HUMAN REACTION TIME IN RELATIONSHIP TO PHASIC OCCURRENCES IN THE HEART AND RESPIRATORY CYCLES

by

Catherine Mary Shisslak

A Thesis Submitted to the Faculty of the DEPARTMENT OF PSYCHOLOGY In Partial Fulfillment of the Requirements For the Degree of MASTER OF ARTS In the Graduate College THE UNIVERSITY OF ARIZONA 1975
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SIGNED: Catherine Mary Shillakin

APPROVAL BY THESIS DIRECTOR

This thesis has been approved on the date shown below:

NEIL R. BARTLETT
Professor of Psychology

Date November 14, 1974
ACKNOWLEDGMENTS

Acknowledgment is made to Dr. Neil R. Bartlett, Thesis Chairman, for suggesting and directing this project and for many helpful contributions given during each stage of its completion and to Dr. Cecil A. Rogers and Dr. Robert W. Lansing for their valuable suggestions and supervision throughout construction of the research project and preparation of the manuscript.

Many thanks are also due to Dr. Alice F. Chang who agreed to replace Dr. Cecil A. Rogers, upon his departure, on the thesis committee and whose constructive suggestions helped in the final preparation of the manuscript.

The author wishes to express appreciation to Mr. Louis A. Pfautsch for his advice and construction of the research apparatus.
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ABSTRACT

Simple reaction times to visual stimuli occurring randomly at different phases of both the cardiac and respiratory cycles were investigated in five college students. The Ss were required to respond to a ready signal to initiate the 21 sec. reaction time interval. This response initiated a randomly varied foreperiod ending in warning signal flash. Following a second randomly varied foreperiod and concomitant with warning signal offset a stimulus light flashed to which the S responded to as rapidly as possible by pressing the response key. In relationship to their speed and occurrence in different phases of both the cardiac and respiratory cycles, 480 reaction times were analyzed. The results show no significant differences between phases of the heart, a significant difference between phases of respiration and a significant heart x respiration interaction. Holding heart phase constant, the interaction reveals the fastest mean reaction time at inspiration and the slowest mean reaction time at expiration, both occurring during heart phase II. It is argued that the nature of this interaction is complex and theories to account for cardiac influence on reaction time via blood pressure variance do not reflect the highly specific nature of this phenomena. The data do suggest that statements involving heart phase influence on reaction time must be qualified by statements of respiratory phase and vice versa. The data suggest consideration of these two autonomic events as a possible source of variance in reaction time studies.
CHAPTER 1

INTRODUCTION

Periodic changes of many physiological and behavioral events during the cardiac cycle have led to investigations of the cardiovascular system in relation to sensory-motor activities. Using the simple reaction time paradigm to relate performance in sensorimotor and perceptual-cognitive motor tasks to spontaneous autonomic fluctuations of the heart, investigators have found significant relationships. Lacey (1967), Obrist, Webb, and Sutterer (1969), Webb and Obrist (1970), and Lacey and Lacey (1970) have found consistent heart rate changes occurring during performance of certain reaction time tasks. The nature of the predominant change was heart rate acceleration at the onset of the warning signal and subsequent deceleration prior to the termination of the randomly varied foreperiod ending in stimulus onset. This deceleration has been found to correlate at a modest level with reaction time, the greater the cardiac deceleration, the faster is the reaction time. Theorized models to account for these heart rate fluctuations have included treating cardiac deceleration as evidence of a feedback mechanism. Attentional demands, inferred to be occurring in reaction time performance, influence cortical arousal via baroreceptor activity which is reduced during deceleration (Lacey 1967; Lacey and Lacey 1970). In this feedback model cardiac activity plays an integral role in influencing motor discharge. An alternative explanation is the
cardiac-somatic model which formulates cardiac deceleration as part of an overall inhibitory response related to other somatic events (Webb and Obrist 1967; Obrist 1968; Obrist, Webb and Sutterer 1969; Webb and Obrist 1970). These responses function to eliminate unnecessary activity to allow optimal motor discharge. In general these models differ in that the latter does not conceptualize the cardiac response as the integrating activity but as one aspect of the overall motor response. An important feature of both these models is the inference that cardiac activity plays an influential role in affecting the nature of motor output.

