one of the six possible combinations of the 3 trial blocks when they appeared at the lab. The assignment of order was counterbalanced for each group so that there was an equal number of Ss in each order. The inter-trial-interval varied among 10, 15, and 20-secs.

The S was alone during the entire experimental session, but could communicate with E by a talk-a-phone intercom system. Five practice trials preceded the first trial block. If S did not respond or responded too frequently, E communicated this to him during the practice period. At the end of practice, all Ss were told that the real test would now begin.

Data Quantification. Only the last 10 trials in each PI condition (or, block of trials) were scored for HP and respiration. In the counter-balanced design, there were 6 possible orders of presentation of the 3 PI conditions. Given the assignment procedure, there were 2 Ss

in each of the 6 orders in each group. Although a 2-min. rest period intervened each block of trials, the small number of Ss in each cell made it difficult to statistically determine the effect of preceding conditions on subsequent blocks of trials. Consequently, the first 5 trials in each PI condition were omitted from the analyses in an attempt to avoid possible transfer effects or disruptions from preceding conditions.

Heart period (HP) was scored by determining the number of milliseconds between successive heart beats, or R-R intervals. The HP reading for each 1-sec interval consisted of the number of milliseconds between R-R intervals which occurred during that sec. If more than one R-R interval was completed during any given sec, only the first cycle was scored for that sec. The HP data was evaluated in separate sec-by-sec analyses for each PI condition. Seconds which were analyzed included 4-secs prior to PI onset, all secs during the PI, and 4-secs following PI offset.

Respiration was analyzed by counting the frequency of initiations and terminations of inspirations during "pre", "PI", and "post" periods for each of the 3 PI conditions. In the 4-sec PI condition, the "pre" period consisted of the 4-secs prior to the onset of the light, the "PI" period consisted of the 4-secs during light presentation, while the "post" period consisted of the 4-secs following the light. In the 7-sec PI condition, the "pre"

period consisted of the 7-secs preceding the light, the "PI" period consisted of the 7-secs during light presentation, while the "post" period consisted of the 7-secs following the light. The 13-sec PI condition was analyzed in 2 different ways: In the first analysis, the "pre" period consisted of the 13-secs preceding the light onset, the "PI" period consisted of the 13-secs during light presentation, while the "post" period consisted of the 13-secs following the light. In the second analysis, the 13-sec PI was analyzed with the "pre" period consisting of the 6-secs preceding light onset, the "PI" period was broken down into "early PI" and "late PI" components, with "early PI" consisting of the 6-secs following light onset, and "late PI" consisting of the 6-secs preceding light offset, while "post" period consisted of the 6-secs following light offset.

RESULTS

Reaction Time. An analysis of variance was performed on the RT data. This analysis revealed that retardates had significantly slower RT than normals in all three PI conditions ($F(1,66)=17.79$, $p. < 0005$). The analysis of variance summary table is presented in Appendix B. The group x PI interaction ($F(2,66) < 1.0$) was not significant. These results are consistent with the majority of studies that have examined RT in retarded and normal individuals (Baumeister & Kellas, 1968). Mean RTs and standard deviations for each group and for each PI are presented in Table 1. These means were calculated on the

Table 1. Reaction Time: Mean and standard deviations expressed in milliseconds for retarded and normal groups in 4, 7, and 13-sec PI conditions.

	Group			
	Normals		Retardates	
PI	Mean	SD	Mean	SD
4	293.58	76.17	408.68	240.45
7	299.73	73.34	450.65	243.00
13	324.20	83.51	465.48	214.05

last 10 trials of each PI condition for each S. All 10 trials were included in the calculation of the means with the exception of trials on which false responses occurred prior to the reaction signal. Such false responding occurred 5 times in the retarded group and 8 times in the normal group. In each of these cases the mean RT score of the other trials was assigned to the false response trial in order to maintain equal frequencies in all cells.

Correlations Between RT and IQ Scores. Pearson Product moment correlations between mean RT score and IQ scores were performed for the retarded group. Wechsler combined IQ scores were available for all 12 retarded Ss; verbal and performance scores were available for all but one of these Ss. These correlations are presented in Table 2. A negative correlation indicates that low IQ is

Table 2. Pearson product moment correlations between total IQ, verbal IQ, performance Wechsler IQ scores and mean reaction times for 4, 7 and 13-sec PI conditions for the retarded group.

PI	TOTAL IQ (df=10)	VERBAL IQ (df=9)	PERFORMANCE IQ (df=9)
4	-.388	-.224	-.707*
7	-.384	-.351	-.782**
13	-.347	-.336	-.750**

* $p < .02$
** $p < .01$

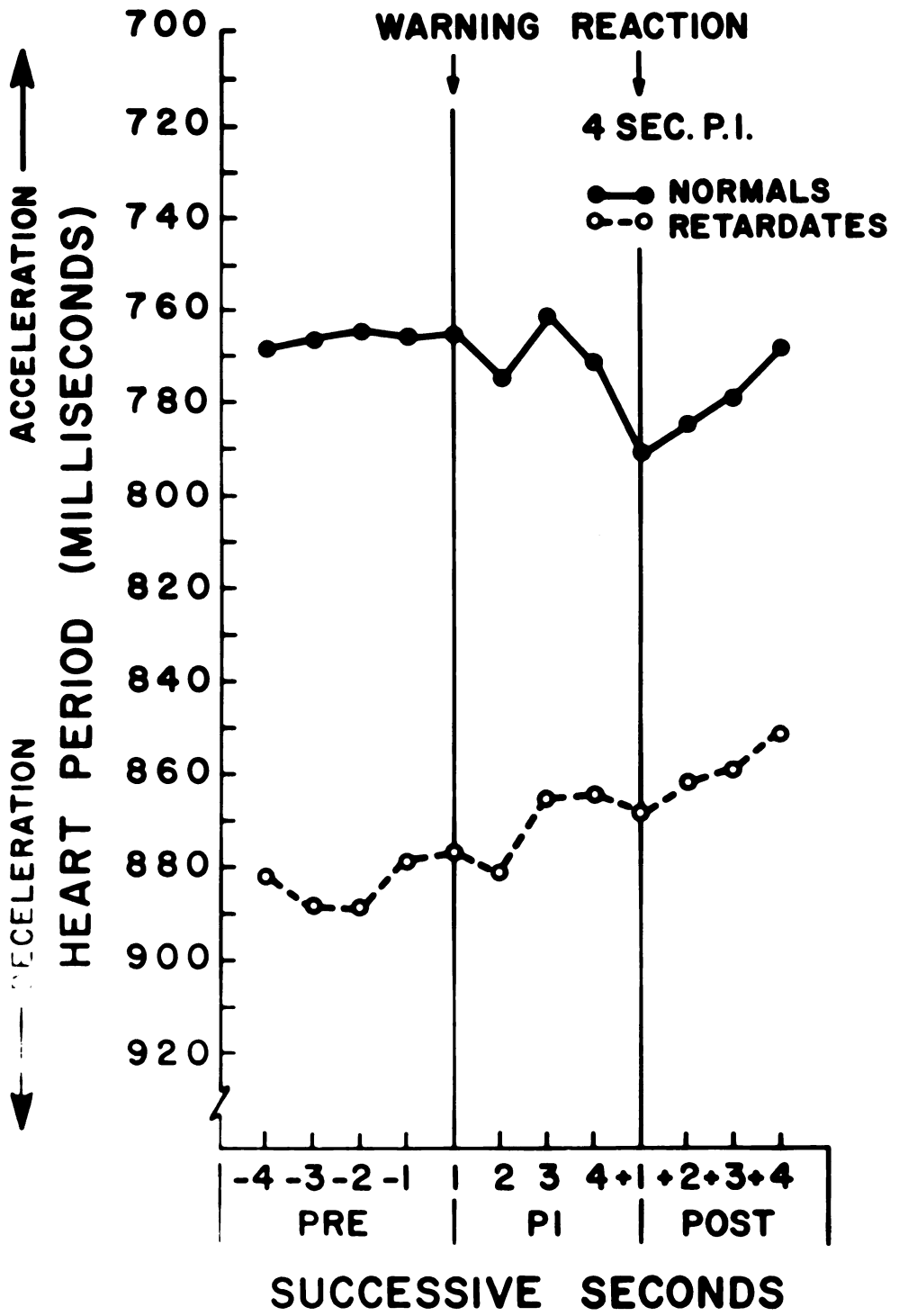
related to a slow RT. As can be seen from the table, significant correlations were found between performance IQ and RT.

