ULTRASTABLE RESPONSE-STIMULUS SYNCHRONIZATION

ULTRASTABLE RESPONSE-STIMULUS SYNCHRONIZATION: ACQUISITION, STIMULUS CONTROL, AND A MODEL OF STEADY STATE PERFORMANCE

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Abstract

Human beings have a remarkable ability to accurately anticipate the time of occurrence of a predictable sensory event and synchronize an overt response with that event. Presumably this behaviour is mediated by central temporal mechanisms which are involved in timing the delay required to trigger the response at some precise point in time. It was the purpose of this investigation to examine the nature and functioning of these human temporal mechanisms.

The basic task, modelled after Kristofferson (1976), involved presenting two brief stimuli, separated by a short time interval. Interstimulus interval was fixed for a particular subject and the subject was instructed to anticipate the second stimulus, timing from the first, in order to trigger a response which would be manifested in synchrony with onset of the second stimulus. Several modifications were made to this basic response-stimulus synchronization procedure, which included subject-paced trials, and provision of highly salient feedback.

These modifications resulted in a significant reduction in the lowest, previous estimate of minimum response latency variance. Minimum variances under 35 msec^2 were obtained and the data indicated that response latency variance was independent of mean latency over a range of

synchronization intervals from 310 to 550 msec. Within this range, latency distributions were the same, symmetrical, and sharp-peaked, unlike typical reaction time. All responses fell within a 50 msec time window. This independence of mean latency and latency variance was present throughout acquisition.

A special technique allowed isolation of the controlling stimuli used for synchronization timing and showed that, in some situations, subjects were able to transfer timing control for synchronization responding from one modality to another with no loss of performance. This was true when transferring between auditory and tactile modalities, but not when visual interval markers were employed. A study of these dissimilar intra-modality findings, using a simple reaction time procedure, suggested that the differences could be attributed to a large afferent latency variance associated with visual stimulation which was not inherent in the other modalities.

The role of feedback in acquisition and maintenance of synchronization performance was also examined, using manipulations which either selectively removed a particular source of feedback or altered the integrity of the feedback information. These manipulations provided data which indicated feedback to be one of the most important factors responsible for producing ultrastable stimulus-response

latencies and maintaining low levels of response latency variance. The other important factor appears to be prolonged practice at a particular synchronization interval.

Results are discussed in terms of support for the notion of nonvariable, centrally-timed delays which can be inserted into the stimulus-response chain. These delays are easily adjustable, but once set, are deterministic. A formal, mathematical model was formulated which describes the response-stimulus synchronization data remarkably well and provides a well-defined theoretical framework for conceptualizing this type of behaviour. From the model, independent variance estimates were derived for both central and efferent components in the stimulus-response chain. Values obtained are consistent with previous estimates derived from quite diverse methodologies in the literature. The model also provided some insight into what changes in processing occur during acquisition of this skill.

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Table of Contents

List of Figures

List of Tables

I. Introduction

Much of human behaviour is highly dependent upon the ability of the organism to temporally structure events in it's environment, make several kinds of decisions regarding those events, and time actions so as to respond appropriately within that temporal framework. We are constantly constrained in our interactions with the real world by the actions and movements of other people and objects in our environment. In many situations, these actions require accurate timing and anticipation of imminent events in order for the organism to adjust its behaviour to the environmental requirements.

We know from everyday experience (i.e., playing music or playing tennis, etc.) that anticipation of a sensory event and organization of an appropriate response, such that the result is temporal coincidence between the stimulus and overt response, can be exceedingly accurate. Presumably this ability to accurately anticipate the time of occurrence of a predictable sensory event and synchronize a response with that event is mediated by central temporal mechanisms which are involved in timing the delay required to trigger the response at some precise point in time. It is the purpose of this investigation to examine the nature and functioning of these human temporal mechanisms.

The ability of subjects to exert control over the

delay of their responses has been studied using numerous experimental paradigms. Part of this variety in methodologies has been due to an array of theoretical questions stimulating the research.

A. Anticipation in Simple Reaction Time

Several experiments were involved with measuring the accuracy of anticipatory responses in the simple reaction time situation. Their primary concern was with isolating the "true form" of the reaction time (RT) distribution in order to facilitate theorizing about elementary timing mechanisms underlying such behaviour. Their goal was hampered, however, by the fact that the observed RT distributions contained response latencies which were too short to have been triggered by the action stimulus. The source of these aberrantly fast responses was attributed to anticipatory responding triggered by the warning signal. In an attempt to control this confounding factor, a general paradigm was developed for approaching the so-called "true reaction time" distribution which involved experimentally shifting the simple RT distributions right or left along the time axis until minimum response latency variance was obtained. The rationale behind the use of such a procedure was based on the assumption that time delayed responses triggered by either the warning signal (when the distribution is shifted left) or the action stimulus (when

the distribution is shifted right) are more variable than true reaction time responses triggered by the action stimulus. The goal, therefore, was to maximize the proportion of "true" reactions in the observed distribution by minimizing variance via these shifting procedures.

Shifting of response latency distributions has been accomplished either by simply instructing subjects to produce a target response latency and informing them of their accuracy on each trial (Kornblum, 1973; Ollman & Billington, 1972), or by use of narrow payoff bands centred around different points following the action stimulus (Saslow, 1968, 1972, 1974; Snodgrass, 1969; Snodgrass et al., 1967). In the latter procedure, reinforcement was given contingent upon the subject producing a response latency within the bounds of the specified payoff band. Information was also provided on non-reinforced trials as to the direction of error.

Payoff bands proved to be most effective in modifying the subject's delay of response. Using this technique, Snodgrass et al. (1967) observed that subjects' mean response latencies closely approximated the temporal extent of the payoff band position even out to 5 seconds. As the payoff band was moved closer in time to the action stimulus both mean response latency and the associated variance decreased until the subject had to include response latencies triggered by the warning signal in order to meet

the payoff band requirements. When this occurred, the response latency variance inflated rapidly. These results support the basic assumption underlying the use of the shifting procedures and indicate that the standard deviation of the response latency distribution is a fixed proportion of the mean (Saslow, 1974; Snodgrass et al., 1967). For well trained subjects this ratio of standard deviation to mean is about 0.100. This value is in close agreement with estimates by Woodrow (1930, 1933). These studies suggest that accuracy of anticipatory timing for elicitation of a response at some precise point in time is a monotonically decreasing function of the duration of the time interval to be anticipated.

B. Sensorimotor Synchronization

Another general paradigm for investigating anticipatory timing has been termed sensorimotor synchronization. With this procedure, the target response latency to be produced is indicated by the onset of a second stimulus which follows the action stimulus by a specific interval of time. The subject's task is to synchronize an overt response with the time of occurrence of the second stimulus. In order to accomplish this, the subject must anticipate the occurrence of the second stimulus, timing from the first, and initiate a response at some time prior to the second stimulus so that the overt consequences of the response and the stimulus

onset are perfectly coincident in time. Such a procedure seems at least as precise as the payoff band technique in controlling subjects' response latencies.

Response-stimulus synchronization procedures have been applied to both discrete trial and repetitive synchronization timing situations. The latter type of studies, however, have typically been primarily concerned with analyzing the regularity of interresponse timing between successive movements in a repetitive key-tapping task (e.g., Michon, 1967; Wing & Kristofferson, 1973a). Requiring the subjects to synchronize responses with each of a train of stimuli served only as a means to specify the desired interresponse delay. This was followed by a continuation phase during which the rhythmic responding, established during the synchronization phase, was to be continued without provision of the stimulus sequence. The results from the synchronization phases, however, indicate that subjects can time their response latencies with considerable precision over a range of interresponse intervals from 170 to 3,333 msec, although achieved levels of performance differed substantially between studies. With well-practiced subjects, the ratio of standard deviation to mean was approximately 0.023. This is a reduction by a factor of four over the comparable measures obtained with the payoff-band technique.

A close analogy to the repetitive key-tapping experiments, but within the realms of discrete trial synchronization, was an experiment by Bartlett and Bartlett (1959). Subjects were instructed to attend to a train of auditory clicks, equally spaced in time, and to respond in synchrony with any one of the stimulus presentations once they were satisfied that they were following the rhythm. The subject's response terminated the sequence and a new temporal spacing for the stimuli of the next trial was selected from a range of 125 to 4,000 msec for one experiment and from 167 to 500 msec for another two experiments. Long periods of practice were required to master this simple task, even when provided with feedback which specified whether their response was early or late. The results from well-trained subjects revealed mean response latencies which were within ±10 msec of perfect synchrony and standard deviations from 20 to 30 msec. Although proportionality between mean and standard deviation was evident for temporal spacings of over 500 msec, as was typical, there was a strong suggestion that over the range from 200 to 500 msec standard deviation of response latencies was constant. Although Bartlett and Bartlett never drew the inference, this was one of the first indications that deterministic delays might be available in this type of response timing situation. In a subsequent experiment with light flashes and well-trained subjects they

found the same pattern of results but the standard deviation of response latencies was almost twice as large as that which had been obtained with auditory stimuli.

Naatanen, Muranen, and Merisalo (1974) investigated anticipatory timing with a slightly different procedure. They instructed subjects to anticipate the moment of the action stimulus in a simple reaction time situation by estimating the foreperiod duration, which was fixed over trials, and producing a response that was synchronous with the action stimulus. Foreperiods ranged from 250 to 4,000 msec and feedback was provided as to whether the subject's response latency over or underestimated the foreperiod duration. Beyond 500 msec, accuracy of anticipation was observed to monotonically decrease, as observed in other studies. But from 250 to 500 msec the standard deviation of response latencies was relatively constant at about 25 msec. These values are not substantially different from values obtained for simple reaction time with well-trained subjects. Naatanen et al. (1974) argued that this small variability might be related to the delivery of the action stimulus on each trial. Presumably the perceived temporal relationship between the action stimulus and the subject's overt response acted as an effective source of feedback in conjunction with the verbal feedback which was also provided.

The general indication from all these studies is that internally timed intervals, presumably underlying the observed response delays, can be quite accurately generated with the degree of accuracy generally being a monotonically decreasing function of the length of interval to be estimated. For well-trained subjects, standard deviation of time estimates, measured in terms of variability of anticipatory response latencies, has been shown to be only slightly higher than that obtained in simple reaction time experiments (about 400 $msec^2$).

C. Kristofferson's (1976) Experiments

Recently, however, Kristofferson (1976) has reported experiments which indicate that, under certain conditions, time estimation responses can be made with considerably less variance than is typical of simple reaction time. The procedure involved a response-stimulus synchronization task similar to that employed by Naatanen et al. (1974). Two brief auditory pulses were presented, separated by a short, fixed interval of time. The subject was instructed to anticipate the occurrence of the second stimulus, timing from the first, and make his response in synchrony with the second stimulus. Much practice was given with the same interval, and the response was depression of a microswitch with the index finger.

Using this procedure, Kristofferson found extremely accurate synchronization with mean response latency values within a couple of milliseconds of perfect synchrony. He also observed that the anticipation interval between the two stimuli could be gradually changed from values typical of simple RT (160 msec) all the way to 550 msec with no change in variance, or shape, of the response latency distribution. This led him to reject the idea that response latency variance is necessarily a monotonically increasing function of mean latency over this range and to propose the existence of deterministic delays. Such a notion was not entirely new in the literature. For example, McGill (1962) postulated a deterministic periodic process underlying several types of biological processes and several other earlier studies hinted at a similar deterministic phenomenon, although not necessarily periodic (Bartlett & Bartlett, 1959; Naatanen et al., 1974; Saslow, 1974; and Wing & Kristofferson, 1973a). The results obtained by Kristofferson, however, provided the first strong empirical support for such a notion.

These variance-free, adjustable delays were thought to be in the afferent latency of the stimulus. Although speculative, such a notion is consistent with certain assumptions and conclusions derived from work on successiveness discrimination (Allan & Kristofferson, 1974; Kristofferson, 1967), duration discrimination (Kristofferson, 1977), temporal order discrimination (Allan,

1975), and reaction time (Ollman & Billington, 1972).