Heart rate variability is another aspect of the spontaneous fluctuations of the heart that has been found to be related to motor activity in a reaction time task. Forges (1972, 1973) has regarded heart rate variability as reflecting changes in attentional demands during reaction time tasks. He has found heart rate variability significantly related to reaction time in the variable but not the fixed foreperiod condition. He hypothesizes that greater attentional demands existed in a variable foreperiod situation, indicating the sensitivity of heart rate variability to differential task demands.

The purpose of the present study was to consider still another component of the spontaneous cardiac fluctuation, phases of the heart cycle as an influential activity in psychomotor response shown in the speed of simple reaction time. Lacey and Lacey (1958) noted that spontaneous fluctuations of the heart, as shown in heart rate variability, were related to speed of reaction time. Possibly heart fluctuations
affect reaction time from systolic blood pressure changes within the heart cycle, which in turn affect cortical activity via baroreceptors along the arterial pathway. Evidence shows that specific baroreceptors, in particular the carotid sinus, does have an inhibitory effect on some central nervous system functions as noted in the following experiments. Koch (see Heymans and Neil 1958) by directly stimulating the carotid sinus nerve, or increasing pressure within the carotid sinus itself, produced decreased muscle tone in anesthetized animals. Bonvallet, Dell and Hiebal (1954) showed a change in cortical electrical activity, from low-voltage fast activity to high-voltage slow activity, by distention of the carotid sinus in animals. These same authors also showed a direct neural inhibitory effect on an evoked monosynaptic reflex by increasing pressure within the carotid sinus. Baroreceptors discharge at an increased rate when the pressure in them rises and tend to have an inhibitory influence on the central nervous system. Lowering pressure in the carotid sinus causes increased discharge of activity of sympathetic fibers and discharge is reduced as long as arterial pressure is elevated (Downing and Siegal 1963). Afferent pathways from arterial baroreceptors pass via glossopharyngeal and vagus nerves to depressor portions of the vasomotor and cardio-inhibitory centers. Impulses generated in these baroreceptors inhibit tonic discharge of the vasoconstrictor nerves and excite the cardio-inhibitory center causing vasodilation and bradycardia. In the human heart, blood pressure changes, within a certain range of mmHg, in one heart cycle, Fig. 1. This fluctuation in blood pressure, associated
Events of the cardiac cycle at a heart rate of 75 beats/min.
The phases of the cardiac cycle consisted of the following: 1, atrial systole; 2, isometric ventricular contraction; 3, ventricular ejection; 4, isometric ventricular relaxation; 5, ventricular filling. Redrawn from Ganong (1973) p. 410.
with baroreceptor activity, thus can be said to occur from beat to beat. Studies were initiated to determine if these sequential changes in blood pressure occurring during the phases of the cardiac cycle influenced motor speed.

Birren, Cardon and Phillips (1963) arbitrarily divided the heart cycle into four phases and found that reactions to auditory stimuli occurring during the P-phase (see Fig. 2) of the cardiac cycle were significantly faster than reactions to stimuli occurring in subsequent phases; however the use of fixed foreperiods—1 sec.—and fixed intertrial intervals—2 secs.—might have permitted some temporal conditioning to occur (Porges 1972, 1973). The possibility of predictability and consequent autonomic and motor conditioning in a fixed foreperiod condition will be treated further in the discussion section.