Heart Period. An attempt was made to evaluate the HP data in a comprehensive analysis of variance that included the three PI conditions. Unfortunately this type of analysis presented several inherent difficulties: In order to have equal cell frequencies, only the first 4-secs or last 4-secs in each PI was examined in the comprehensive analysis, resulting in a deletion of a large amount of data. Moreover, the results of this analysis revealed a number of 3 and 4-way interactions which, though interpretable, left more questions unanswered than answered. Therefore, to facilitate meaningful analysis of the data the sec-by-sec HP data was analyzed separately for each PI condition.

The analysis of variance results for the 4-sec PI condition is presented in Figure 2. This figure shows HP as a function of successive secs in the 4-sec PI for each group. Since HP is the number of milliseconds between successive heart beats, the larger values indicate a slower HR, or HR deceleration, and smaller values indicate acceleration. These values are plotted so that a downward slope indicates deceleration, and an upward slope indicates acceleration. Successive secs are plotted along the abscissa; secs -4 to -1 represent the prestimulus period, or the 4-secs preceding the warning signal. Secs 1 to 4

FIGURE 2

Mean heart period change as a function of successive seconds in the 4-sec PI condition for normal and retarded groups.


WARNING
REACTION
4 SEC. P.I.
NORMALS
RETARDATES
ACCELERATION
DECELERATION
HEART PERIOD (MILLISECONDS)
700
720
740
760
780
800
820
840
860
880
900
920
-4 -3 -2 -1
2 3 4 +1 +2 +3 +4
PRE
PI
POST
SUCCESSIVE SECONDS

represent the PI, while secs +1 to +4 follow the reaction signal. The analysis of variance revealed significant group differences in overall HP base levels with retardates having significantly slower HR (or, longer R-R intervals) than normals ($F(1,22)=4.20$, $p < .05$). In the 4-sec PI, the HR of normal Ss was 78 beats per minute (bpm) while the HR of retardates was 68 bpm. The significant ($F(11, 242)=1.92$, $p < .038$) sec-by-sec HP pattern for combined groups during the PI was triphasic in nature: A slight HP deceleration followed the warning signal followed by a small acceleration which peaked on sec 3 of the PI. This acceleration was succeeded by a deceleration which reached its nadir on sec +1, i.e., the sec on which the reaction signal occurred. Heart period returned to base level during the 4 secs following the reaction signal.

There was also a significant group x sec interaction ($F(11, 242)=4.22$, $p < .0005$) which is illustrated in Figure 2. Following the warning signal, both retarded and normal groups showed a small deceleration followed by an acceleration. However, in normal Ss this acceleration was followed by a deceleration while retarded Ss did not evidence this decelerative HP response.

In order to assess the statistical significance of the accelerative and decelerative components of the HP response during the PI, t-tests for related measures were performed between mean basal HP and HP acceleration score, and mean basal HP and HP deceleration scores, for each

group, and for each PI condition. Mean base level HP was determined for each S by computing the mean HP for the 4-secs prior to PI onset. The greatest HP acceleration occurring within the 4-sec PI was recorded as the HP acceleration score for each S, while the HP deceleration was socred in 2 ways: The first HP deceleration score (I) was computed by recording the greatest deceleration which occurred during a 5-sec period which included the last 3-secs of the PI and the first 2 secs following the reaction signal. Thus, in this analysis, the HP deceleration score was based on the lowest HR (or, longest R-R interval) in any one of 5 secs surrounding the reaction signal. The second HP deceleration score (II) was computed by recording the HP occurring during sec +1, or the sec on which the reaction signal occurred. Thus, in this analysis, the HP deceleration score was based on the HP of only one sec. In order to compensate for the problem of correlated errors inherent in non-independent t-tests, the appropriate alpha level was set at .01 (2-tailed test).

The t-test analysis performed on the HP acceleration score in the 4-sec PI condition revealed that the normal S's HP acceleration following the warning signal was not significantly different from HP base level ($t(11)=2.65$, $p<.05$), while the retardate's HP acceleration was significantly different from HP base level ($t(11)=4.11$, $p<.01$). In both HP deceleration analyses, the normal group's deceleration approached the predetermined significance

level (I:t(11=2.999, p <.02; II:t(11)=2.263, p <.05), whereas the retarded group's HP deceleration was clearly not statistically significant (I:t(11)=0.836, ns; II:t(11)= 1.502, ns). Following the reaction signal, HP acceleration occurred in both groups, however, in the normal group it approached prestimulus levels, while in the retarded group it exceeded prestimulus levels. The analysis of variance summary table for the 4-sec PI is presented in Appendix B.

The analysis of variance of HP during the 7-sec PI condition revealed significant group differences in overall base levels with retardates having significantly slower HR (or, longer R-R intervals) than normals (F(1,22)= 5.78, p <.025). This finding is like the 4-sec PI where retardates also had slower HRs. In the 7-sec condition, the normal's mean HR was 78 bpm and the retardate's mean HR was 67 bpm. There were also significant trial effects (F(9,198)=2.50, p <.01) which indicated that HR increased over trials for both groups (or, R-R intervals shortened).

As in the 4-sec PI, significant sec-by-sec HP changes were found for combined groups in the 7-sec PI condition (F(14,308)=10.97, p <.0005). One sec after the warning signal marked the onset of HP acceleration which peaked on sec 4 of the PI. A deceleration followed which reached its nadir on sec +1, i.e., the sec on which the reaction signal occurred. After the reaction signal, HP increased, reaching prestimulus levels 4-secs after the reaction signal.

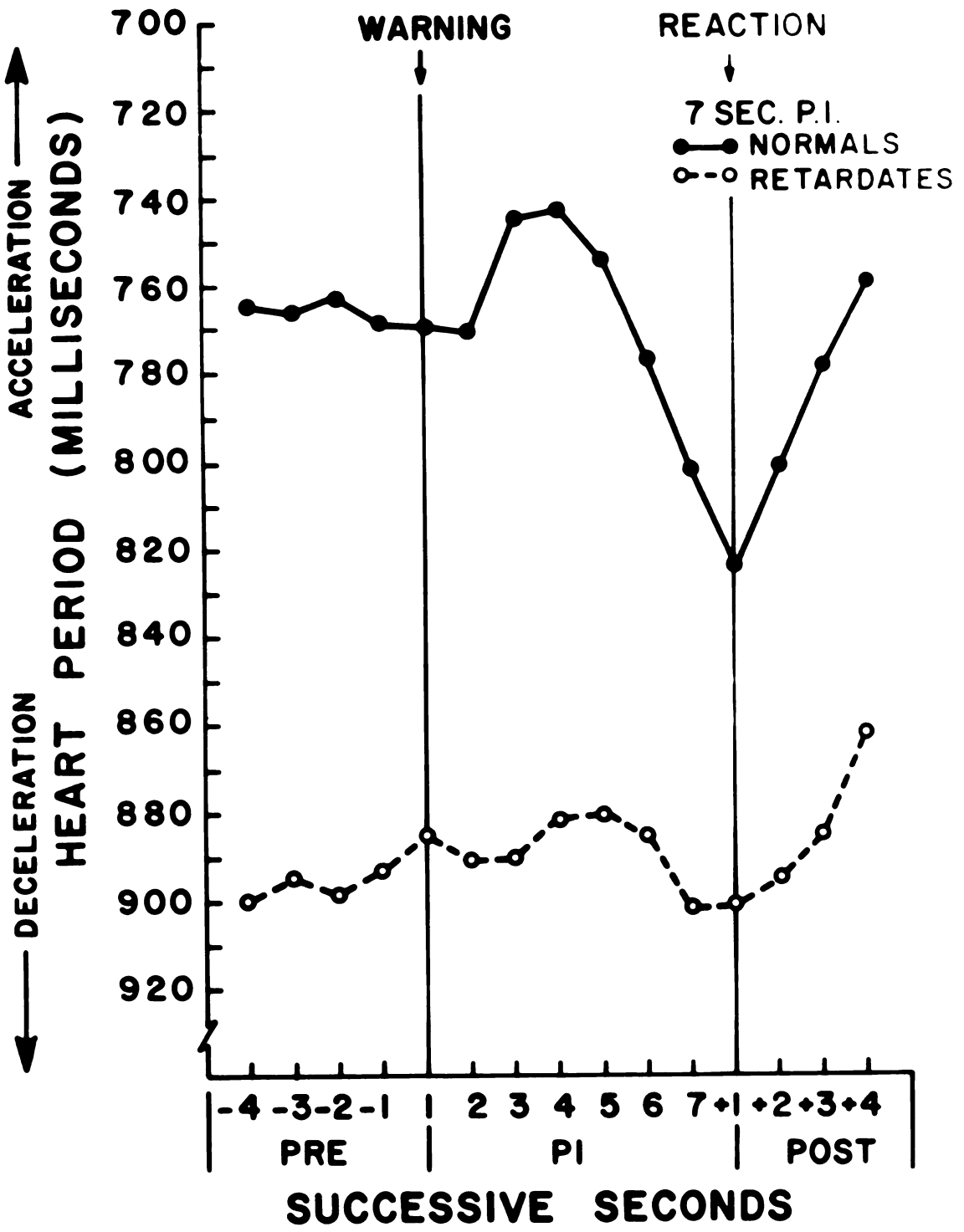
As in the 4-sec PI condition, a significant group