Beyond 550 msec Kristofferson (1976) found the typical monotonic increase in standard deviation of response latencies as a function of mean latency with the ratio of standard deviation to mean usually between 0.020 and 0.025, in good agreement with the value of 0.023 obtained by Wing and Kristofferson (1973a) using the interresponse timing procedure. The problem is that the value provided by Wing and Kristofferson's results was obtained using synchronization intervals in the same region where Kristofferson (1976) observed decidedly constant levels of variability. The conclusion was reached that under certain conditions subjects are able to insert an additional delay into an S-R chain without the delay contributing any additional variance itself but that this mechanism is unavailable for the timing of repetitive movements because it is in the afferent system, which is not involved in such timing. The added delay can be set at any value between zero and about 400 msec but, once set, is deterministic. This accounts for the range of equi-variance observed between 170 and 550 msec when the minimum reaction time latency of 150 msec is added to the deterministic delay. To account for the proportional increase in response variability, as a function of mean latency, for intervals longer than 550 msec Kristofferson postulated a tandem process model of synchronization performance. For long

intervals, the maximum deterministic delay is maintained in the S-R chain and any further delay is provided by the insertion of a timer like the central response timekeeper of Wing and Kristofferson (1973a).

With respect to the shape of response latency distributions obtained using response-stimulus synchronization, Kristofferson (1976) reported that all distributions were sharp-peaked, symmetrical and well centred about the point of veridical synchrony over the entire range tested. In contrast to most RT findings, symmetrical distributions were obtained even at response latencies characteristic of simple reaction time. Reaction time distributions are usually positively skewed, with high tails and typically more than four times the 100 $msec^2$ response latency variance reported by Kristofferson. Saslow (1974) has obtained some symmetrical RT distributions using narrow payoff bands, but minimum variances were still more than double those obtained with response-stimulus synchronization. Time estimation response distributions are usually symmetrical, but with variance levels much higher than any of those mentioned above.

Kristofferson's (1976) findings, that the degree of temporal certainty associated with time estimation responses is independent of mean latency over a substantial range of intervals and is the same as when the response is a simple RT, are contrary to most current thinking about timing

mechanisms (see Allan, 1979 for a review). They indicate that added delays can be inserted into an S-R chain without any associated increase in variance. Moreover, the shape of the distribution obtained when response latency variance is minimized is very symmetrical, sharply peaked, and very similar in form despite large changes in mean.

The simple form of the distribution, especially the sharp peak, suggests the contribution of relatively few sources of variance, because if the distribution was a convolution of many component distributions a sharp peak would not be expected.

Wing and Kristofferson (1973b), using an interresponse timing procedure, were able to provide independent estimates of two variance components — efferent delay variance and central timing variance. Subject's responses were similar to those used in Kristofferson's (1976) experiments but were part of a series of repeated finger taps, as discussed earlier. The obtained variance estimates for the efferent component ranged from 10 to 50 $msec^2$. Subtracting this amount from the minimum total variance of 100 $msec^2$ obtained by Kristofferson led to speculation that the central component could be represented by a triangular distribution with a base of somewhat less than 50 msec. The existence of such a unit of central temporal variability is implied by Kristofferson's (1967) time quantum theory.

The delay, as mentioned earlier, may be part of the afferent latency, and if so, it adds no variability to the S-R chain. Kristofferson (1976) argued that if this possibility is correct, these delays could play a significant role in individual acts of information processing. However, many questions related to the existence and use of such delays in synchronization performance remained.

D. Michaels' (1977) Experiments

A subsequent study by Michaels (1977) sought to fill in some of the unknowns by measuring some of the characteristics of time estimation responses which were relatively uncertain in the literature and extending the description of the events involved in response-stimulus synchronization.

The general procedure was similar to that employed by Kristofferson (1976), with the following changes: The microswitch was replaced by a touch-sensitive button and the synchronization response was changed to a finger withdrawal response. Other changes included self-pacing of trials and the addition of a visual display to augment the feedback information presumably inherent in the task simply due to the delivery of the synchronization pulse.

The first experiment was primarily concerned with training high levels of performance in four subjects at four

different synchronization intervals which ranged from 240 to 890 msec. The results showed the typical course of acquisition in this type of task; namely, drastic improvements over the first several sessions of practice followed by a long period of slow improvement. After 25 sessions of 400 trials each, the three subjects at the shorter synchronization intervals (up to 640 msec) exhibited virtually identical performance in terms of variance measures, supporting the notion of deterministic delays being used over this range; but the level of performance was relatively poor (about 600 $msec^2$) compared to the 100 $msec^2$ levels obtained by Kristofferson (1976). In all other respects, such as shape of response latency distributions and sequential dependencies between successive response latencies, the results were consistent with Kristofferson's.

In an attempt to reconcile the performance difference observed between the two studies, Michaels argued that part of the difference may have been due to the lack of the same type of perceptual feedback which was inherent in Kristofferson's study. This feedback, regarding the accuracy of the subject's synchronization response, was conveyed by discrimination of the temporal ordering of the synchronization pulse and the audible response sound produced by activation of the microswitch. In contrast, it was argued that Michaels' procedure did not provide for such discriminations because synchronization responding on a

touch-sensitive button did not generate the necessary auditory cues for making the discriminations. Of interest, though, is the fact that removal of the synchronization pulse did have a large and deleterious effect on performance. This suggests, contrary to Michaels' reasoning, that perceptual feedback of some sort is available and is being effectively used in these experiments. Visually displayed knowledge of results was also provided but apparently this information was of little use because when it was removed, in Michaels' third experiment, there was no significant change in performance.

This leaves the question regarding the cause of the differences in performance between Kristofferson's and Michaels' experiments unanswered. A second examination of the two studies, however, suggests that the visual feedback used in Michaels' procedure may have, in fact, contributed to the poorer performance by delaying or preventing the automatization of synchronization responding. This type of feedback required a relatively high level of cognitive activity to translate spatial information (displacement of a cursor) into temporal information regarding the extent of one's error of synchrony. The fact that no improvements in performance were noted when this feedback was removed probably was a result of not enough practice to show a trend. Only 5 sessions were run on this condition.

Michaels' final two experiments were concerned with isolating characteristics of the hypothesized distribution of response triggers. The paradigm involved the addition, on half of the trials, of a third, discriminably different, auditory pulse (countermand signal) during the synchronization interval. Subjects were instructed to withhold their synchronization response on those trials in which the countermand signal was presented. By varying the temporal placement of this countermand signal, prior to the synchronization pulse, Michaels was able to trace out the psychophysical function for the underlying distribution of response trigger times. The results of these experiments indicated that subjects have the ability to preempt the output of their synchronization response up to within about 100 msec of the synchronization stimulus, irrespective of the actual interval to be timed. Such findings argue against the participation of efferent stages in the delay of anticipatory responses and support the notion of some central timekeeper.

E. Summary of Previous Research

Based on the findings of all the studies reviewed, some general conclusions can be drawn. It is clear that despite the seemingly simple nature of the task (delaying a response by some fixed interval of time so that it is coincident with some temporally predictable event), proficiency at the task

requires incredibly extensive levels of practice. That practice effects are in part responsible for many of the discrepant findings in the literature is perfectly clear. At relatively low levels of practice, results indicate that response latency variance is a monotonically increasing function of mean latency with a ratio of standard deviation to mean of about 0.100 (Saslow, 1974; Snodgrass, 1969; Snodgrass, Luce, & Galanter, 1967). For well-trained subjects, however, this ratio can be reduced to under 0.025 (Kristofferson, 1976; Wing & Kristofferson, 1973a) and several studies provide indications that below 500 to 600 msec response latency variance is a constant — independent of mean latency (Bartlett & Bartlett, 1959; Kristofferson, 1976; Michaels, 1977; and Saslow, 1974). The observation of response timing which seems to make use of deterministic delays also seems to be highly dependent on the provision of feedback regarding the subject's performance (cf. Bartlett & Bartlett, 1959 vs. Triesman, 1963). Degree of dependency between successive response latencies also seems to be directly related to the amount and type of feedback information available (Kristofferson, 1976; Michaels, 1977). Only two types of results are relatively consistent across all the studies. These include the finding that mean response latencies are always closely centred about the desired point in time and that the shape of the response latency distributions is typically symmetrical and sharply

peaked with short tails.

In short, the conclusion to be drawn from this review is that methodological considerations with respect to amount of practice and characteristics of the feedback given to the subject are of paramount importance in obtaining accurate, reliable, and stable data. Also of importance is the refinement of response latency measurements and scheduling of trial events so as to provide the most information about the underlying mechanisms involved in response-stimulus synchronization while reducing unwanted sources of variance to a minimum. It was with these considerations in mind that the experimental paradigm for the investigation to be reported was developed and applied to the range of synchronization intervals where deterministic delays appear to be utilized.

F. Present Research Concerns

Kristofferson's (1976) procedure seemed to be the most efficient, in terms of producing highly skilled synchronization behaviour, and was thus taken as the basic methodology on which to institute certain methodological refinements, some similar to those incorporated by Michaels (1977). In Kristofferson's procedure, some variability associated with recording the response could have been encountered because the microswitch used entailed some movement time and required that a force be exerted against a

spring in order for electrical contact to occur. Consequently, the synchronization response in the present procedure is changed to a finger-withdrawal response from a touch-sensitive button which involves no movement time, similar to that used by Michaels (1977). Electrical contact is effected as soon as the finger breaks contact with the button.

Michaels argued, however, that part of the reason for his subjects' relatively poor performance was due to the use of the noiseless response button. He contended that absence of the response sound removed the source of immediate perceptual feedback that Kristofferson had suggested was of critical importance in the attainment of low levels of response latency variance. In Kristofferson's study, this information was provided by the perceived temporal relation between the sound of the response button, which was audible to the subject, and the auditory synchronization pulse. Michaels tried to compensate for this lack by providing delayed feedback information on a visual display, but this method proved unsatisfactory. As mentioned earlier, however, immediate perceptual feedback of some sort did appear to be present in Michaels' study even when a silent switch was used, it was just of a different form. With no response sound, the subject has to make an evaluation of the temporal relation between the tactile and/or proprioceptive stimulation produced by the

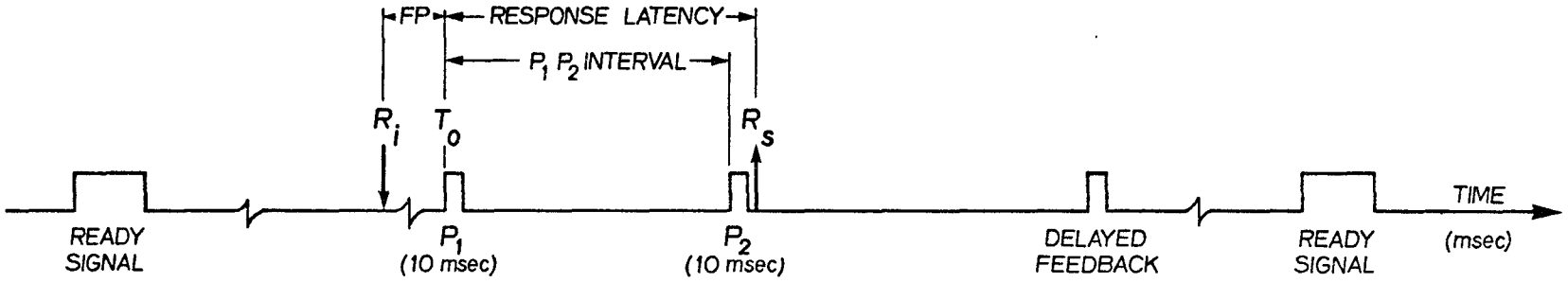
finger-withdrawal response and the auditory stimulation produced by the synchronization pulse. Evidence indicates that this comparison can be made as precisely as an intra-auditory comparison of temporal relations (Hirsh & Sherrick, 1961).