Callaway and Layne (1964) presenting light flashes within ten varying intervals of time after the EKG Q-wave, with no warning signal, found reaction time significantly faster in the latter half of the cardiac cycle. In a warning signal condition, three pulse beats, similar results were obtained. Callaway (1965) has proposed an autonomic cardiovascular cycle theory to account for these changes. The idea was that conditions of blood pressure fall after mechanical systole with discharges from the carotid sinus and other pressor receptors decreasing and resulting in speeding of reaction time. Callaway (1965) proposes that the P-phase is the time of minimal autonomic afferent discharge. Also, with electrical systole, a series of inhibitory discharges impinge on the medulla and peak after the carotid
Fig. 2. The Cardiac Cycle

pulse, falling to a minimum just before the next systole. These inhibitory waves, then, retard in some manner effector motor activity. Callaway and Layne (1964) summarize this cardiac influence by regarding the "early portion as the low arousal phase and the latter portion as the high arousal phase." (The adequacy of the term "arousal" as used to relate motor readiness in a reaction time task will be considered in the discussion section.) Thus, speed of reaction time in relationship to cardiac phase is accounted for by the rising and falling in blood pressure via baroreceptor stimulation and altered state of arousal.

The present study investigated this phasic influence on reaction time by dividing the heart cycle in five phases (see Fig. 2) in order to reflect relevant pressure changes that might be influencing motor response. Systolic pressure fluctuations might contribute to baroreceptor stimulation along the arterial pathway. In particular, aortic and left ventricular pressure influences and their occurrence in relation to cardiac phase may shed new light upon phasic influences via pressure changes.

Respiration is another cyclic event which has important inter­relationships with the cardiac cycle (known as respiratory sinus arrhythmia). Because of pressure changes related to respiratory phase and possible interaction of these components with cardiac phase, respiration was monitored as well.

Regarding respiration as consisting of two phases, inspiration and expiration, various investigators have concluded inhibitory and facilitory components of both phases on motor responses in reaction
time studies. Buchsbaum and Callaway (1966) measured spontaneous respiratory cycles while S's responded to auditory stimuli. At each 10th heartbeat the Q-wave of the EKG started a fixed delay of either 200 or 400 msecs., each S receiving 100-120 trials with each. Consistently faster reaction times were found during expiration. The authors cite the inspiratory phase as reflecting inhibitory factors which might slow reaction time; vagal fibers discharging on inspiration and decreasing at the start of expiration. They infer stimulation of the vagus supposedly provokes a reflex motor inhibition as shown in slower reaction time. Roitbak, Dedagrishuili and Gotsiridze (1962) had Ss responding in a reaction time task to stimuli presented in either the inspiratory or expiratory phase. No warning signal was given. Reaction time was found to be shorter during inspiration. They concluded that during inspiration, a wave of excitation is discharged through the CNS facilitating reaction time. Similarly Hildebrandt and Engel (1963), with a reaction time task with both auditory and visual signals but no warning signal, found shorter reaction times during inspiration. Thus the evidence on the nature of the effect of phasic occurrences in breathing on reaction time is inconclusive. That respiratory phases influence reaction time by acting on motor rather than sensory systems is suggested by a study by Callaway and Buchsbaum (1965). They found pairs of averaged evoked responses to be most similar when evoked by stimulation at the same phase of both cardiac and respiratory cycles.
It thus appears that stimuli occurring in different phases of both the cardiac and respiratory cycles result in variation in human reaction time. Both cycles have been investigated separately although both occur simultaneously and are not mutually exclusive autonomic events. Therefore the present study monitored both cycles to allow for evaluation of possible interaction effect on reaction time. To control for possible temporal conditioning influence on motor output, the present study used variable foreperiods in a procedure designed to ensure good attention to the signal and readiness to respond.
CHAPTER 2

METHOD

Subjects

Three female and two male graduate and undergraduate students in Psychology volunteered to serve as Ss.