x second interaction was also found in the 7-sec PI condition ($F(14,308)=4.69$, $p < .0005$). This interaction is illustrated in Figure 3. As can be seen from the graph, both normals and retardates showed a significant acceleration following the warning signal (normals: $t(11)=4.82$, $p < .001$); retardates: $t(11)=4.18$, $p < .01$). In the normal group, the acceleration was followed by a dramatic deceleration significantly different from base level (I: $t(11)=6.57$, $p < .001$; II: $t(11)=6.093$, $p < .001$) which reached its nadir simultaneously with the onset of the reaction signal. On the other hand, retardates showed only an attenuated deceleration, not significantly different from base level (I: $t(11)=1.71$, ns; II: $t(11)=0.382$, ns), which reached its nadir one sec before the onset of the reaction signal and was sustained through the sec on which the reaction signal occurred. Following the reaction signal, as in the 4-sec condition, normals showed an acceleration back to prestimulus level, whereas retardates showed an acceleration which exceeded prestimulus levels. The Analysis of variance summary table for the 7-sec PI is presented in Appendix B.

As in previous analyses, the analysis of variance of the 13-sec PI condition revealed significant group differences in overall HP base levels with retardates having significantly slower HR (or, longer R-R intervals) than normals ($F(1,22)=4.91$, $p < .037$). In the 13-sec PI the mean HR of normal Ss was 78 bpm while the mean HR of

FIGURE 3

Mean heart period change as a function of successive seconds in the 7-sec PI condition for normal and retarded groups.

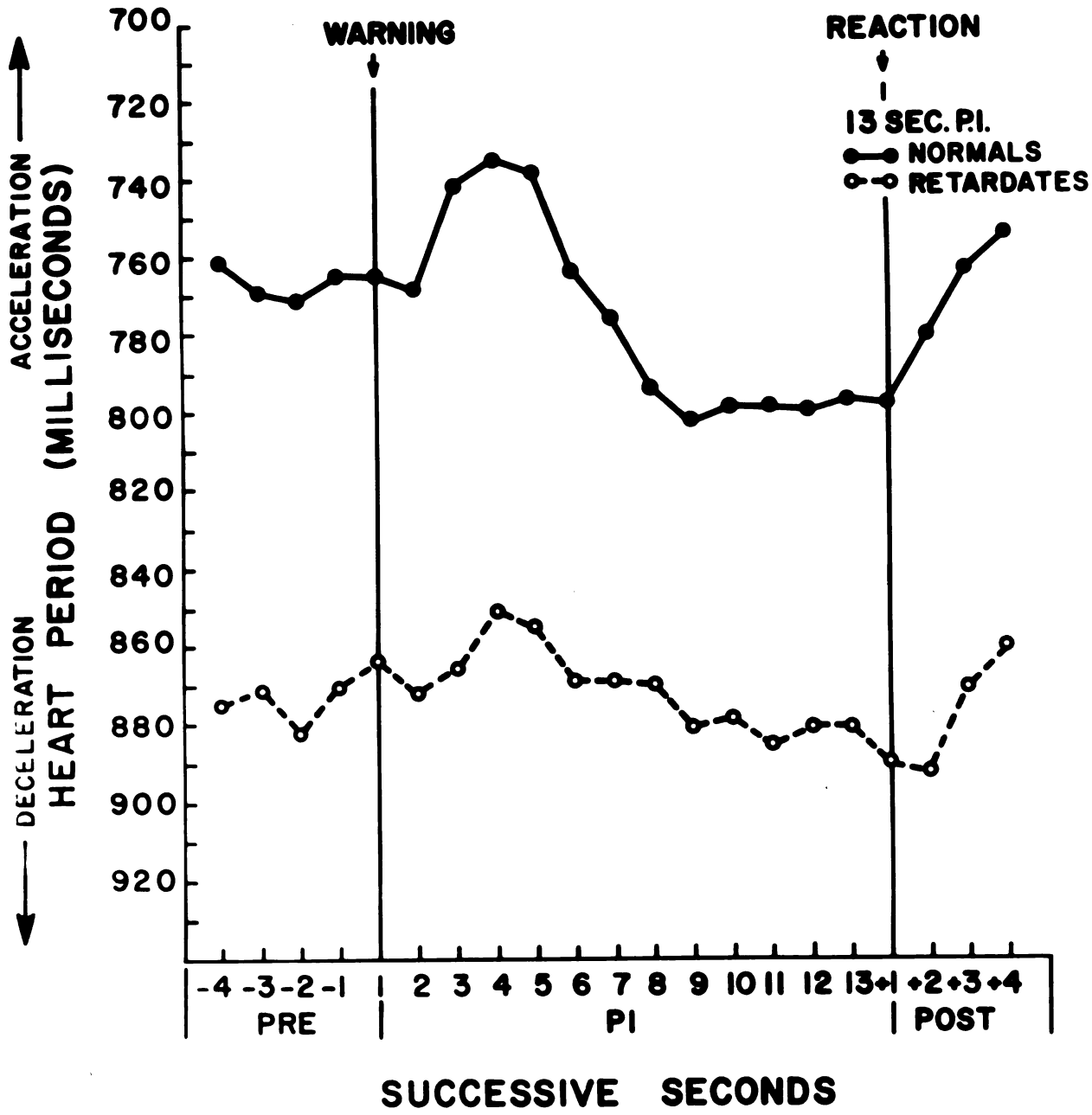

700
720
740
760
780
800
820
840
860
880
900
920
WARNING
REACTION
7 SEC. P.I.
NORMALS
RETARDATES
ACCELERATION
DECELERATION
HEART PERIOD (MILLISECONDS)
-4 -3 -2 -1 1 2 3 4 5 6 7 +1 +2 +3 +4
PRE
PI
POST
SUCCESSIVE SECONDS

retarded Ss was 69 bpm. Also consistent with previous analyses were significant seconds effects ($F(20,440)=14.99$, $p < .0005$) and significant group x seconds effects ($F(20,440)=3.16$, $p < .0005$). Second-by-second HP changes for combined groups, i.e., seconds effects, were characterized by an acceleration beginning 1 sec after the warning signal and peaking on sec 4 of the PI. A deceleration followed which leveled off and became sustained from sec 9 to the end of the PI. Following the reaction signal, HP gradually increased, reaching prestimulus base levels 4-secs after the reaction signal.