This type of perceptual feedback, however, is very complex and difficult to specify to the subject. Consequently, the revised procedure to be used here incorporates an additional delayed feedback signal that can be well specified in instructions to the subjects. This delayed source of feedback differs from that used by Michaels (1977) in that it is presented aurally with the duration of the signal equal to the error of synchrony on each trial. This is done for three reasons. First, there is some indication that auditory stimuli convey more temporal information than visual (Efron, 1973; Goldstone & Lhamon, 1971; Goodfellow, 1934; and Lhamon & Goldstone, 1974). Secondly, presentation of the feedback signal in the same modality as the stimuli seems desireable in order to reduce the amount of attention switching required when two modalities are involved. And thirdly, use of the temporal extent of the feedback pulse to indicate the magnitude of the error of synchrony is more direct than the translation of spatial to temporal information required in Michaels' procedure.

Another change, also instituted by Michaels, was made to alleviate a potential problem encountered with experimenter-paced trials. In Kristofferson's procedure, trials were presented at regular intervals whether the subject was ready or not. Under these conditions, momentary lapses of attention or any other kind of disruption could affect response latencies on some trials and inflate variance measures. Consequently, experimenter-paced trials are replaced by subject-paced trials in which the presentation of the stimulus sequence is initiated by the subject.

Although the use of subject-paced trials reduces the problem of momentary inattention, it makes it difficult to determine which stimulus, or event, is controlling synchronization responding because the subject's initiation response, R*i*, occurs just prior to the first auditory pulse, P1 (see Figure 1), by the amount of a brief foreperiod. The second auditory pulse, P2, follows P1 by a fixed interval, and the subject's task is to time an appropriate interval, and trigger his synchronization response, Rs, such that it coincides in time with the onset of P2. Since Ri and P1 occur in such close temporal proximity, some technique is required to discriminate which of these two events is the controlling stimulus for the timing of RS. The technique used involves providing the experimenter with the ability to manipulate foreperiod variability and to omit P1 from the

Figure 1 - Diagram of a typical synchronization trial. Spacing of trials is paced by the subject's initiation response, R_i, which can be made at any time following the ready signal. FP refers to the foreperiod duration.


FP
RESPONSE LATENCY
P1 P2 INTERVAL
Ri
To
Rs
READY SIGNAL
P1 (10 msec)
P2 (10 msec)
DELAYED FEEDBACK
READY SIGNAL
TIME (msec)

trial sequence. Objective timing of the subject's response latencies is always made relative to the time-point To, which corresponds to P1 onset. Thus, when P1 is present, it provides a good cue for synchronization timing because it is time-locked to the onset of P2. The same is true of Ri only if the foreperiod is constant. Therefore, with a variable foreperiod, if the subject is ignoring P1 and timing his synchronization response from Ri, then removal of P1 should have little effect on performance. However, if the subject is using P1 for timing control then its removal should be reflected in an increase in response latency variance by an amount equal to, if not greater than, the variability of the foreperiod. With the appropriate controls, these stimulus manipulations allow unambiguous identification of the controlling stimulus in this task.

One of the goals to be met in estimating minimum response latency variance is to eliminate, or at least minimize, any sequential dependencies in responding because such dependencies introduce an additional, extraneous component of variance into the measurements. Since feedback is a major part of the present procedure, if subjects adopt an error correction strategy on a trial-to-trial basis, such unwanted inflation of variance might occur. Thus, analysis of temporal autocorrelation of response latencies is important, both for determining the effect of feedback and for determining that dependencies do not introduce variance,

thereby insuring validity of the minimum variance estimates.

G. Synchronization Model

The theoretical model of response-stimulus synchronization behaviour to be entertained in this investigation is one derived from a model of single-stimulus duration discrimination (Kristofferson, 1977). The model, simply stated, proposes that the first stimulus event, P1, gives rise to an internal time-point event, denoted as the stimulus trigger, which triggers a time interval delay ending at another time-point event, *C*, the criterion. Then *C* acts as a response trigger, producing an overt response, in the present case a finger-withdrawal, after some variable efferent delay.

The afferent latency between the stimulus and stimulus trigger has some non-zero delay but the variance is assumed to be negligible. This assumption is based on some auditory time discrimination studies (Divenyi, 1976) which estimated the variance of the latency of detection for a 60 dB sinusoid to be about 0.2 msec^2. The model also assumes the time generator is deterministic and contributes no variance to the distribution of response latencies.

Variance obtained in the response latency distributions is hypothesized as originating from three sources. Two of these sources of variance result from variable delays (quantal in nature) which occur sometime

during the processing of temporal information. The model doesn't specify the locus of these delays but does assume each delay is variable over a range from zero to q msec, independent, and uniformly distributed. Consequently, the convolution of these two units of temporal variability produces a triangular distribution spanning a range of $2q$ msec. The q referred to here is presumed to be related to the base periodicity of an autonomous central "clock" (Kristofferson, 1967) in which the delays represent waiting times for information transfer within the central nervous system.

The third source of variance arises from the variability in the efferent delay associated with the output of a motor response. It is assumed to be relatively small and normally distributed. Thus, the convolution of all three sources of variance results in a response latency distribution which is basically triangular, with small tails and a slightly rounded peak.

The total variance of such a distribution can be represented as follows:

$$V(total) = V(2q) + V(D)$$

where $V(2q)$ represents the variance of the triangular component and $V(D)$ represents the efferent delay variance. Both these values have been estimated in other types of time perception paradigms. Typically, the lowest value for q obtained in duration discrimination studies is near 25 msec

(Kristofferson, 1973) and the value for $V(D)$ ranges from 10 to 50 msec^2. This latter value comes from the work of Wing and Kristofferson (1973b) on interresponse timing. They were able to isolate this component of variance from their data using lag 1 autocorrelation information. The variance of the triangular component can be rewritten as $q^2/6$; thus an estimate of the minimum $V(total)$ for response-stimulus synchronization is:

$$V(total) = q^2/6 + V(D) = 625/6 + 10 = 104 \text{ msec}^2$$

This value is almost exactly what was obtained in Kristofferson's (1976) study with well-practiced subjects.

However, recent findings in duration discrimination (Kristofferson, 1980) suggest that q-values substantially less than 25 msec might also be available. These experiments show that the relationship between q and the base duration to be discriminated is not simple, and furthermore, that it depends upon specific practice. Over certain ranges of base duration, increases in q occur slowly, whereas over other ranges, changes in q occur quite fast. These rapid changes, or steps, correspond with base durations of approximately 200, 400, and 800 msec which produce q-values of close to 25, 50, and 100 msec, respectively. Below 200, at a base duration of 100, the estimate for q is 13 msec. Consequently, Kristofferson proposed a "doubling" rule to describe this relationship in which "doubling or halving base duration a given number of

times doubles or halves q the same number of times" (p. 302). Although the evidence for a q-value near 13 msec is relatively weak (only 20 sessions with one subject), and there are small individual differences in the exact level of performance observed at each base duration, a minimum value for q could be assumed in the range from 10 to 13 msec. In this case, the prediction of minimum response latency variance in response-stimulus synchronization, based on this data, would have to be revised to a substantially reduced value ranging from 27 to 38 $msec^2$. This is a truly remarkable accuracy of response timing if, in fact, it can be realized.

H. Present Research Interests

With the prospect of finding such new, low levels of response latency variance, the present investigation was undertaken. Prolonged practice, with a particular synchronization interval, was deemed imperative and it was hoped that the inclusion of highly accurate feedback information in conjunction with the other methodological changes, previously discussed, would provide the means for observing highly stable and low variance data for theorizing about elementary central temporal mechanisms involved in gating the flow of information within our central nervous system and which could be used to govern our behaviour relative to our environment.

As a result, several empirical, as well as theoretical questions, generated the set of experiments to be reported. The major empirical concerns were: (1) examining the effects on performance of extensive levels of practice with a single synchronization interval; (2) determining the utility of different types of feedback information, both during and after acquisition of the skill; (3) isolating the stimuli actually controlling synchronization performance; (4) monitoring the ability of a subject to transfer synchronization stimulus control to another modality; and (5) analyzing the nature of sequential dependencies between response latencies during and after acquisition.

Of primary theoretical interest was the gaining of support for the synchronization model and its generality in time perception. This includes: (1) finding new, low levels of response latency variance which are in line with the predictions of the model using independent estimates from other temporal research enterprises; (2) obtaining evidence of deterministic delays being used over the range of synchronization intervals to be tested; and (3) providing stable data for mathematical modelling in an attempt to partial out the different variance components predicted by the synchronization model.

II. Experiment 1 - Acquisition and Stimulus Control of Synchronization

Using a revised procedure, this experiment was concerned with answering the following questions related to the remarkable human timing abilities observed by Kristofferson (1976): Can special procedures further reduce the minimum variability of S-R latencies; and if so, will the shape and variability of the distribution be consistent with the predictions of the synchronization model? Moreover, at the ultimate limit of response-stimulus synchronization performance, will the variability of these centrally timed delays be independent of mean delay over the range of interest, as the principle of determinism, inherent in the model, would predict? Of related interest was the question of whether this independence of mean and variance is maintained throughout acquisition. Another question of primary importance was concerned with what stimuli actually control synchronization performance? And finally, are successive response latencies independent of one another or do they indicate that the variance measures are inflated by exhibiting a temporal autocorrelation?

A. Method

Subjects

Five graduate students, three female and two male, including the author, participated in the experiment. Two of the subjects, C.H. and G.H., were both well practiced in reaction time and synchronization experiments.

Procedure

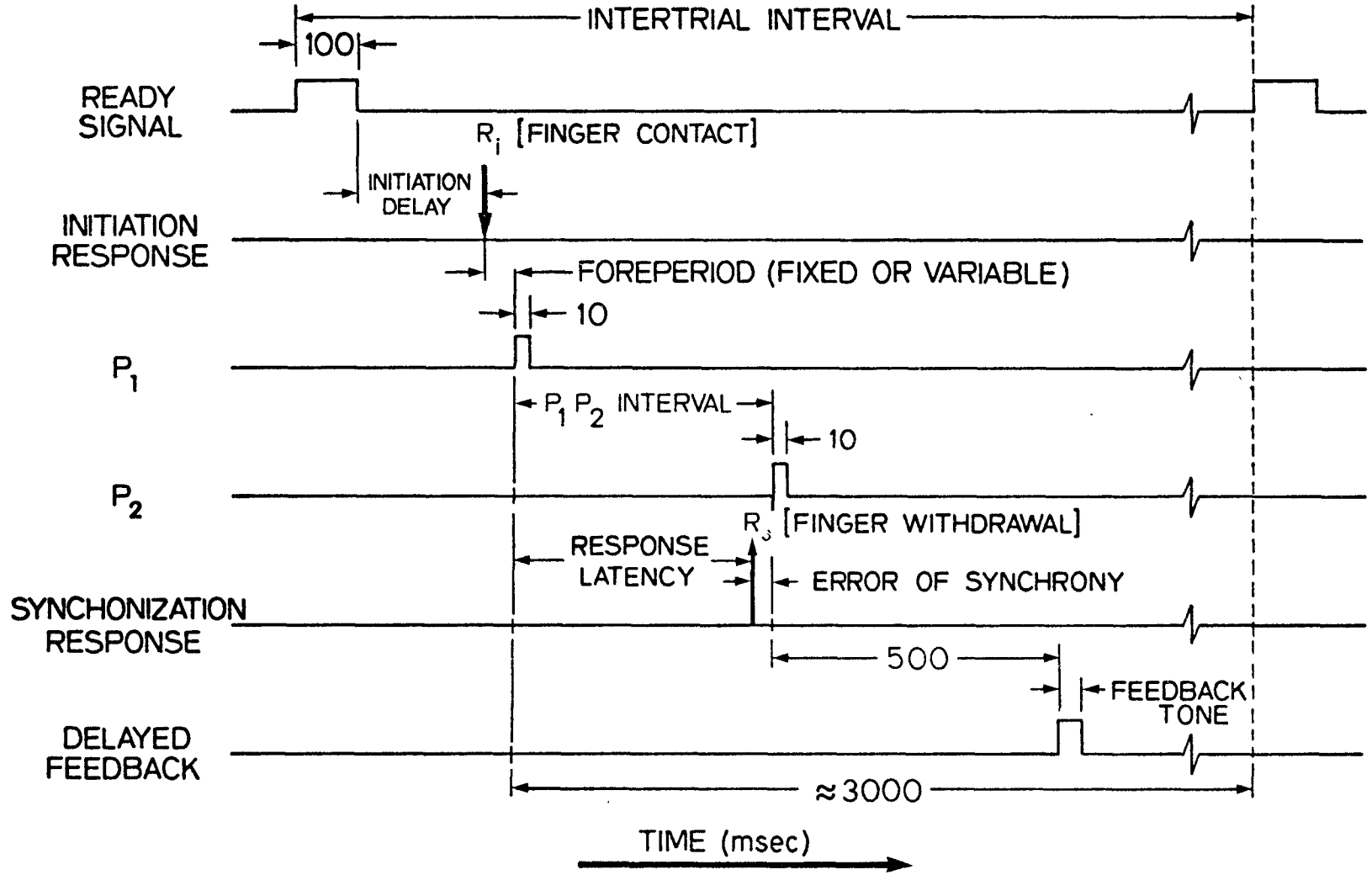
A response-stimulus synchronization task, similar to that used by Kristofferson (1976), was employed. All sequencing of stimuli, recording of responses, and analysis of data were under control of a Digital Equipment Corporation PDP-8e digital computer. Subjects were run one at a time in a small, sound-attenuated room. A detailed diagram of a typical trial sequence is diagrammed in Figure 2. Each trial began with a 100 msec visual ready signal which indicated to the subject that a trial sequence was available. To initiate the sequence, the subject had to place his index finger onto a touch-sensitive button. This was referred to as the initiation response, R*i*. The electronic switching produced by finger contact triggered a short, variable foreperiod. (Note that the use of foreperiod here does not refer to the interval between the ready signal and action stimulus, as is customary.) Foreperiod durations were selected randomly, on each trial, from a uniform distribution spanning a range from 51 to 71