Apparatus

The stimuli were three Tung-Sol A313, 28 volt lamps displayed on a 38.1 x 22.8 cm. backboard and spaced horizontally 8.8 cm. apart. Ss rested on a couch in a supine position and binocularly observed the stimulus flash. Distance between eye and stimuli lamps measured approximately 236.2 cm., so the extent of the visual angle of lamp display was 10°. The stimuli were presented automatically and controlled by two electronic timers. The response key was a common telegraph key which closed when S pressed it. The response key moved down approximately 2 mm before the circuit opened with a pressure weight of approximately 46.6 gm. The telegraph key was mounted to the right side of the couch on a 8.8 x 50.8 cm. board which was adjusted to Ss arm length. A foot pedal was attached to the left corner of the couch and closed when pressed by the left foot. Reaction time was recorded in milliseconds by a Hewlett Packard model 522B Electronic Counter. Cup electrodes were attached laterally approximately at the fifth intercostal space and used to record heart cycle from an EKG lead II. Respiration was monitored using a thermistor attached just inside the right nostril free from skin
Responses were recorded on a Grass model 70b polygraph at a paper speed of 25mm/msec. Resolution was 40 msec/mm. with this method. The first channel recorded the two phases of the respiratory cycle; the second the phases of the heart; the third the occurrence of the stimulus and response; the fourth the occurrence of the warning signal; and the fifth indicated foot switch onset and offset, as shown in Fig. 3.

**Procedure**

The Ss were screened for relevant previous activities that might add variance to reaction time such as recency of alcohol consumption and/or smoking activities. Ss were instructed that their task was to respond as fast as possible to the appropriate signals. For each recording these signals consisted of the flash of the foot switch light (blue light) after which the S responded by depressing the foot pedal when he/she felt fully prepared to respond. Depression of the foot pedal initiated a period of variable duration controlled by timer #1 (1 to 4 seconds). Termination of timer #1 initiated timer #2 (0.18 to 5 seconds)* during which the warning light (yellow light) was illuminated indicating that the stimulus light was about to occur. Concurrent with the termination of the warning signal, the response light (white light) flashed, and S responded by pressing the telegraph key with the right finger as fast as possible, as depicted in Fig. 4. Ss thus responded to two simultaneous events. Fifteen percent of the trials

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*Order of variable foreperiod times for timer #2 were: .186, .614, 4.46, .837, 2.69, .438, 2.25, .333, 3.66, 3.20, 1.55, .998, 1.99, .493, 4.94. Times were approximately in the same order of presentation for timer #1.
Fig. 3. Schematic Display of the Events

Display illustrates mode for selecting trials for analysis
In the 21 sec. Reaction Time Interval

Fits • 4* Evens Occur

BLUE LIGHT ON
OKAY TO PRESS FOOT SWITCH

FOOT SWITCH PRESSED
YELLOW LIGHT ON
TIMER NO. 1 ON

WHITE LIGHT OFF
RESPONSE KEY PRESSED

WHITE LIGHT ON

YELLOW LIGHT ON
TIMER NO. 2 ON

21 SEC.
MAXIMUM

STIMULUS

RESPONSE

WARNING

FOOT SWITCH

READY SIGNAL

Diagram showing the sequence of events with light switches and timers.
consisted of randomly administered trials with the foreperiods extending well with the fixed range so that Ss would not be conditioned to a terminating foreperiod. Since these extra long foreperiods non-terminated, there are no reaction speed data for them.

Each S was pre-trained in the reaction time task for approximately five hours until reaction time variance decreased and stabilized around a standard deviation of 31 msec. Each session consisted of 110 trials, one trial lasting 21 secs. from foot switch offset to the next foot switch onset.