The group by second interaction is illustrated in Figure 4. Following the warning signal, the normal group showed a significant acceleration ($t(11)=5.72$, $p < .001$) which peaked on sec 3 of the PI, followed by a significant deceleration (I: $t(11)=7.23$, $p < .01$; II: $t(11)=3.228$, $p < .01$) which leveled off and was sustained from sec 9 to sec 13 of the PI. The reaction signal was followed by a sharp acceleration which reached the prestimulus base level 4 secs after the reaction signal. Retardates also showed a significant acceleration after the warning signal ($t(11)=4.83$, $p < .001$) which was followed by a deceleration that attained statistical significance in analysis I ($t(11)=5.65$, $p < .001$), and approached statistical significance in analysis II ($t(11)=2.890$, $p < .02$). The reaction signal was followed by a slight deceleration and then an acceleration. The analysis of variance summary table for the

FIGURE 4

Mean heart period change as a function of successive seconds in the 13-sec PI condition for normal and retarded groups.

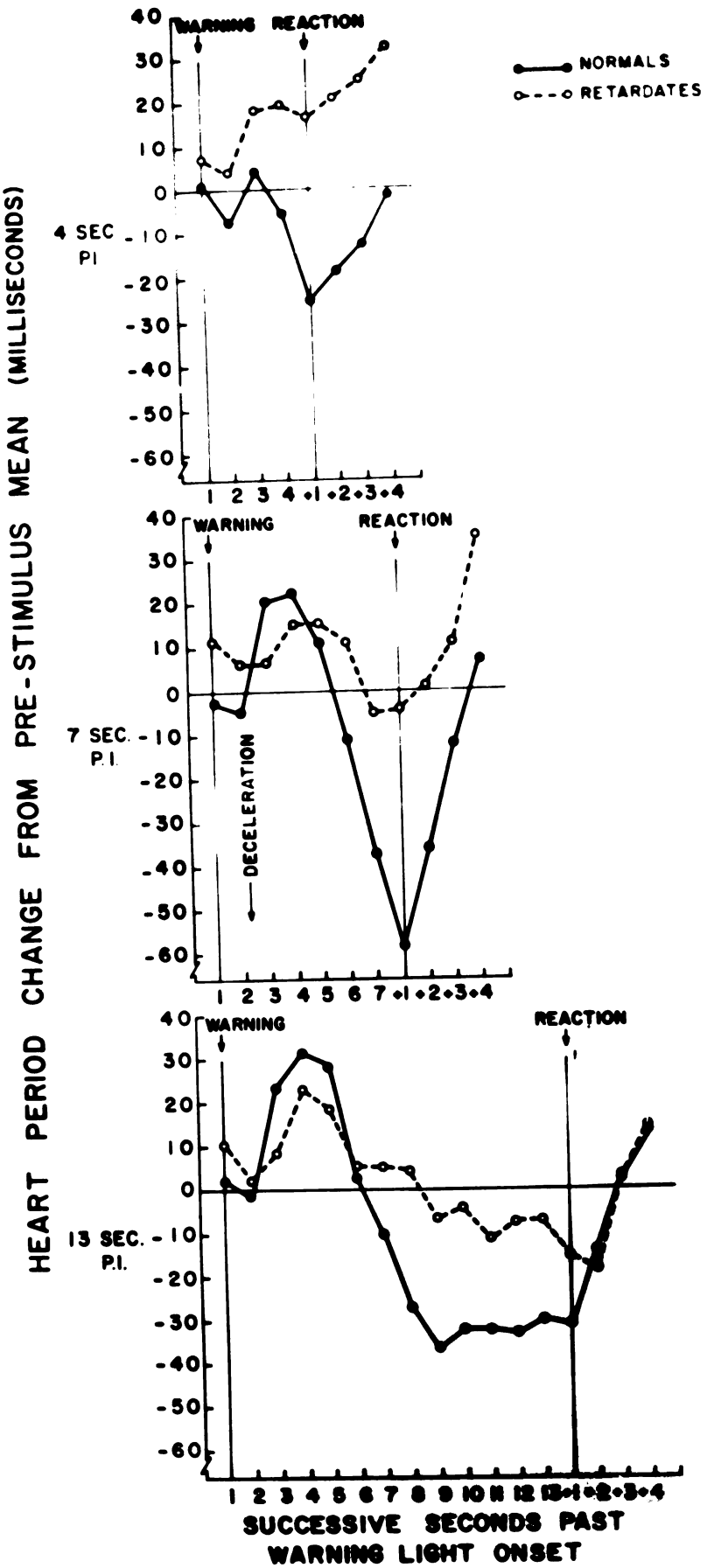

700
720
740
760
780
800
820
840
860
880
900
920
WARNING
REACTION
13 SEC. P.I.
NORMALS
RETARDATES
ACCELERATION
DECELERATION
HEART PERIOD (MILLISECONDS)
-4 -3 -2 -1
PRE
2 3 4 5 6 7 8 9 10 11 12 13+1 +2 +3 +4
PI
POST
SUCCESSIVE SECONDS

13-sec PI is presented in Appendix B.

Figure 5 depicts HP change from prestimulus mean as a function of seconds past warning light onset for each of the 3 PI conditions, for both groups. These are the same data as presented in the previous 3 figures, however, in this case they are plotted as difference scores. Heart period for each second was subtracted from the prestimulus mean to obtain a difference score. As in the previous figures, an upward slope indicates HR acceleration and a downward slope indicates deceleration. These figures clearly show the difference between groups in both response to the warning signal and in anticipatory HP decelerations preceding the reaction signal for each PI condition. As described previously, the statistical significance of responses to the warning signal and in anticipation of the reaction signal were assessed by a t-test for related measures between mean basal HP and acceleration score, and mean basal HP and deceleration scores. Both groups showed HR acceleration in response to the warning signal however, the magnitude of this acceleration differed between groups as a function of PI. Normal Ss showed a non-significant acceleration from prestimulus base level following the 4-sec PI warning signal, a larger and significant acceleration in the 7-sec PI, and a progressively larger and significant acceleration in the 13-sec PI. Retardates, on the other hand, showed about the same magnitude of acceleration in all three PI conditions, all of them being significantly different from prestimulus base levels. Group differences in HP deceleration

FIGURE 5

Mean heart period change from prestimulus mean as a function of seconds past warning light onset in 4, 7, and 13-sec PI conditions for normal and retarded groups.


HEART PERIOD CHANGE FROM PRE-STIMULUS MEAN (MILLISECONDS)
NORMALS
RETARDATES
WARNING
REACTION
4 SEC P.I.
7 SEC. P.I.
13 SEC. P.I.
DECELERATION
SUCCESSIVE SECONDS PAST WARNING LIGHT ONSET

prior to the response signal were also apparent for all three PI conditions. In each case, normal Ss showed significant deceleration just prior to and simultaneous with the onset of the reaction signal; for the retarded group only the deceleration in the 13-sec PI was significant.

Correlations between RT and HP. Pearson product moment correlations between RT and basal HP, RT and HP acceleration score, and RT and HP deceleration scores were performed for each group in an attempt to assess the relationship between RT performance and HP activity. The correlations between RT and basal HP for each PI, and for each group are presented in Table 3. Mean basal HP was

Table 3. Pearson product moment correlations between mean basal heart period and mean reaction time score for 4, 7, and 13-sec PI conditions for normal, retarded, and combined groups.

PI	NORMALS (df=10)	RETARDATES (df=10)	COMBINED GROUPS (df=22)
4	.171	.218	.369#
7	.231	.298	.438*
13	.306	.221	.396##

\# $p < .10$
\#\# $.10 < p > .05$
* $p < .05$

calculated, as before, as the mean HP for the 4-secs prior to PI onset for each S. A positive correlation in this case means that longer R-R intervals, or slower HRs, are related to slower RTs. As can be seen from the table, none of the correlations for the groups are significant. However, when the correlation is performed on the combined groups, it is significant in the 7-sec PI and approaches significance in the 4- and 13-sec PI. These results are consistent with the analyses of variance results which showed that retardates had significantly slower RTs than normals and also significantly slower HRs (or, longer R-R intervals) than did normals.

In the analysis relating HP acceleration following the warning signal to RT, the acceleration score (i.e., the greatest acceleration following the warning signal) was subtracted from the mean basal HP score to get an acceleration difference score. This acceleration difference score was correlated with the mean RT score for each PI, for each group, and for the combined groups. None of these correlations reached statistical significance; they are presented in Table 4.