msec, with a mean of 61 msec and variance of 33.3 msec^2. Following the foreperiod, two 10 msec auditory pulses (P1 and P2) were presented, separated by an empty interval. This interval, measured from onset to onset, was referred to as the P1P2 interval and remained constant for a given subject throughout the entire experiment.

Timing of the critical durations and events (i.e., P1, P2, P1P2 interval, and FP) was periodically checked for accuracy with an independent frequency counter. Accuracy was typically within ±0.10 msec.

The subject was instructed to withdraw his index finger from the touch-sensitive button in synchrony with the onset of P2. In order to accomplish this, the subject had to generate an appropriate delay and trigger his response at some time prior to P2 if his overt synchronization response, RS, was to coincide with the onset of P2. Response latency was measured, to the nearest millisecond, from P1 onset, to the moment of finger withdrawal. Error of synchrony was defined as the response latency minus the P1P2 interval; thus, a negative value represented an early response. Immediate perceptual feedback was available to the subject by comparing his time of response with the time of occurrence of the synchronization pulse, P2. A delayed feedback signal, indicating the subject's error of synchrony, was also provided on each trial. It consisted of a third auditory pulse 500 msec after P2 onset. The

Figure 2 - Detailed diagram of a typical synchronization trial. Intertrial interval is dependent on both the subject's initiation delay and the P1P2 interval. Duration of the feedback tone is equal to the subject's error of synchrony on the particular trial. Direction of error is indicated by a brief flash of light accompanying the feedback tone on trials where RS occurs prior to P2 onset.


INTERTRIAL INTERVAL
100
READY SIGNAL
R_i [FINGER CONTACT]
INITIATION DELAY
INITIATION RESPONSE
FOREPERIOD (FIXED OR VARIABLE)
10
P_1
P_1 P_2 INTERVAL
10
P_2
R_s [FINGER WITHDRAWAL]
RESPONSE LATENCY
ERROR OF SYNCHRONY
SYNCHONIZATION RESPONSE
500
FEEDBACK TONE
DELAYED FEEDBACK
≈3000
TIME (msec)

duration of this feedback pulse was equal in magnitude to the absolute value of the error of synchrony in milliseconds while the direction of error was indicated by a brief 100 msec light flash which accompanied the feedback pulse if the subject's response occurred early – prior to P2 onset. Following feedback, there was a fixed delay of about 2,000 msec (depending upon the P1P2 interval) before the ready signal for the next trial. Some experimental manipulations altered this stimulus configuration for a typical trial. These manipulations included selective omission of one or both of the auditory pulses, P1P2, and substitution of a constant foreperiod instead of a variable one. In the case of a constant foreperiod, its duration was fixed at 91 msec. The extra delay, relative to the mean variable foreperiod of 61 msec, was necessary for technical reasons.

A session consisted of four blocks of 100 consecutive trials each, with a rest of one minute between blocks. These rest periods between blocks were indicated to the subject by a distinctive, loudspeaker-produced sound of 500 msec duration presented at beginning and end. Length of session was dependent on the subject's delay in initiating trials, but typically a session lasted less than 45 min. Subjects generally ran one session a day, at a fixed time, except weekends. Due to the large number of sessions required of the subjects, however, some lapses in practice were permitted. The first block of 100 trials was

considered warm-up and discarded from every session, leaving 300 trials per session for analysis. On occasion, subjects accidentally brushed a finger against the response button producing an extremely aberrant response which was also discarded from analysis. This error was rare, however, and generally a full 300 responses per session were analyzed. Following each session, the subject was informed of means and variances and encouraged to reduce both his synchronization error and variance.

Stimuli and Response Characteristics

The auditory pulses, **P**1 and **P**2, were both 2,000 Hz sinusoids, gated at zero-crossing, with a rise-decay time of 2.5 msec. They were delivered binaurally over earphones at a loudness of 68 dB relative to .0002 dynes/cm^2 when on continuously. The feedback pulse was a distinctive sound produced by the mixture of 2,000 and 10,000 Hz sinusoids. It was slightly louder than **P**1 and **P**2.

The responses produced no sound and electronic switching was produced instantaneously. There was no travel time associated with use of the response button. The touch-sensitive button was a small brass knob 1.2 cm in diameter which protruded 0.5 cm out of a padded armrest positioned at a comfortable level on a table in front of the subject. The armrest provided support for the entire forearm and the heel of the hand rested on a brass plate

mounted flush into the armrest at the appropriate position. Contact with both brass plate and button had to be made before a resistance change caused electronic switching to occur (see Appendix D for a schematic of the electronic circuitry used in the response key). The response consisted of a vertical, ballistic extension of the index finger about the joint with the hand. Other parts of the hand and forearm remained flat against the armrest and relatively stationary. Between trials, the index finger was rested against the middle finger, which rested immediately adjacent to the response button, until ready to initiate the next trial. Such a strategy reduced the chance of accidental contact with the response button.

Summary of Procedural Changes and Experimental Conditions

To summarize, the basic procedure differed from Kristofferson's (1976) in several ways. A finger-withdrawal response was required from a touch-sensitive button which produced no response sound. Thus the only source for immediate perceptual feedback remaining was that provided by comparison of the temporal relation between auditory (P2) and tactile or proprioceptive (RS) stimulation. To supplement this, a well-specified, delayed feedback signal was added to the stimulus sequence. Finally, the trials were subject-paced with a very short foreperiod between the initiation response and the beginning of the P1P2 interval.

The P1P2 intervals were fixed at 460 msec for subjects G.H. and J.B., 310 msec for subjects G.T. and C.H., and 360 msec for subject M.W. Each subject received several stimulus manipulations during the experiment and remained on a particular condition until performance was stable, or at least until a definite trend toward stability was evident. A chronology of each subject's experience with the various stimulus manipulations, or conditions, used in the experiment is given in Table 1 in terms of session number. It should be noted that each subject is assigned to only a single P1P2 interval because a great amount of practice at a particular interval is thought to be crucial for obtaining valid estimates of minimum response latency variance.

B. Results

Acquisition

All subjects started training with the full procedure as described above (both P1 and P2, with the delayed feedback). Figure 3 shows the effect of practice upon the variance of response latencies with the data grouped into blocks of 20 sessions. The points plotted represent the arithmetic average of only those sessions, within the group of 20, which were obtained under the baseline condition – both P1 and P2 present. Consequently, there are some gaps where practice on intervening conditions continued over more than 20 sessions. Acquisition data for all subjects

Table 1

Chronology of Experimental Conditions in Experiment 1

	Subject				
Condition	G.H.	G.T.	C.H.	J.B.	M.W.
P1P2	1- 30	1- 30	--	--	1-105
P2 Only	31- 40	31- 45	--	--	106-115
P1P2	41- 45	46- 50	1- 35	1- 35	--
P1 Only	46- 55	51- 60	36- 45	36- 45	--
P1P2	56- 60	61- 65	46- 50	46- 50	--
P2 Only	61- 90	66- 90	51- 75	51- 80	--
P1P2	91-100	91-100	76- 90	81-125	--
P1 Only	101-155	101-120	91-105	126-193	--
P1 Only(CFP)	156-165	--	--	--	--
P1P2	166-180	121-135	106-125	--	--
P1P2(CFP)	181-200	--	--	--	--
No P1 or P2	--	136-160	--	--	--
P2 Only	--	161-170	126-135	--	--
P2 Only(CFP)	--	--	136-145	--	116-120
P1P2	201-270	171-180	146-175	--	--

Note. Numbers of the sessions devoted to each experimental condition are given in the body of the table. CFP means constant foreperiod.

generally show a relatively rapid decline in response latency variance over the first 20 to 40 sessions, followed by increasingly slow improvements. Although performance becomes highly stable with prolonged practice, it is not clear that any of the subjects reached a truly asymptotic level of performance. Note especially that performance for subject G.H. continues to improve even after 250 sessions, or more than 100,000 trials. Also note that his variance levels approach the new, low levels hoped for based on predictions of the synchronization model. Naive subjects started at variances of 1,000 to 1,500 $msec^2$, but in less than 40 sessions all of them were exhibiting response latency distributions with variances lower than the lowest reported variance levels for simple RT (Saslow, 1974). Subjects C.H. and G.H. started out with lower variances, presumably due to their prior experience with similar tasks, but any advantage was short-lived and not readily apparent after the first group of 20 sessions.

A detailed, graphical representation of early acquisition performance, for each of the naive subjects, is shown in Figure 4. It can be seen that mean response latency stabilizes about the point of veridical synchrony within the first few sessions. Mean error of synchrony, even on the first session, is less than 15 msec for all subjects. Although subject M.W. showed somewhat less stability, in terms of mean response latency, than the other

two subjects, by session 30 the mean error of synchrony rarely exceeds ±5 msec and, in most cases, mean response latency is within ±2 msec of perfect synchrony, with no evidence of preferential responding, early or late, for any of the subjects.

Response latency variance, on the other hand, takes much longer to stabilize. Rapid improvements in performance are evident over the first 6 to 8 sessions, followed by much slower gains. By session 30, all subjects are exhibiting response latency variances in the 100 to 200 $msec^2$ range, quickly approaching the level of performance obtained by Kristofferson (1976) with similar **P**1**P**2 intervals.

The important point to be noted in the acquisition curves of Figure 3 and Figure 4 – neglecting the first point for subjects C.H. and G.H. – is that they all show very similar time courses and absolute levels of variance despite the fact that: (1) different subjects are involved; (2) three different **P**1**P**2 intervals are represented; and (3) the subjects have different histories of experience on intervening stimulus manipulations (see Table 1).

Similarities in response latency variance across all three of these differences are shown in Table 2, for subjects at equivalent levels of practice. Only those subjects on the baseline condition (both **P**1 and **P**2) during the sessions tabled are compared. It is clear from these results that, with similar amounts of practice, response

Table 2

Similarities in the Response Latency Variances of Different Subjects at Several Levels of Practice with Different P1P2 Intervals and Different Histories of Task Experience.