**Data Analysis**

Data used for analysis of cyclic events and reaction time were measured using the stimulus onset point and its occurrence in relationship to respiration and heart cycles, Fig. 4. Each heart cycle was divided into five phases, each representing a separate physiological event; the respiratory cycle was divided into two phases, inspiration and expiration. Results of the measurements of heart phase, respiration, and speed of reaction time were analyzed using a Harvey's least-square ANOVA. A post hoc Neuman-Keuls test was performed on the significant heart X respiration interaction, Fig. 5.
Fig. 5. Reaction Time Means for All Ss

Summary plot of reaction time data and phase. For convenience the cardiac cycle is illustrated.
CHAPTER 3

RESULTS

Heart Cycle

The data revealed that mean reaction times did not differ significantly for the phases of the heart cycle, $F(4, 466) = 0.680$, $p < 0.05$. This may have resulted from the averaging of means across respiration, a procedure which cancelled out significance because of the opposing nature of some of the averaged means.

Respiratory Cycle

The mean inspiratory phase was associated with significantly faster mean reaction times than expiratory phases, $F(1, 466) = 4.48$, $p < 0.05$. Mean reaction times for inspiration were 211.5 msec.; expiration were 221.4 msec.

Heart X Respiration Interaction

A significant heart X respiration interaction was found, $F(4, 466) = p < 0.05$, Fig. 5. This suggests that a statement concerning reaction time in terms of heart phase must be qualified by the particular phase of respiration involved and vice versa. The effect of inspiration and phase of the heart on reaction time appears to be inverted when considering expiration at the same heart phase. Holding heart phase constant, it appears that reaction time is the fastest at phase II during inspiration (200 msec.) but the slowest at expiration.
This opposing trend in speed of response continues in heart phase III although to a lesser extent: inspiration reaction time was 210 msec. and expiration reaction time was 226 msec. At heart phase I this inverted trend changes with inspiratory reaction time 214 msec. and expiratory reaction time 211 msec. Heart phases IV and V reveal this change also with inspiratory reaction time at phase IV 220 msec. and expiratory reaction time 218 msec. and at heart phase V inspiratory reaction time 215 msec. and expiratory reaction time, 217 msec. Thus, as shown in Table I, heart phases I, IV, and V seem to reflect the least degree of inversion with heart phase II followed by heart phase III the most. To test for significant difference among particular means within this set of heart and respiratory phases, a Neuman-Keuls test was performed. Significant difference was found between the following sets of means: heart phase II at inspiration and heart phase II at expiration, $s_2 (6, 466) = 4.36, p < .05$; heart phase II at inspiration and heart phase IV at expiration, $s_2 (5, 466) = 4.39, p < .05$. No significance was found testing sets of means with heart phase III although the difference in mean reaction times among means in this phase at both phases of respiration are greater than differences, proven significant, using heart phase II at both phases of respiration. This might have resulted from an unusually small number of n's in heart phase III at both inspiration and expiration. The nature of this opposing difference in reaction time relative to respiratory cycle suggests the presence of an effect during inspiration and heart phase not occurring during expiration at the same heart phase, or vice versa.
Subjects

All our subjects showed similar patterns in reaction time among the various phases. No significant subject X heart interaction was found, $F(12, 450) = 1.26, p < .01$. Subject X respiration interaction also showed no significance, $F(4, 450) = .447, p < .01$.

TABLE 1

MEAN SIMPLE REACTION TIMES ACCORDING TO PHASE OF CARDIAC AND RESPIRATORY CYCLES

<table>
<thead>
<tr>
<th>Phase of Heart</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
<th>V</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time (msec.)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inspiration Mean</td>
<td>214.9</td>
<td>200.0</td>
<td>210.2</td>
<td>220.7</td>
<td>215.8</td>
</tr>
<tr>
<td>Standard Error</td>
<td>6.0</td>
<td>7.7</td>
<td>9.4</td>
<td>3.8</td>
<td>4.2</td>
</tr>
<tr>
<td>Expiration Mean</td>
<td>211.9</td>
<td>228.7</td>
<td>226.3</td>
<td>218.3</td>
<td>217.8</td>
</tr>
<tr>
<td>Standard Error</td>
<td>5.5</td>
<td>5.6</td>
<td>8.7</td>
<td>3.2</td>
<td>6.1</td>
</tr>
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CHAPTER 4