In the correlational analysis relating RT to the HP deceleration occurring at about the time the reaction signal occurred, HP deceleration difference scores I and II were correlated with mean RT for each PI, for each group, and for the combined groups. The correlations between RT and HP difference scores I and II are presented in Table 5.

Table 4. Pearson product moment correlations between mean HP acceleration score and reaction time for retarded, normal, and combined groups, and 4, 7, and 13-sec PI conditions.

PI	NORMALS (df=10)	RETARDATES (df=10)	COMBINED GROUPS (df=22)
4	-.041	.368	.373
7	-.148	-.100	-.072
13	.518	.047	.015

Table 5. Pearson product moment correlations between heart period deceleration difference scores I and II and mean reaction times for 4, 7, and 13-sec PI conditions for normal, retarded, and combined groups.

I

PI	NORMALS (df=10)	RETARDATES (df=10)	COMBINED GROUPS (df=22)
4	.394	.619**	.613***
7	.524##	.310	.492*
13	.351	-.332	.105

II

PI	NORMALS (df=10)	RETARDATES (df=10)	COMBINED GROUPS (df=22)
4	.534##	.485	.576**
7	.523##	.449	.572**
13	.754**	-.208	.213

.10 < p > .05
* p < .05
** p < .01
*** p < .001

A positive correlation indicates that a large HP deceleration is related to a fast RT. Correlations between RT and HP deceleration difference score I revealed significant relationships between RT and decelerations in the 4-sec PI for the retarded and combined groups, and in the 7-sec PI for combined groups. This means that large decelerations occurring in the 5-secs surrounding the reaction signal were significantly related to faster RTs in the 4-sec PI condition for retardates, and in the 4 and 7-sec PI conditions for combined groups.

Correlations between RT and HP deceleration difference scores II revealed significant relationships between RT and HP decelerations in the 13-sec PI condition for normal Ss and in the 7 and 13-sec PI condition for combined groups. Correlations for the normals in the 7 and 4-sec PI conditions approached significance. These correlations mean that large decelerations occurring on sec +1, or the sec on which the reaction signal occurred, were significantly related to faster RTs in the 13-sec PI condition for the normal group and in the 7 and 4-sec PI conditions for combined groups.

Respiration Frequency. Two of the retardate's respiration records were rendered unscorable because of mechanical difficulties. In order to have equal cell frequencies in the analysis of variance, 10 normals were matched with the 10 retardates on the basis of PI order. Consequently, the respiration frequency analyses of

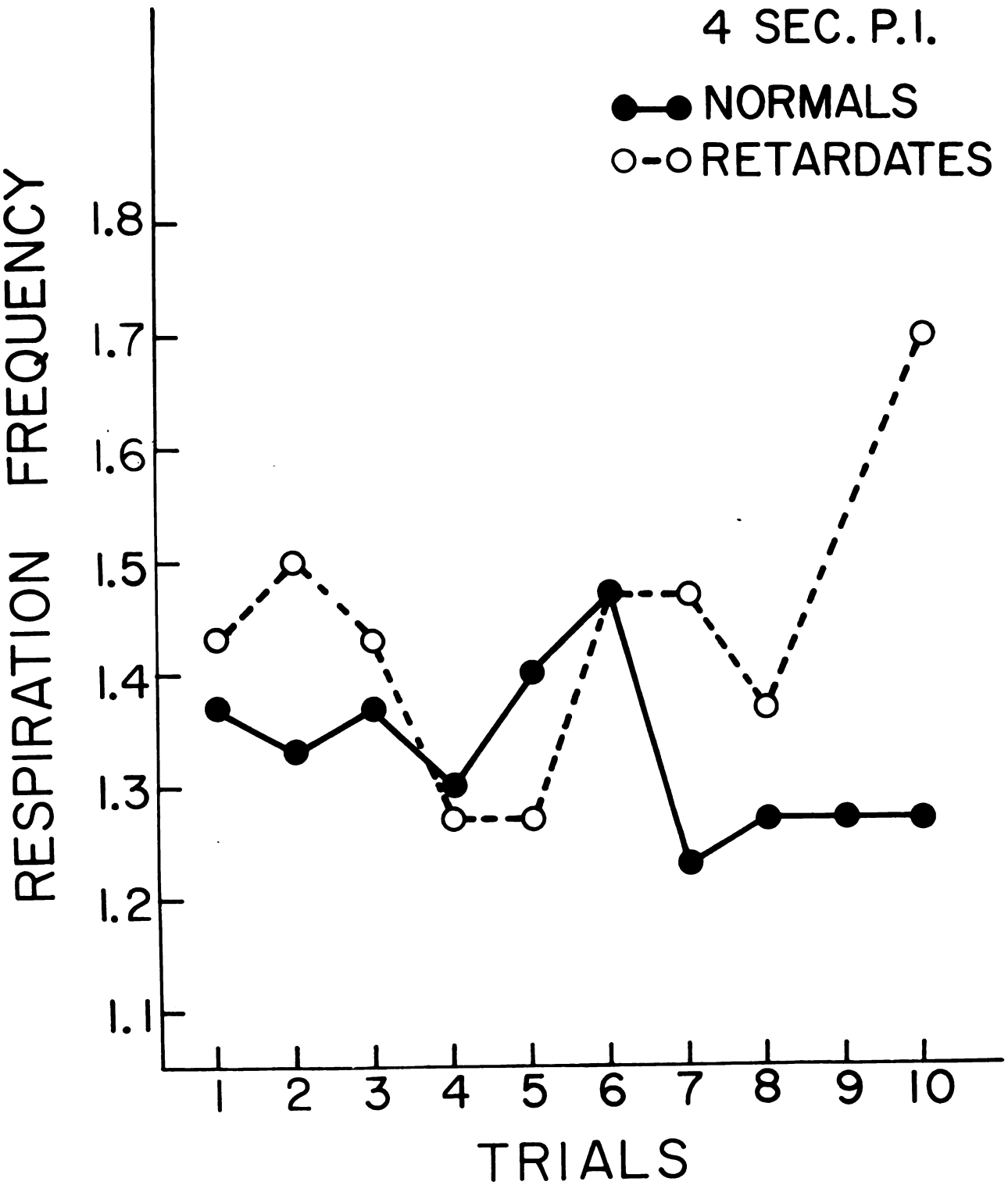
variance used only 10 Ss per group.

Respiration frequency was analyzed in 4 separate analyses of variance for 4, 7, and 13-sec PI conditions. In the 4-sec PI analysis, frequency of initiations and terminations of inspirations were counted during the 4-secs prior to PI onset, the 4-secs during the PI, and the 4-secs following the PI. These periods were labeled "pre", "PI", and "post". As illustrated in Figure 6, the groups x trials interaction was the only significant finding in this analysis ($F(9, 162)=1.991$, $p < .043$). Both normal and retarded groups showed about the same level of respiration activity until trial 7. At this point, the normal group's respiration decreased slightly from initial levels and became sustained for the remaining trials. The retarded group, on the other hand, showed a slight decrease in respiration activity on trial 8, a sharp increase immediately after trial 8, and continued increase across the remaining trials. The analysis of variance summary table is presented in Appendix B.

In the 7-sec PI analysis, the "pre" period consisted of the 7-secs that preceded the PI, the "PI" period consisted of the 7-secs during the PI, while the "post" period consisted of the 7-secs following the reaction signal. The analysis of variance performed on this data only revealed significant trial effects ($F(9,162)=2.046$, $p < .037$); respiration frequency decreased across trials for both groups. The analysis of variance summary table

FIGURE 6

Mean respiration frequency as a function of trials in the 4-sec PI condition for normal and retarded groups.


4 SEC. P.I.
NORMALS
RETARDATES
RESPIRATION FREQUENCY
1.8
1.7
1.6
1.5
1.4
1.3
1.2
1.1
1 2 3 4 5 6 7 8 9 10
TRIALS

is presented in Appendix B.