Sessions	Subject	Variance	P1P2 Interval
101-105	M.W.	114.9	360
	J.B.	119.8	460
121-125	G.T.	73.6	310
	C.H.	72.3	310
	J.B.	84.7	460
176-180	G.T.	54.1	310
	G.H.	51.2	460

Note. Variances are expressed in $msec^2$.

latency variances exhibit minimal differences both between individuals and across a moderate range of P1P2 intervals. Thus, asymptotic performance is independent of mean synchronization latency for the intervals tested (cf. Kristofferson, 1976), and so is rate of acquisition.

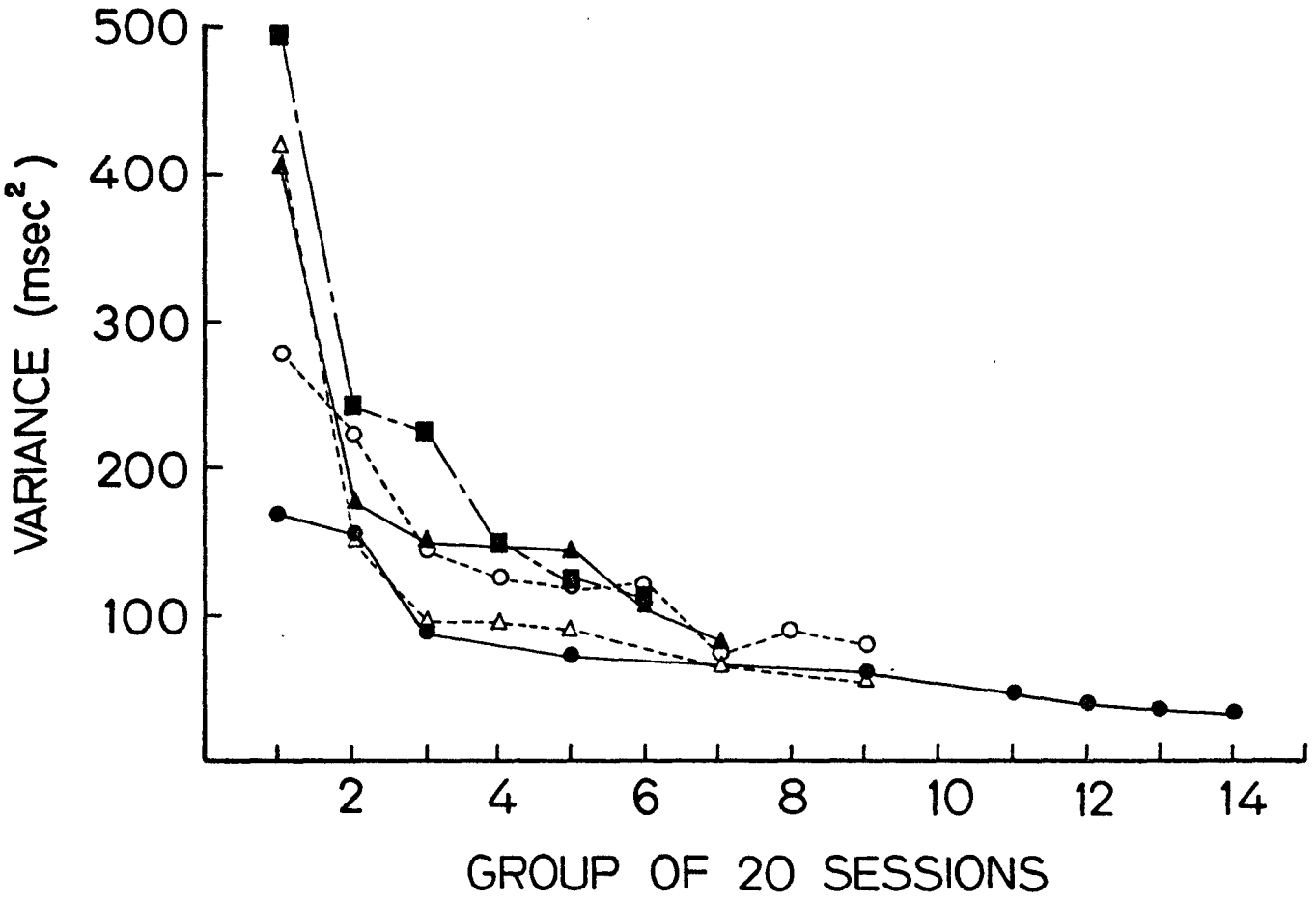
It has been suggested by Fitts (1964) and others (e.g., Stevens, 1964; and Stevens & Savin, 1962) that performance acquisition in a skilled task can be described by a power function such that the logarithm of the performance variable is linearly related to the logarithm of the measure of practice. Figure 5 shows the result of such an analysis on the same data that were presented for the naive subjects in the lower panel of Figure 4. Linear plots seem to provide a good description of the data. The parameter values obtained from the regression analysis are provided in Table 3. Correlation coefficients range from -0.89 to -0.96, accounting for from 79 to 92% of the variance. If the two rather deviant points for G.T. on sessions 12 and 14 are omitted from the analysis his correlation coefficient jumps to -0.93, thereby accounting for over 86% of the variance observed in his acquisition performance. Similarities in the time course of acquisition of this skill are again evident from the comparable estimates of slope and intercept obtained for each of the subjects.

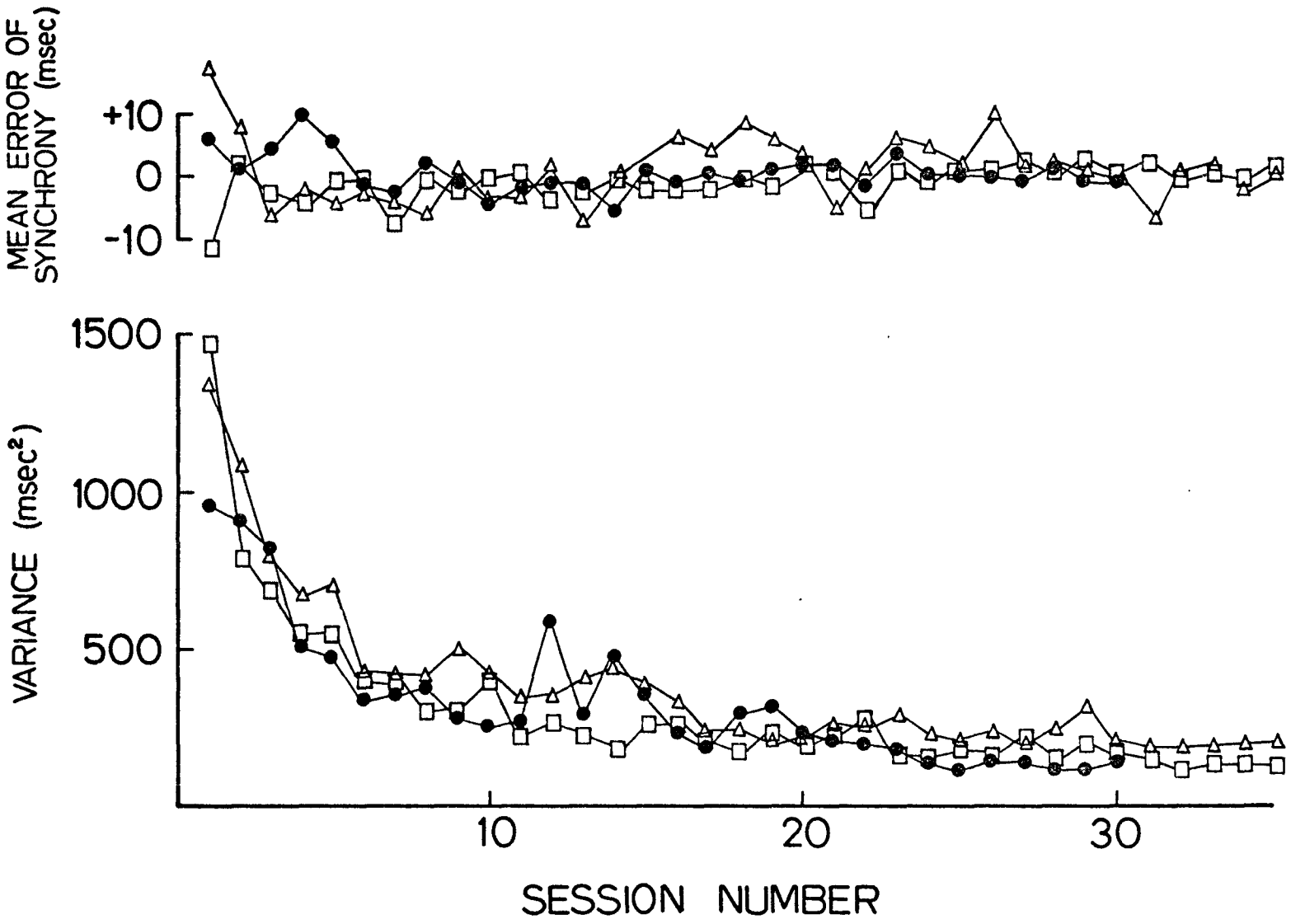
When the parameter values obtained from the regression analysis of all data combined are transformed

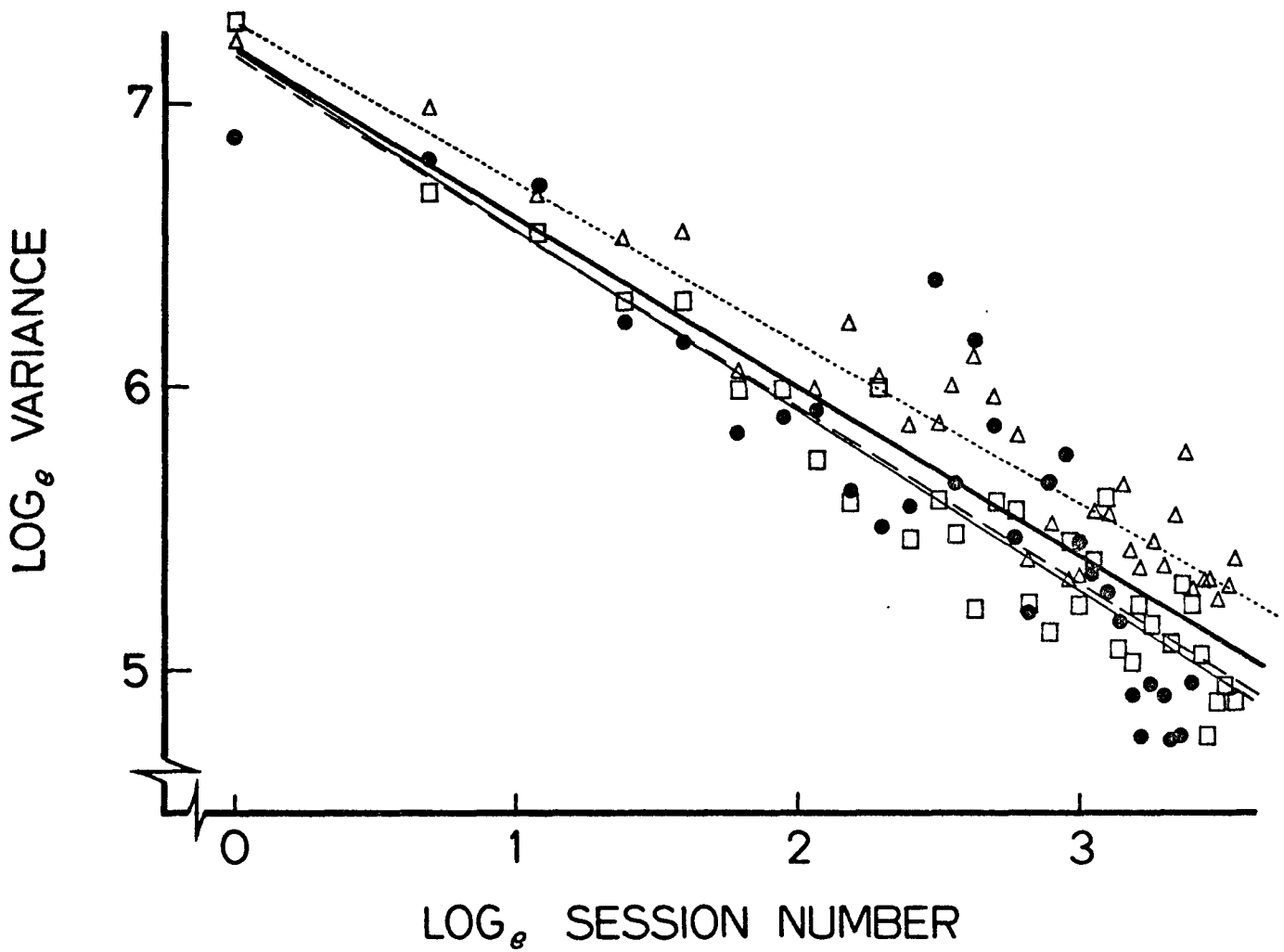
Figure 3 - Decrease in mean within-block variance as a function of practice for five subjects at three different P1P2 intervals. Solid lines are for subjects G.H. (filled circles) and J.B. (filled triangles) with a P1P2 interval of 460 msec. Dashed lines are for subjects C.H. (open circles) and G.T. (open triangles) at 310 msec and the broken line is for subject M.W. at 360 msec.