DISCUSSION

These findings suggest, first of all, that cyclic phases of both respiration and cardiac activity significantly affect sensorimotor activity in a reaction time task, but because of interaction effects statements concerning heart phase influence on reaction time must be accompanied by the particular phase of respiration. These results, in particular, qualify general statements about the cardiac cycle and its influence on sensory-motor activity such as Callaway and Layne (1964) suggest. Our data show that the early portion of the cardiac cycle has unique effects on reaction time. Callaway and Layne (1964) consider this early portion to be a time of "low arousal" with reaction time becoming progressively faster as the cycle goes on. A number of inhibitory activities are said to be occurring during this portion of the cycle. Callaway and Layne (1964) and Callaway (1965) observed EEG alpha to be time-locked 200-250 msec. after the EKG Q-wave. Callaway (1965) notes also that with electrical systole, impulses bombard the CNS and inhibit activity that might influence motor output. He considers this 250 msec. period after the EKG Q-wave a "complex period". Our data confirm that this is indeed a period of complex activity but suggest it is a period of either "high" and "low" arousal, as judged by reaction time, depending on the respiratory cycle. Considering the value of the term "arousal" in implying behavioral arousal, Thompson
(1967) presents evidence that "cortical EEG arousal and behavioral arousal are not concomitant; either can occur without the other" (p. 457). It appears therefore that referring to portions of the cardiac cycle as high arousal or low arousal from EEG electrical patterns is inappropriate. With this in mind our data also indicate that heart phase alone is not a sufficient indicator of relevant chest activity that influences reaction time. Only when heart phase is considered with respiratory phase can a proper statement be made. Heart phase II is not only a time in which speed of response is the slowest, as shown in Birren et al. (1963), but a time when speed of response is the fastest, Fig. 5. This opposing effect is determined by phase of respiration, inspiration at phase II showing activity resulting in the fastest mean reaction time with expiration the slowest. This trend continues to a lesser extent at phase III. It thus appears that monitoring these two cyclic events simultaneously reveals a unique effect on reaction time, one that must be qualified relative to specific phase and not classifiable simply in terms of early or late portions of the cardiac cycle.

It also appears that postulating baroreceptor stimulation and consequent influence on "arousal" and reaction time does not help in interpreting our results. Callaway and Layne (1964) postulate fall in blood pressure during the cardiac cycle effecting pressor receptors and arousal evidenced in speed of response. That their reaction time graphs tend to show faster reaction times as time progresses from the EKG Q-wave is contradictory to ours. A finer look at pressure changes
within the early portion reveals that aortic pressure and left ventricular pressure do not peak until some approximate time after phase II, Fig. 1. Indeed the aortic valve, which releases ventricular blood into the arterial pathways where sinus and aortic baroreceptors exist, does not open until some time approximate to phase III. It appears that phase I and II, relative to sinus and aortic baroreceptors, are phases of minimal afferent effect and not just phase I as Callaway (1965) proposes. Pressure must usually rise above 50 mmHg to stimulate sinus baroreceptor activity and left ventricular pressure does not surpass this threshold until some time approximately after phase II. The carotid pulse also does not peak until at about phase IV, Fig. 1. Thus it appears doubtful that the cardiovascular cycle theory can be invoked in accounting for pressure changes influencing reaction time especially in the early portion of the cardiac cycle. If this were applicable phase III and IV should be the time of slowest reaction time. This is not the case in our data. By ignoring inspiration, expiration might follow the patterns observed in reaction time speed in the three studies by Birren et al. (1963), Callaway and Layne (1964), or Callaway (1965), however our interaction directs attention to both respiratory phases. Our contradictory findings may result from two points. One, baroreceptor stimulation is an inadequate hypothesis for explaining reaction time in relationship to cardiac cycle when respiration is monitored and its influential activity measured, as discussed above. An alternative reason is the artifacts that fixed foreperiods may uncontrollably add in reaction time paradigms. Porges (1972) used
both variable and fixed foreperiods in a visual reaction time task. In the fixed foreperiod condition he found evidence for temporal conditioning of motor responses necessary in the reaction time task. Performance improved in the first half of the trials but not the second half. Also for the fixed group, none of the heart rate responses were significantly correlated with reaction time. Porges relates that heart rate variability and reaction time may be obscured by motor responses that have been temporally conditioned. In another study Porges (1973) found similar temporal conditioning in a fixed foreperiod condition in a visual reaction time task. Only in the variable foreperiod condition did a significant relationship exist between heart rate variability and reaction time. He notes that unpredictability obscured in the fixed foreperiod groups may have resulted in this lack of significance and it is this signal unpredictability which enables autonomic lability. Also, superior performance was shown in the fixed group, this again perhaps resulting from predictability. It is offered that temporal conditioning relative to heart and respiratory phase may confound the true nature of motor speed in similar fixed foreperiod conditions. Birren et al. (1963) used fixed foreperiods of 2 seconds duration with a fixed intertrial interval of 1 second duration. This may have resulted in some temporal conditioning and possibly account for their results being contradictory to ours. Callaway and Layne (1964) utilized 10 varying foreperiods administered after the EKG Q-wave. Although temporal conditioning cannot be said to be occurring another reason for their results contradicting ours is offered. Our data
showed a significant interaction between respiration and heart phase. Perhaps reaction time was confounded as heart phase varies with respiration phase and heart rate also varies with respiration.