The 13-sec PI condition was analyzed in 2 ways: In the first analysis, the "pre" period consisted of the 13-secs preceding the light onset, the "PI" period consisted of the 13-secs of light presentation, while the "post" period consisted of the 13-secs following the light. In the second analysis, the 13-sec PI was analyzed with the "pre" period consisting of the 6-secs preceding light onset, the "PI" period was broken down into "early PI" and "late PI" components, with "early PI" consisting of the 6-secs following light onset, and "late PI" consisting of the 6-secs preceding light offset, while the "post" period consisted of the 6-secs following light offset. There were no significant effects in either analysis. The analyses of variance summary tables are presented in Appendix B.

Correlations Between Mean Respiration Frequency and Mean HP. In order to assess the relationship between respiration frequency and heart period, Pearson product moment correlations were performed between mean respiration frequency and mean HP for each group, combined groups, each period, combined periods, and each PI condition. In the 4-sec PI condition, mean respiration frequency was calculated for each period (i.e., "pre", "PI", "post") and for each S. Overall respiration frequency was calculated by recording the mean respiration frequency for all three periods for each S. The same procedure was followed in the 7 and 13-sec PI conditions except that in the 13-sec

PI mean respiration frequency was examined for 4 periods instead of 3.

In all three PI conditions, mean HP for the "pre" period consisted of the mean HP during the 4-secs preceeding the warning signal. In the 4 and 7 sec PI conditions mean HP for the PI period was based on the mean respiration frequency during the PI; for the 13-sec condition, mean HP during the "early PI" period was based on the mean HP during the 6-secs following the warning signal while mean HP during "late PI" was based upon the mean HP during the 6-secs preceeding the reaction signal. In all three PI conditions, mean HP during the "post" period was based on the mean HP during the 4 secs following the reaction signal.

Correlations between mean respiration frequency and mean HP are presented in Table 6. A negative correlation indicates that slower HR (or, longer R-R intervals) are associated with fewer respiration cycles. As can be seen from the table, significant correlations were found for the normal group in the 7 and 13-sec PI overall means, and for normal Ss in the 13-sec "early PI" period.

Table 6. Pearson product moment correlations between mean respiration frequency and mean HP for retarded, normal, and combined groups in 4, 7, and 13-sec PI conditions, and for "pre", "PI", "post" periods, and overall.

4 SEC PI CONDITION

	PRE	PI	POST	OVERALL
NORMALS (df=8)	-.399	-.358	-.103	-.390#
RETARDATES (df=8)	.090	.114	-.052	.060
BOTH (df=18)	.067	.046	-.000	.056

7 SEC PI CONDITION

	PRE	PI	POST	OVERALL
NORMALS (df=8)	-.493	-.627#	-.521	-.612***
RETARDATES (df=8)	-.472	-.232	-.378	-.367
BOTH (df=18)	-.226	-.153	-.177	-.192

13 SEC PI CONDITION

	PRE	ERL PI	LTE PI	POST	OVERALL
NORMALS (df=8)	-.540#	-.638*	-.469	-.364	-.620***
RETARDATES (df=8)	-.418	-.405	-.413	-.301	-.394#
BOTH (df=18)	-.323	-.311	-.226	-.185	-.243

\# $p < .10$
* $p < .05$
** $p < .02$
*** $p < .01$
**** $p < .001$

DISCUSSION

Correlations between IQ and RT. Correlations between Wechsler performance IQ and RT scores were significant for the retarded group. Most other studies reporting significant relationships between intelligence and RT performance have used MA measures rather than IQ (Bensberg & Cantor, 1957; Berkson, 1960; Ellis & Sloan, 1957; Pascal, 1953). The results of the present study suggest that performance IQ might also be a good predictor of RT performance.

Base Level HP. Base level HP was significantly different between the groups in all three PI conditions, with retardates having slower HP, or longer R-R intervals. This represents a discrepency with published data (Clausen & Karrer, 1970; Holloway & Parsons, 1970; Karrer, 1966; Wallace & Fehr, 1970); a discrepency which unfortunately has no obvious explanation since Ss had not taken drugs for at least 2 weeks prior to the experiment. Moreover, no retardated S was institutionalized and almost all were employed at least part-time in the Lansing community. It also seems unlikely that the difference in base level reflects a motivational difference as the personal observations of the experimenter suggest that if anything, retarded Ss were more highly motivated than were the normal

Ss. Almost all of the retarded Ss expressed a great deal of excitement about coming to the University campus and to the laboratory. They also seemed very eager to "please" the female experimenter. For example, during the practice session, these Ss frequently asked if their RTs were "fast enough", and if they were "doing ok". It appeared to the experimenter that frequent comments such as these reflected a high degree of motivation to perform well. One plausible explanation for the slower HR of retardates may lie in the sample used. Most previous studies that reported no differences between base level HP of retardates and normals employed retardates sampled from populations with IQs that were lower than 55. Since none of the retardates in the present sample fell below this IQ level the possibility exists that base level varies as a function of IQ level.

Sec-by-Sec HP: Anticipatory HP Deceleration. The present study found significant second effects, or sec-by-sec HP changes for combined groups, in all three PI conditions. These data will not be discussed in detail here as they are not central to the purpose of this paper. The main interest of the present study was to obtain information on differences between normal and retarded Ss--effects that are interactive in nature. Consequently, main effects such as the seconds effects are only of peripheral interest at this time.

Significant group differences in sec-by-sec HP responding were found in the HP analysis. A dramatic

difference in HP deceleration at about the time the reaction signal was to occur was found between retarded and normal groups. In all three PI conditions, normals showed a deceleration which reached its nadir just prior to or at the time the reaction signal was to occur, while retardate's HP pattern prior to the reaction signal was markedly different: In the 4-sec PI condition retardates showed a complete absence of HP deceleration, while in the 7-sec PI condition their deceleration was severely attenuated. In the 13-sec PI condition retardates exhibited a significant deceleration, but the pattern of this deceleration was very irregular and markedly different in form from the response pattern of normal Ss.

Such data would seem to demonstrate the retardate's inability to prepare for appropriate responding regardless of the length of the preparation time. If one accepts the assumption that HP measures during the PI reflect an attention process, the data clearly support the idea that the retardate suffers from an attention deficit. Although this notion is not a novel one in retardation research, little real empirical evidence has been advanced to support it. Not so incidentally then, the results of the present study underlie the potential value of psychophysiological techniques for studying the capabilities of retardates.

The correlational analyses lend some support to the notion that faster RTs are related to greater HP decelerations which occur prior to and during the reaction

signal. The HP deceleration score II, or the deceleration which occurred on sec +1, was significantly related to RT in the 13-sec PI condition for the normal group, and in the 7 and 4-sec PI conditions for the combined groups. Correlations in the 7 and 4-sec PI conditions also approached significance for the normal group. The specific role that this anticipatory HP deceleration plays in RT performance and attention has been interpreted in several different frameworks.

Lacey (1967), for example, believes that the occurrence of HP deceleration facilitates the intake of information, and hence, prepares the individual to deal with the environment in an efficient manner. In the case of the RT task, the enhanced attention reflected by HR deceleration would enable the organism to respond more quickly. The absence of deceleration in retardates would indicate that retardates are less sensitive to environmental stimuli. In the present study, a lowered sensitivity to the reaction signal would explain the poor RT performance of the retardate.

It is also possible to interpret the role of anticipatory HP deceleration in terms of an inhibition deficit hypothesis such as that advanced by Denny (1964). This interpretation holds that when all task-irrelevant or competing activities, including irrelevant physiological activities, are suspended or inhibited, a fast reaction

time should result. This view is supported by the findings of Obrist who has shown that a number of physiological activities including muscle tension, eye movements, respiration, as well as HR, markedly decrease simultaneously during a RT situation at about the time S is to make a response (Obrist et al., 1969b). The magnitude of these decreases is directly related to RT performance. In this framework, the retardate's primary deficit is explained as an inability to inhibit or suspend activities which do not contribute to good RT performance. In the present study, the absence of HR deceleration prior to the reaction signal can be interpreted to reflect the retardate's deficit in the ability to suspend or inhibit ongoing activities which interfere with the ability to respond quickly.