Figure 4 - Mean error of synchrony (top) and mean within-block variance (bottom) as a function of practice. Filled circles are for subject G.T. (P1P2 interval = 310 msec), open triangles for subject M.W. (P1P2 interval = 360 msec), and open squares for subject J.B. (P1P2 interval = 460 msec). Only 30 sessions are plotted for subject G.T. because he was given one of the stimulus manipulations 5 sessions earlier than the others.

Figure 5 - Best linear fits and scattergrams of the acquisition data, presented in Figure 4, plotted on log-log coordinates. Solid line and filled circles are for subject G.T. (P1P2 interval = 310 msec), open triangles and dotted line are for subject M.W. (P1P2 interval = 360 msec), and open squares and dashed line are for subject J.B. (P1P2 interval = 460 msec). The bold solid line represents the best fit to all the data combined.


500
400
300
200
100
VARIANCE (msec²)
2
4
6
8
10
12
14
GROUP OF 20 SESSIONS


MEAN ERROR OF SYNCHRONY (msec)
+10
0
-10
VARIANCE (msec²)
1500
1000
500
10
20
30
SESSION NUMBER


7
6
5
0
1
2
3
LOG$_e$ VARIANCE
LOG$_e$ SESSION NUMBER

back to the original coordinates, extrapolated to include larger amounts of practice, and plotted; the result is the acquisition curve shown in Figure 6. The extrapolation, based on only the first 35 sessions, seems to provide a fairly accurate description of the diminishing returns observed with prolonged practice. Similar extrapolations based on each subject's initial acquisition performance were calculated and compared with the actual data obtained. Some of these comparisons are shown in Table 4. It is clear that this model of acquisition, based on only the first 35 sessions of practice, is able to give fairly accurate predictions of subsequent performance even after 175 sessions of practice. For, in spite of the fact that the predicted variances are consistently less than those obtained, when based on the five sessions worth of data combined in Table 4, the correspondence is much closer when the best session variance in each five session set is compared with that predicted.

Subjects G.H. and G.T. exhibited the most stable and similar performance throughout the experiment, or at least after the first 20 sessions. Consequently, the parameter values obtained for G.T. were used to predict the performance of G.H. at various points in practice. The correspondence was extremely good. For example, extrapolation out to session 270 resulted in a predicted response latency variance of 35.5 compared to the observed

Table 3

Regression Analysis of Data from Log-Log Plots of Acquisition Performance for the Three Naive Subjects in Experiment 1.

Subject	Slope	Intercept	r	r^2	SE
G.T.	-0.64	7.19	-0.89	0.79	0.29
M.W.	-0.57	7.29	-0.95	0.90	0.16
J.B.	-0.63	7.18	-0.96	0.92	0.17
Overall	-0.60	7.20	-0.90	0.81	0.24

Note. SE refers to standard error of the estimate of y on x and r^2 represents the proportion of variance accounted for by the regression.

Table 4

Log-Log Extrapolation of Synchronization Performance with Comparisons of Observed vs. Predicted Response Latency Variances at Selected Levels of Practice Under Baseline Conditions.

		Variance	
Subject	Sessions	Predicted	Observed
G.T.	131-135	60.5	63.3
	176-180	50.2	54.1
J.B.	121-125	63.6	84.7
M.W.	101-105	106.0	114.9

Note. Variance refers to mean within-block variance for the sessions tabled and is expressed in $msec^2$. Predicted variances are obtained from extrapolation of the best-fitting linear function (see Figure 5) obtained for each subject's acquisition data when plotted on log-log coordinates.

value of 34.3 msec^2 (mean within-block variance for sessions 266-270). These findings not only provide strong support for the proposal that acquisition performance can be well described by a power function, but also indicate that relatively early acquisition performance can provide quite accurate predictions of subsequent performance, even after almost ten times the initial amount of practice.

It is obvious that the power function relationship must break down at some point or one would have to predict zero variance with sufficient practice. Presumably this limit is imposed by the underlying physiology involved with producing response-stimulus synchronization behaviour. That the lowest variance of 34.3 msec^2 is within the range of predicted values for minimum response latency variance, in this type of task, suggests that this limit may be imminent.

Shape of Response Latency Distributions

Performance similarities at different P1P2 intervals were evident not only in terms of variances but also in terms of shape of response latency distributions, as shown in Figures 7 and 8. Each distribution represents five sessions, or 1,500 responses, at equivalent levels of practice. However, P1P2 intervals and antecedent conditions of synchronization training are different for the subjects involved, as shown in Table 1. Figure 7 is obtained from sessions 176 to 180 for subjects G.T. and G.H. with P1P2

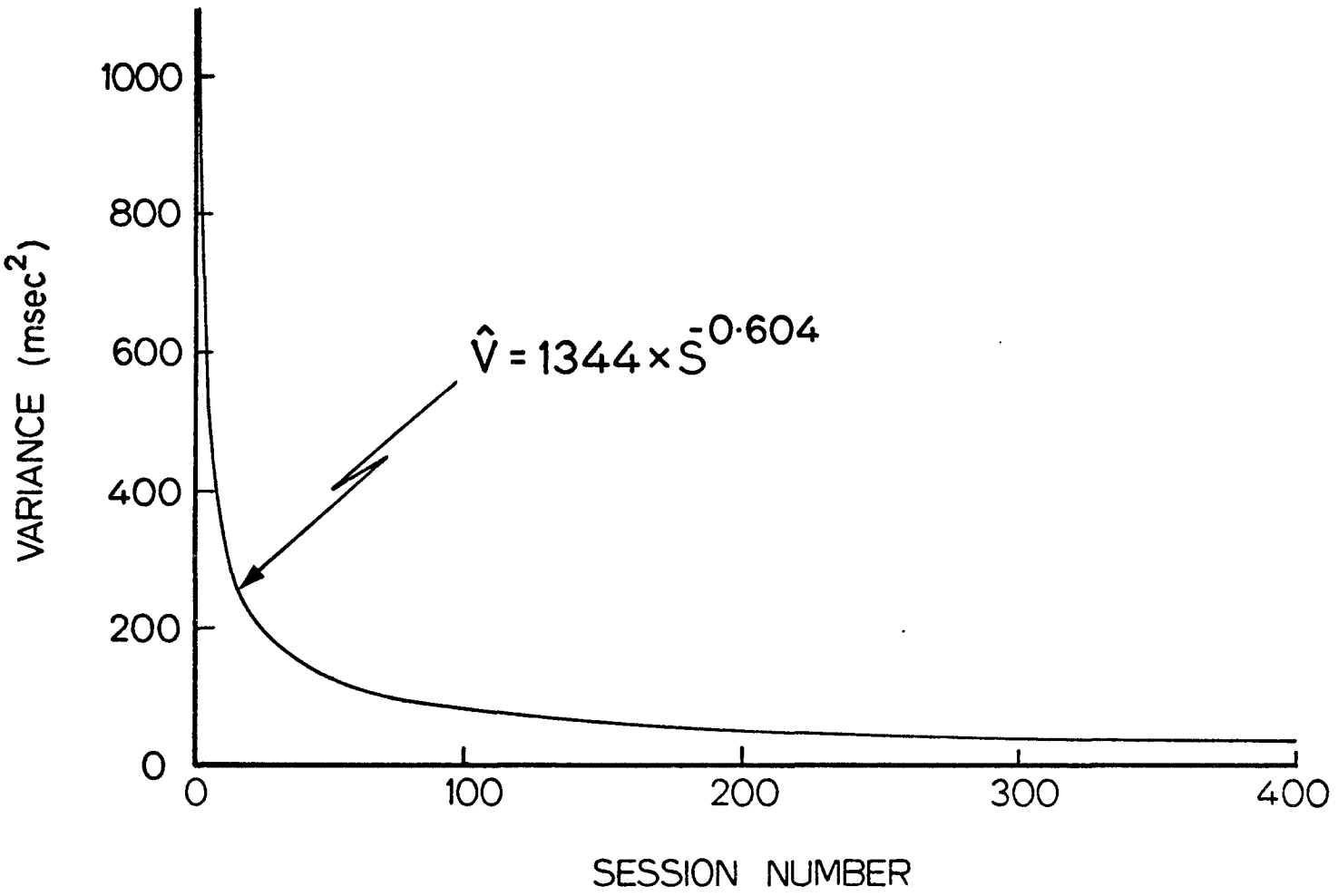
intervals of 310 and 460 msec respectively. Figure 8 is for subjects C.H. and J.B. with P1P2 intervals of 310 and 460 msec, respectively, after 121 to 125 sessions of practice. Each of the distributions is very symmetrical and sharp-peaked with short tails. All responses fall within either a 50 msec (Figure 7) or 60 msec (Figure 8) time window centred about the time-point corresponding to P2 onset. In the case of the former, 90% of the response latencies fall within a 24 msec time window.