Another point to be brought up concerns the observation of alpha being time locked 200-250 msec after the EKG Q-wave (Callaway and Layne 1964; Callaway 1965). Thompson and Botwinich (1966) showed in a reaction time task using different types of stimuli and fore-period durations that EEG alpha was not significantly related to reaction time improvement. Both reaction time and alpha were significantly related to foreperiod interval. Thus EEG phase may be more a function of foreperiod condition and not necessarily cardiac or respiratory phase. This finding also gives impetus to the controversy over whether EEG arousal patterns may actually infer motor facilitation.

Regarding respiratory phase, our results showing mean reaction time to be significantly faster during inspiration, 211.5 with expiration mean reaction time, 221.4 conflict with the findings of Buchsbaum and Callaway (1966). They found, in an auditory reaction time task, mean reaction time to be somewhat faster during expiration, than during inspiration. Again the possibility of motor temporal conditioning is raised as they used fixed foreperiods of either 200 or 400 msec. Our results confirm those by Roitbak et al. (1962) and Hildebrandt and Engel (1963) although the reaction time paradigms differed in that they did not control the warning signal. Buchsbaum and Callaway (1966) accounting for faster reaction time during expiration
note that stimulation of the vagus during inspiration results in a
general motor inhibition reflected in slower reaction times. However,
other respiratory activity occurring during inspiration can be offered
to account for faster reaction times during this phase (Ganong 1973,
pp. 410-492; Berne and Levy 1967, pp. 126-192; Guyton 1971, pp. 293,
700; Lehmann and Knauss 1964, pp. 710-715). The author will not make
an attempt to cite specifics of respiratory physiology to support the
data as the nature of the supporting findings vary regarding human and
animal subject source. As these highly specific motor and pressure
activity occurring during respiration were not monitored exact inter­
pretation would only remain speculative. What can be said is the
nature of the interaction appears biphasic, those events occurring in
inspiration which may facilitate reaction time possibly receding during
expiration.

That biphasic influence of the respiratory cycle in conjunction
with the cardiac cycle significantly effect reaction time impels con­
sideration of these two cycles in reaction time tasks.

The data support the role of both cardiac and respiratory cycle
phases in altering speed of response and impetus is given to the need
to control these two autonomic events in reducing variance in reaction
time tasks.
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