A third interpretation could be based on the cardiac temporal conditioning notion advanced by Fitzgerald & Porges (1970), Grossman, Fitzgerald, & Porges (1970), and Porges (1970). In this framework, the absence or attenuated anticipatory HR deceleration in the retarded group would reflect an absence of temporal conditioning, whereas the dramatic decelerations which were observed in the normal group would presumably reflect successful temporal conditioning. This notion is not incompatible with Denny's contention that retardates suffer from an inhibition deficit. It could be that retardates are unable to make

use of the temporal cue because they lack the ability to inhibit competing responses. Normal Ss, on the other hand, inhibit competing responses and simultaneously attend to the signal value of temporally fixed stimulus events.

Group x trial x second effects were not significant in the present study, probably because of the deletion of the first 5 trials. These early trials were eliminated to avoid transfer and possibly disruption effects from prior conditions which were different for each S. Consequently, a conditioning interpretation could not be directly assessed from the reported data. However, the possibility of group differences in temporal conditioning suggests future research which would be potentially relevant to the understanding of neural mechanisms in both retarded and normal individuals.

In a literature review on neural timing mechanisms and conditioning, Prescott (1966) points out that use of the classical conditioning paradigm in studying the precision of neural timing mechanisms provides a valuable technique for research in developmental processes. He suggests that an organism capable of inhibiting a response until the exact moment of reinforcement would reflect a neural system characterized by high precision and efficiency. Increased error in timing, on the other hand, would reflect a poorly integrated and biologically nonadaptive neural system. Hence, it appears as though future

research designed to investigate differences in neural timing mechanisms through the established conditioning paradigms would have important developmental implications. In addition to Prescott, a number of other investigators (Brackbill & Fitzgerald, 1969; Fitzgerald & Brackbill, 1971; Fitzgerald & Porges, 1971) have made a similar case for the use of classical conditioning to study developmental phenomena including temporal.

Sec-by-Sec HP: Acceleration following the Warning Signal. Another important group difference in sec-by-sec HP responding was in response to the warning signal. Onset of the warning light is an important signal to S as it indicates the beginning of a trial; this is the point at which S is instructed to begin paying attention. Both groups primarily showed HP acceleration in response to the warning light; however the magnitude of this acceleration differed for groups as a function of PI.

Normals responded to the warning signal differently for each PI, with progressively greater accelerations for the longer PIs. Retardates, on the other hand, showed the same magnitude of acceleration for each PI. This is an interesting difference as the warning signal onset was identical during each PI; the only difference between PIs was the length of time that the light remained illuminated. These data lend support to a contention made by Baumeister & Kellas (1968) who tentatively hypothesized that retardates as a group have an inappropriate response set in the RT

situation such that their attention is directed toward the reception of stimuli rather than performance of the response, in spite of instructions designed to minimize such a set. Normals, on the other hand, maintain a response set as long as the stimulus intensity is above some minimum value. The appearance of a large acceleration at the onset of stimulation in the retarded group would seem to support this hypothesis.

Denny's inhibition-deficit hypothesis (Denny, 1964) would extend the Baumeister & Kellas notion by suggesting that these data reflect the greater ability of normals to inhibit competing responses which interfere with a fast RT. For example, in the 4-sec PI, normals showed a nonsignificant acceleration following the warning signal. In this short PI a large acceleration and its recovery would interfere or compete with the relevant response--which is anticipatory HR deceleration. In longer PIs, where there is adequate time for larger accelerations and their recovery, the normals exhibited significantly greater accelerations. This is not the case with the retarded Ss who showed similar magnitude accelerations following the warning light in all 3 PI conditions. In the 4-sec PI, retardates showed a significant acceleration which was sustained for the entire PI. Hence, this is a second piece of evidence supporting the idea that retardates are unable to inhibit task-irrelevant or competing responses.

Another way of looking at this early acceleration which follows the warning signal is in terms of Ss perceiving the environmental demands of the situation. The 4, 7, and 13-sec PI vary in the amount of effort needed to sustain attention, with the greatest amount of effort needed to sustain attention for 13-sec and the least amount of effort needed to sustain attention for 4-secs. If this is the case, the small acceleration in the normal group to the 4-sec PI warning signal would reflect the relatively small amount of effort needed for fast RT performance. Progressively greater accelerations in the longer PIs would presumably reflect the higher instrumental demands of these situations. In the case of the normal Ss, then, onset of the warning light not only signals that they must attend, but also signals the degree of effort needed to attend. This interpretation lends considerable support to the conclusions of Chase et al. (1968) and Porges (1970).

The consistent response of retardates in the present study indicates that they are perceiving the warning light. However, the lack of any differential responding to light onset as a function of PI length suggests that they are not relating onset of the light to its subsequent length and consequent task difficulty. This apparent lack of association between differing environmental demands and HR change supports Holloway & Parsons (1970) notion that some groups of retardates suffer from a disruption of the integration between somatic and autonomic activity. They

propose that physiological responsiveness in these retardates becomes dissociated from environmental demands and that such dissociated activity reflects a source of interference with Ss ability to attend to external signals or to efficiently execute the appropriate response.

Sec-by-Sec HP: Overall Response Patterns. A striking feature of the sec-by-sec HP data is that as PI interval increases, the HP pattern of the retarded group comes to more closely approximate the pattern of the normal group. In the 4-sec PI condition, the group curves are quite disparate, with the retardates exhibiting a progressive acceleration and the normal Ss exhibiting a triphasic pattern: deceleration followed by acceleration and a final deceleration. In the 7-sec PI condition, there was more overlap between the curves, but there was a sharp divergence between them at about the time the reaction signal occurred. At this point the normal group exhibited a sharp deceleration while the retarded group exhibited an attenuated deceleration. In the 13-sec PI condition, the group curves overlapped for the first 6-secs of the PI and then diverged, but to a lesser extent than in the shorter PI conditions. The HP pattern of the normal group at the point of divergence was characterized by a large, sustained deceleration. The HP pattern of retardates at this point was one of progressive and significant deceleration, but a smaller deceleration than the normal's and one which lacked the smooth, non-variable appearance of the

normal's pattern.

The progressive resemblance of the retardate's HP pattern to that of the normal's as a function of PI can be explained in several ways. One explanation is that retarded Ss need a longer PI in order to recover from the larger acceleration they exhibit and to show the subsequent deceleration which characterizes the normal's HP pattern. A short PI would not provide adequate time for recovery, but as the time interval increased, recovery from acceleration could occur making it possible to be followed by a deceleration. Or, in the Baumeister & Kellas (1968) framework, a longer PI would provide the opportunity for the retardate to transfer his attention from the reception of stimulation to the execution of a response.

Perhaps another alternative is that it simply takes longer for the retardate to integrate incoming information. In the longer PI, the longer time interval would allow the retarded S more time to integrate and process information.

In any case, the progressive similarity in HP patterns between the two groups as a function of longer PIs is suggestive of future research which would potentially contribute some meaningful information about retardate processes. Systematic studies which explore the conditions under which retardate HP patterns resemble normal patterns could have important implications for understanding the learning process in retardates. For

example, it could be that optimal learning in retardates requires a sustained stimulus beyond some minimal time limit. In the present study RT performance did not interact with PI length, but as mentioned previously, the design of this study was not suitable for the assessment of learning effects. Also, a classical conditioning paradigm might be more suitable for this kind of question. Perhaps a study employing many trials and independent groups in several conditions would provide a more direct evaluation of these hypotheses.

Respiration Frequency. Analyses of variance of the respiration frequency data did not reveal group differences corresponding to group differences found in the HP analyses. In the respiration frequency analyses, only a trial x group interaction in the 4-sec PI and a trials main effect in the 7-sec PI condition attained statistical significance. This absence of any group or period differences suggests that respiration frequency measures are less sensitive to environmental demands than are HR measures.

Correlations between mean HP and mean respiration frequency revealed significant relationship between these measures for normal Ss in the 7 and 13-sec PI conditions. Two of the three significant correlations were in overall means and not specific periods, which is similar to the analysis of variance results.