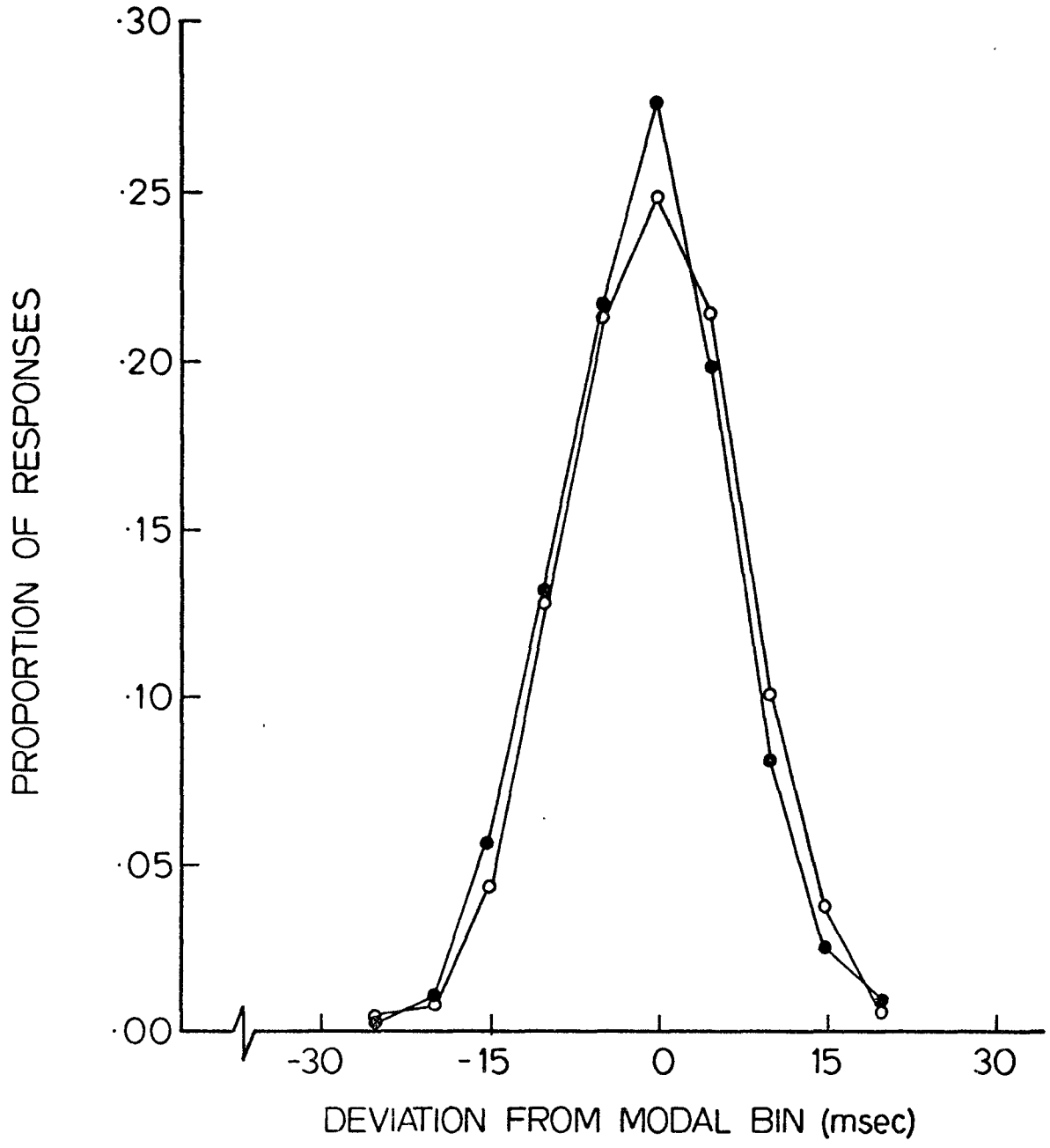
Mean within-block variances and total variances for each of the distributions plotted in Figures 7 and 8 are presented in Table 5. The small discrepancies between these two variance measures attests to the stability of the data and indicates that changes in responding both between blocks and between sessions are negligible. Mean response latency and coefficient of variation for each distribution are also shown in Table 5. It is clear that standard deviation is not a constant proportion of the mean in this task. Furthermore, these ratios are much lower than the estimates of 0.060 to 0.100 obtained by Getty (1975) for duration discrimination and by Woodrow (1930, 1933) for time estimation. The lowest previous estimate of the ratio between standard deviation of response latencies and mean latency is 0.023, obtained by Wing and Kristofferson (1973a) using interresponse times. The important point is that this ratio is not constant in synchronization performance, at

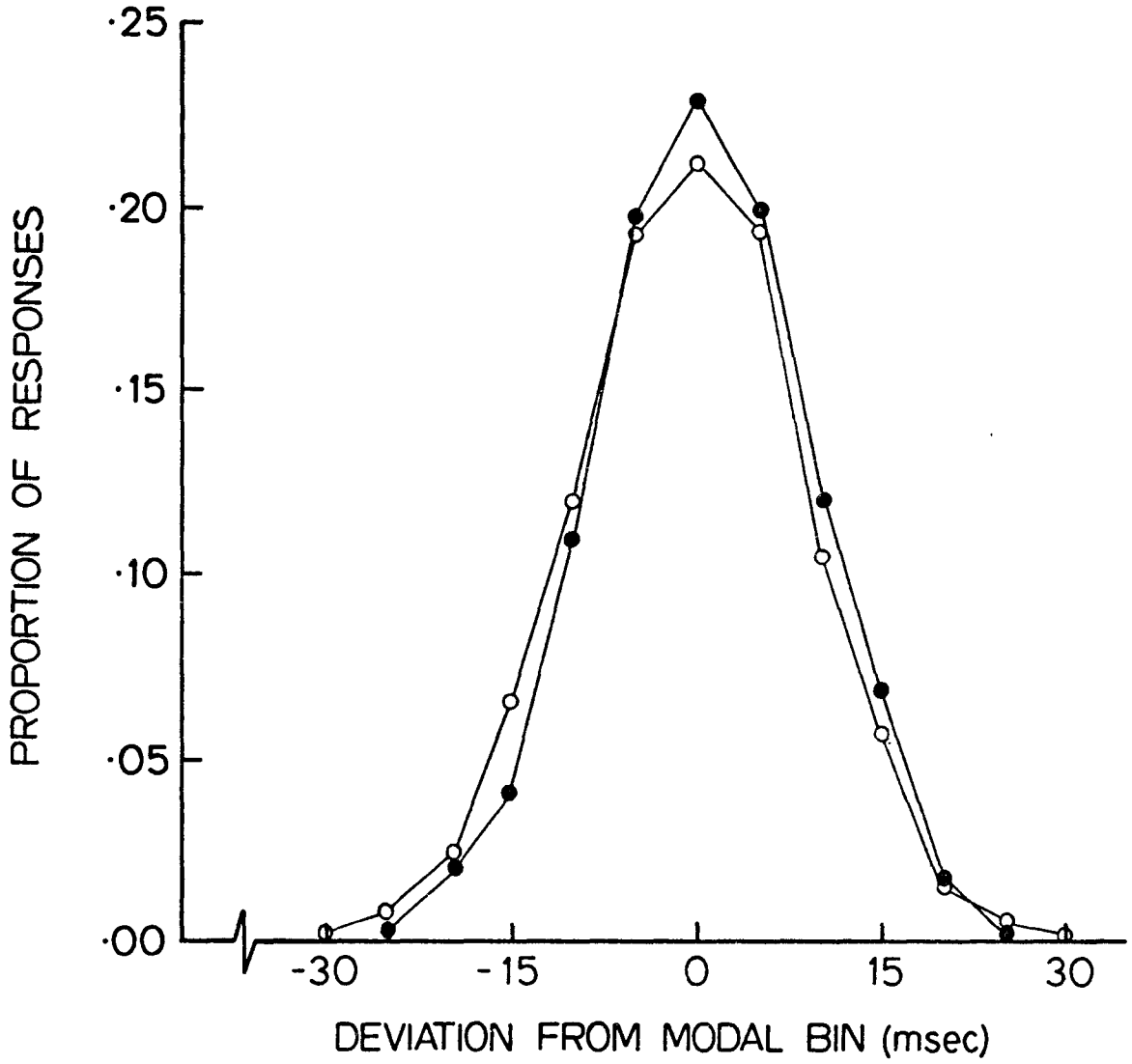
Figure 6 - Predicted, typical acquisition curve for variance measures of performance in the type of response-stimulus synchronization task used in Experiment 1. V refers to predicted within-block variance and S is the session number.

Figure 7 - Superimposed relative frequency distributions from sessions 176-180, with 1,500 responses each, showing similarities in performance for two subjects with different P1P2 intervals. Open circles are for subject G.T. (mean = 308, total variance = 57, mean within-block variance = 54, P1P2 interval = 310 msec). Filled circles are for subject G.H. (mean = 461, total variance = 52, mean within-block variance = 51, P1P2 interval = 460). Bin size is 5 msec.

Figure 8 - Superimposed relative frequency distributions from sessions 121-125, containing 1,500 response latencies each, for subjects C.H. with filled circles (mean = 309, total variance = 73, mean within-block variance = 72, P1P2 interval = 310) and J.B. with open circles (mean = 460, total variance = 86, mean within-block variance = 85, P1P2 interval = 460). Bin size is 5 msec.


1000
800
600
400
200
0
VARIANCE (msec²)
$\hat{V} = 1344 \times S^{-0.604}$
0
100
200
300
400
SESSION NUMBER


·30
·25
·20
·15
·10
·05
·00
PROPORTION OF RESPONSES
-30
-15
0
15
30
DEVIATION FROM MODAL BIN (msec)


·25
·20
·15
·10
·05
·00
PROPORTION OF RESPONSES
-30
-15
0
15
30
DEVIATION FROM MODAL BIN (msec)

least over the range of P1P2 intervals tested here. Rather, absolute performance is constant, such that the ratio of standard deviation to mean response latency becomes increasingly small the longer the synchronization interval.

Low Variances and Effect of Foreperiod Variability

Low variances, defined as the lowest mean within-block variance obtained for a group of five consecutive sessions under the baseline condition (P1P2), are shown in Table 6 for each subject. Effects of practice are evident across subjects and seemingly independent of the mean, as discussed earlier. All response latency variances tabled are well below earlier estimates of minimum S-R latency variance from RT studies. The lowest value of 34.3 $msec^2$ even reduces the minimum S-R latency variance obtained by Kristofferson (1976), using a synchronization task, by a factor of 3. Average mean error of synchrony, neglecting sign, associated with these low variances is 1.2 msec, with the largest being 2.5 msec for subject G.T. There is no indication of a consistent bias for all subjects to respond early or late but some small bias is observed for a couple of individuals. Session means generally vary less than ±2 msec, providing further evidence of the stability of performance in this task.

Figure 9 shows the low variance response latency distribution obtained for subject G.H. on the 5 sessions

Table 5

Mean Within-Block Variance vs. Total Variance Plus Means and Coefficients of Variation for Each of the Distributions in Figures 7 and 8.

	Variance			
Subject	Within-Block	Total	Mean	Coeff. of Var.
G.T.	54.1	56.8	307.5	.024
G.H.	51.2	51.8	460.8	.016
C.H.	72.3	73.4	308.5	.028
J.B.	84.7	86.2	460.4	.020

Note. Variances are expressed in $msec^2$. Coefficient of variation is calculated by dividing standard deviation by the mean.

Table 6

Low Variances Obtained During Subject-Paced Synchronization

Subject	Sessions	Mean	Variance	SE
M.W.	101-105	360.2	114.9	0.28
J.B.	121-125	460.4	84.7	0.24
C.H.	121-125	308.5	72.3	0.22
G.T.	176-180	307.5	54.1	0.19
G.H.	266-270	461.1	34.3	0.15

Note. Variances are mean within-block variances in $msec^2$, the mean is in msec, and SE refers to standard error of the mean.

numbered 266 to 270. All 1,500 responses are included and the figure is plotted using 3-msec bins. This serves to emphasize the extreme regularity and symmetry of the distribution. Mean response latency is 461.1 msec with a mean within-block variance of 34.3 msec^2 and total variance of 34.6 msec^2. The minimum spread between the two variance measures indicates again that changes between blocks and between sessions contribute little to the variance of the overall distribution. The error of synchrony is only 1.1 msec with the mode occurring just slightly after P2 onset. All responses fall within a 35 msec time window, 90% within a 19 msec window, and 50% of all responses occur within ±4.5 msec of the median. The ratio of standard deviation to mean is less than 0.013. The distribution could be well described by the convolution of an isoceles triangle with a base of 24 msec combined with a low variance component which is normally distributed (cf. Kristofferson, 1976; Kristofferson & Allan, 1973).

With respect to the effects of foreperiod variability, Figure 10 shows two relatively low variability, response latency distributions obtained for subject G.H. One distribution is based on five sessions of synchronization performance under the baseline condition, in which the foreperiod is variable, and the other represents five sessions of responding with a constant foreperiod. The level of practice is about the same. It is obvious that any

effect of foreperiod variability is negligible in terms of mean, variance, and/or shape of the response latency distribution at these high levels of performance. The baseline distribution has a mean of 461.2 msec and a mean within-block variance of 46.2 msec^2 while the constant foreperiod distribution has a mean of 461.8 and variance of 44.3 msec^2. Total variances are 47.6 and 45.1 msec^2 respectively. Because both distributions are highly symmetrical, with no responses occurring more than 25 msec after P2 onset, the indication is that all responses are triggered only by P1 and that the shape is not affected in any direct way by the placement of P2. The shape can be described as sharp-peaked, with straight sides and narrow skirts.