Conclusion. The present study succeeded in

finding significant differences between retardated and normal individuals in both RT performance and in sec-by-sec HP changes--two measures which have often been associated with the attention process. Thus, this study provides support to the notion that retardates suffer from an attention deficit. An attempt was made to relate the observed differences to a broader theoretical conception of the attention process. A number of diverse interpretations were discussed, all of them being feasible explanations of the reported data. Of course, the number of these explanations and their diversity both reflects the level of understanding that exists regarding the attention process and also points to the need for systematic investigations for greater understanding of these processes. Suggestions made in the discussion were aimed at providing direction for future research in an attempt to accomplish this goal. Implied in these suggestions is the potential value inherent in employing retarded Ss in such investigations. The present study demonstrates that the inclusion of retarded individuals in the study of the attention process provides much needed empirical data upon which future theoretics can be based.

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APPENDICES

APPENDIX A

INSTRUCTIONS FOR REACTION TIME TASK

The pickups I have attached will record changes in heart rate, sweating, and also changes in the blood vessels in your finger. The elastic band around your chest records changes in your breathing. These electrodes are very sensitive so please try to keep movement at a minimum. Get comfortable before the experiment begins.

This green light (E points to green light) will come on periodically. Your job will be to watch this green light (E continues to point to light) and to press the key when the light goes off. Try pressing the key now. It is important that you press the key as rapidly as possible when the green light disappears. Be sure not to press the key before the light goes off. Remember not to make any unnecessary movements and to respond as rapidly as possible when the green light (E points to green light) disappears.

You will receive 2 rest periods during the experiment. During these rest periods you can just relax. They will be designated by the white light (E points to white light). When this white light (E continues to point to the white light) goes on it is a rest period and you can just relax.

We will begin with a series of practice trials. During these practice trials you can ask questions by simply talking in a normal tone of voice. I will be able to hear you in the next room and give you an answer. I will tell you when the practice trials are over and the

real test is about to begin.

Remember, your job is to press this key (E points to key) as quickly as possible when the green light disappears. IT IS IMPORTANT THAT YOU REMEMBER THIS IS A TEST OF SPEED!

Do you have any questions?

If not, we will begin the practice trials.

APPENDIX B

ANALYSIS OF VARIANCE SUMMARY TABLES

Summary of the analysis of variance of reaction time as a function of groups, trials, and PI conditions (4, 7, and 13-secs).

SOURCE	SS	df	MS	F	p
Groups	3317865.800	1	3317865.800	17.789	<0.0005
PI	230030.486	2	115015.243	0.617	0.543
Group x PI	41197.858	2	20598.929	0.110	0.896
Error	12310136.183	66	186517.215		
Trials	136329.939	9	15147.771	1.044	0.404
Group x Trials	68170.311	9	7574.479	0.522	0.860
PI x Trials	148320.986	18	8240.055	0.568	0.923
G x PI x T	235286.947	18	13071.497	0.901	0.578
Error	8621826.819	594	14514.860		
Total	25109165.328	719			

Summary of the analysis of variance of heart period as a function of groups, trials, and seconds for the 4-sec PI condition.

SOURCE	SS	df	MS	F	p
Groups	72631.378	1	72631.378	4.201	0.053
Error	380355.619	22	17288.892		
Trials	836.855	9	92.984	0.791	0.625
Group x Trials	1142.743	9	126.971	1.080	0.379
Error	23276.860	198	117.560		
Seconds	940.076	11	85.461	1.919	0.038
Group x Seconds	2065.359	11	187.760	4.216	<0.0005
Error	10777.440	242	44.535		
Trials x Seconds	2604.566	99	26.309	0.927	0.682
G x T x S	1908.311	99	19.276	0.679	0.993
Error	61838.165	2178	28.392		
Total	558377.372	2879			

Summary of the analysis of variance of heart period as a function of groups, trials, and seconds for the 7-sec PI condition.

SOURCE	SS	df	MS	F	p
Groups	123575.684	1	123575.684	5.778	0.025
Error	470538.271	22	21388.103		
Trials	2923.360	9	324.818	2.502	0.010
Groups x Trials	1635.582	9	181.731	1.399	0.190
Error	25707.618	198	129.836		
Seconds	7042.416	14	503.030	10.968	<0.0005
Groups x Seconds	3011.849	14	215.132	4.691	<0.0005
Error	14126.362	308	45.865		
Trials x Seconds	3663.740	126	29.077	1.032	0.389
G x T x S	3060.218	126	24.287	0.862	0.862
Error	78124.082	2772	28.183		
Total	733409.183	3599			

Summary of the analysis of variance of heart period as a function of groups, trials, and seconds for the 13-sec PI condition.

SOURCE	SS	df	MS	F	p
Groups	125790.087	1	125790.087	4.914	0.037
Error	563109.346	22	25595.879		
Trials	704.647	9	78.294	0.482	0.885
Group x Trials	1473.458	9	163.718	1.009	0.434
Error	32138.690	198	162.317		
Seconds	11268.616	20	563.431	14.992	<0.0005
Group x Seconds	2376.700	20	118.835	3.162	<0.0005
Error	16535.846	440	37.581		
Trials x Seconds	5578.840	180	30.994	1.145	0.094
G x T x S	5323.629	180	29.576	1.093	0.193
Error	107189.035	3960	27.068		
Total	871488.900	5039			

Summary of the analysis of variance of respiration frequency as a function of groups, trials, and periods in the 4-sec PI condition.

SOURCE	SS	df	MS	F	p
Groups	2.042	1	2.042	1.455	0.243
Error	25.257	18	1.403		
Trials	2.216	9	0.246	1.211	0.291
Groups x Trials	3.642	9	0.404	1.991	0.043
Error	32.910	162	0.203		
Periods	3.000	2	1.500	2.725	0.079
Groups x Periods	0.053	2	0.027	0.048	0.953
Error	19.813	36	0.550		
Trials x Periods	5.200	18	0.289	0.994	0.466
G x T x P	5.813	18	0.322	1.112	0.339
Error	94.120	324	0.290		
Total	194.065	599			

Summary of the analysis of variance of respiration frequency as a function of groups, trials, and periods in the 7-sec PI condition.

SOURCE	SS	df	MS	F	p
Groups	9.375	1	9.375	1.494	0.237
Error	112.950	18	6.275		
Trials	4.875	9	0.542	2.046	0.037
Groups x Trials	2.875	9	0.319	1.207	0.294
Error	42.883	162	0.265		
Periods	1.030	2	0.515	1.263	0.295
Groups x Periods	0.090	2	0.045	0.110	0.896
Error	14.680	36	0.408		
Trials x Periods	4.770	18	0.265	0.971	0.493
G x T x P	7.710	18	0.428	1.5700	0.066
Error	88.387	324	0.273		
Total	289.625	599			

Summary of the analysis of variance of respiration frequency as a function of groups, trials, and "pre", "PI", and "post" periods in the 13-sec PI condition.

SOURCE	SS	df	MS	F	p
Groups	4.335	1	4.335	0.367	0.552
Error	212.497	18	11.805		
Trials	4.948	9	0.550	0.953	0.481
Groups x Trials	4.148	9	0.461	0.799	0.618
Error	93.470	162	0.577		
Periods	0.663	2	0.332	0.671	0.517
Groups x Periods	0.610	2	0.305	0.617	0.545
Error	17.793	36	0.494		
Trials x Periods	7.437	18	0.413	0.791	0.711
G x T x P	6.357	18	0.353	0.676	0.834
Error	169.140	324	0.522		
Total	521.398	599			

Summary of the analysis of variance of respiration frequency as a function of groups, trials, and "pre", "early PI", "late PI", and "post" periods in the 13-sec PI condition.

SOURCE	SS	df	MS	F	p
Groups	4.061	1	4.061	0.954	0.342
Error	76.653	18	4.258		
Trials	1.051	9	0.117	0.361	0.952
Groups x Trials	2.401	9	0.267	0.825	0.594
Error	52.373	162	0.323		
Periods	0.794	3	0.265	0.718	0.545
Groups x Periods	0.704	3	0.235	0.637	0.594
Error	19.878	54	0.368		
Trials x Periods	6.994	27	0.259	1.054	0.392
G x T x P	8.484	27	0.314	1.279	0.160
Error	119.398	486	0.245		
Total	292.789	799			