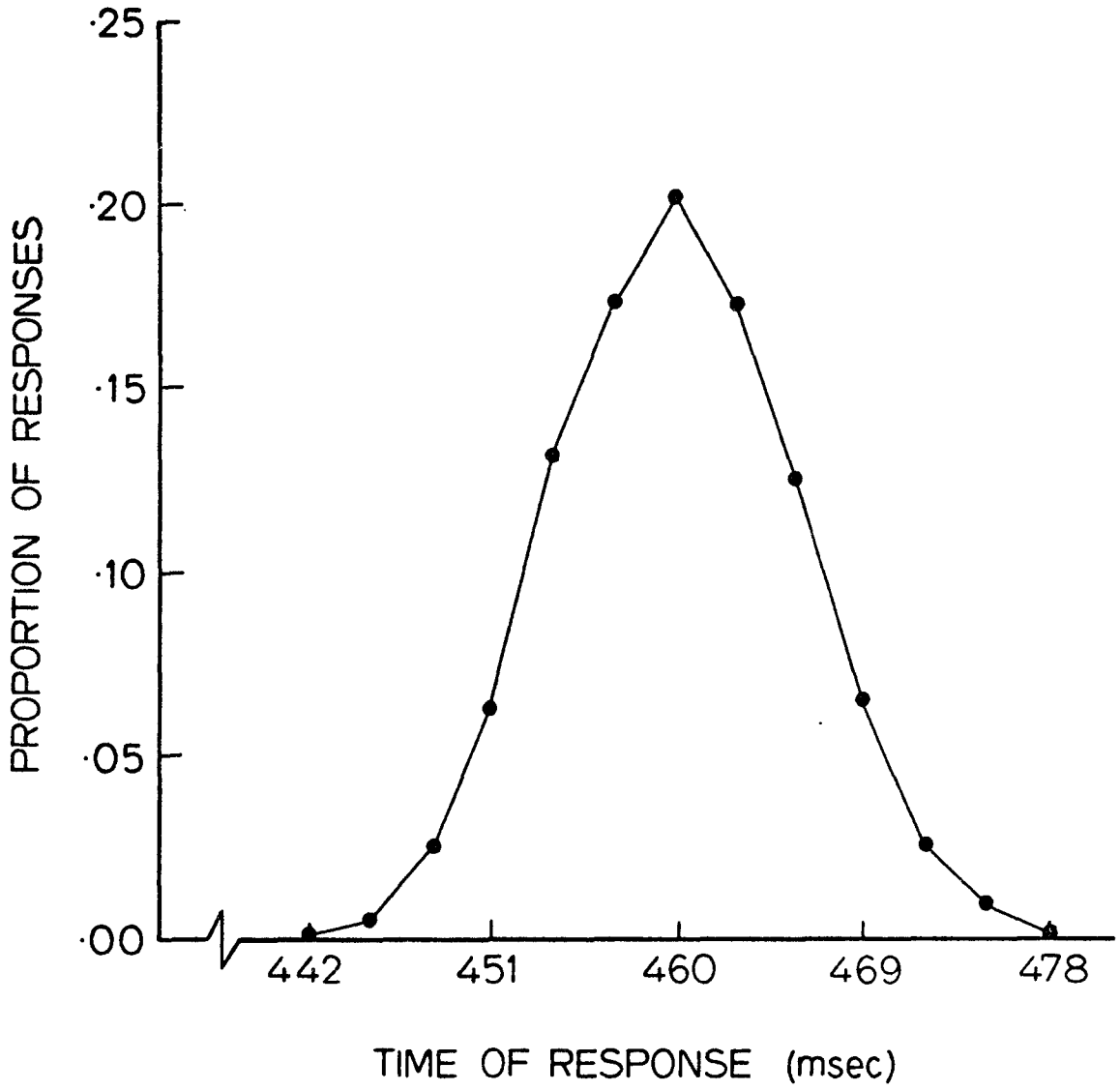
Performance with P2 Only

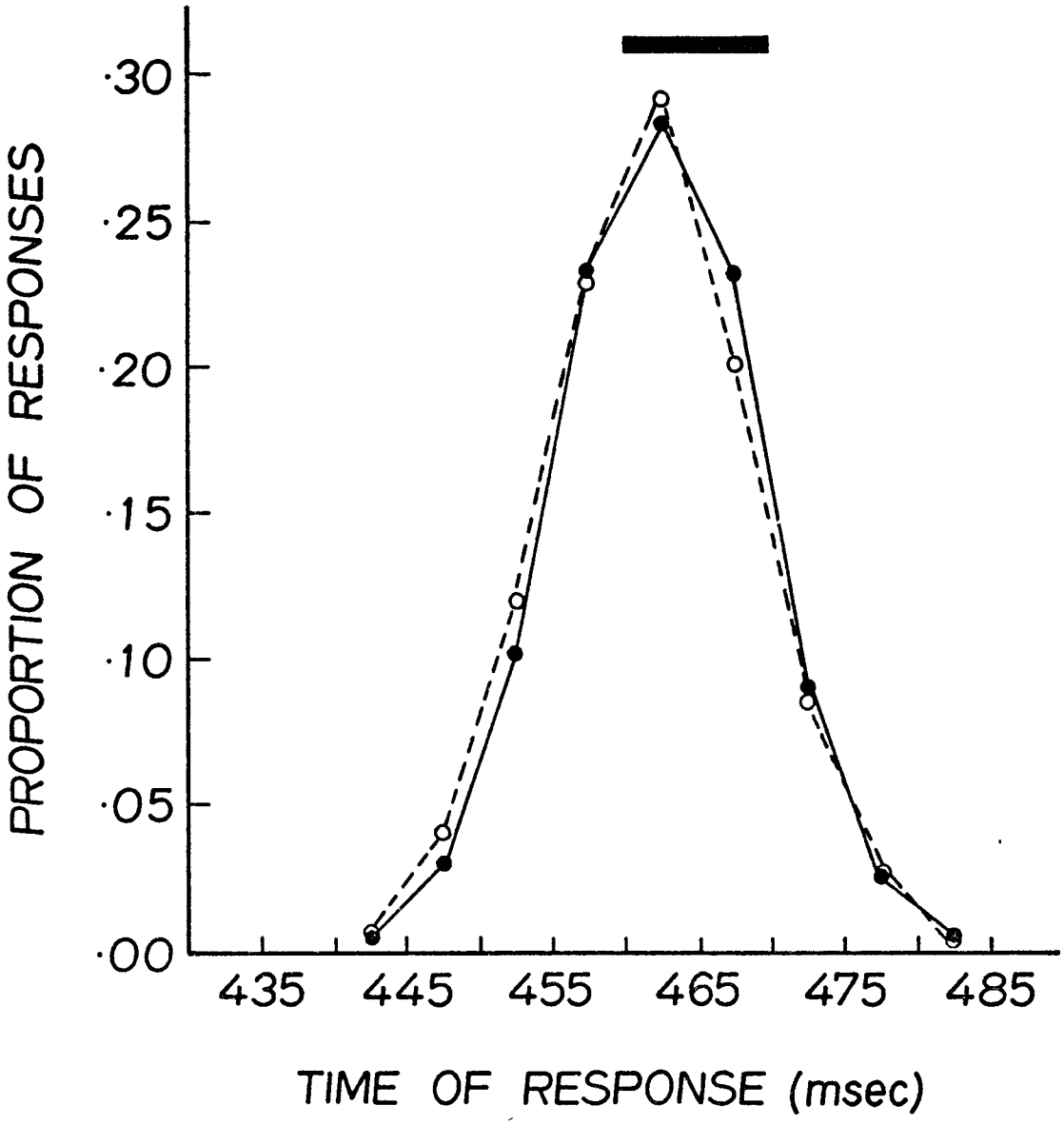
The initial auditory pulse, P1, was removed from the stimulus sequence in order to ascertain the role of P1 in controlling the timing of the synchronization responses. It should be noted that subjects were unaware of the existence of a foreperiod.[1] Their subjective impression was that P1 was generated instantaneously by their initiation response. This illusion was maintained even with the constant foreperiod, in which case the delay between R*i* and P1 was 91 msec. Under the "P2 only" condition, due to technical considerations, response latencies and synchronization

[1]Subject G.H. knew about FP but had same introspections.

Figure 9 - Low-variance, relative frequency distribution containing 1,500 response latencies, for subject G.H. on sessions 266-270 with a P1P2 interval of 460 msec (mean = 461, total variance = 35, mean within-block variance = 34, variable foreperiod). Bin size is 3 msec.

Figure 10 - Relative frequency distributions, of 1,500 response latencies each, for subject G.H. for two different foreperiod variability conditions with a P1P2 interval of 460 msec. Solid line: mean = 462, total variance = 45, mean within-block variance = 44, constant foreperiod. Dashed line: mean = 461, total variance = 48, mean within-block variance = 46, variable foreperiod. Rectangle marks time of occurrence of P2.


·25
·20
·15
·10
·05
·00
PROPORTION OF RESPONSES
442
451
460
469
478
TIME OF RESPONSE (msec)


·30
·25
·20
·15
·10
·05
·00
PROPORTION OF RESPONSES
435
445
455
465
475
485
TIME OF RESPONSE (msec)

intervals were still measured relative to the time-point T*o* which corresponds with P1 onset when P1 is present. This time-point is not defined for the subject under this condition. Thus, if timing control of R*s* were transferred from P1 to some aspect of R*i*, not only would the effective synchronization interval be increased, by an amount equal to the foreperiod duration, but latency variance would also include a component equal to the variability of the foreperiod. An example of the extreme situation is diagrammed in Figure 11. On two successive trials, in the absence of P1, the subject produces the same response latency relative to his initiation response (R*i*), however, the foreperiods differ. On trial *n* the shortest foreperiod possible happens to be presented such that T*o* and the initiation of timing of P2 occur relatively soon after R*i*. In this case, the measured response latency is greater than the P1P2 interval (time from T*o* to P2 onset) resulting in a positive error of synchrony. On the next trial (*n+1*), the longest foreperiod possible is presented which, by analogous logic, produces a measured response latency with a negative error of synchrony. Thus two things happen on "P2 only" trials if the subject times from his initiation response: (1) a variance component equal to the foreperiod variability is added into the measurement of response latencies; and (2) the feedback becomes somewhat erroneous. The first factor provides the means for determining which stimulus is

controlling the timing of the synchronization response, RS. If the subject uses P1 to time RS, when P1 is present, then the variance observed under "P2 only" conditions should increase by an amount equal to the foreperiod variability associated with the condition since *Ri* must be used when P1 is absent. On the other hand, if *Ri* is used for timing RS, even when P1 is available, then no change in variance should be expected.

The results of P1 removal are shown in Table 7. Subjects experienced this manipulation at different points in practice, accounting for the range of values tabled under baseline variance. The interesting result is that differences in response latency variance between "P2 only" and baseline (P1P2) sessions are remarkably similar across subjects. These differences, with a variable foreperiod, range from 30.7 to 38.1 $msec^2$; the mean difference being only 1.1 $msec^2$ greater than that predicted by the simple addition of mean baseline variance with the experimentally induced component of variance (33.3 $msec^2$) associated with the variable foreperiod. With a constant foreperiod used in the "P2 only" condition, baseline and "P2 only" variances do not differ. These results indicate that when P1 is present, it controls timing of the synchronization response; but when P1 is absent, the control of timing can be transferred, all but perfectly, to some aspect of the initiation response. Any changes in performance observed following this transfer

Table 7

Comparison of Performance with the Baseline (P1P2) Condition vs. Performance with "P2 Only" and the Constant Foreperiod "P2 Only" Conditions.

		Variance		
Condition	Subject	Condition	Baseline	Difference
P2 Only	G.H.	100.4	64.8	35.6
	G.T.	84.8	54.1	30.7
	C.H.	105.3	72.3	33.0
	M.W.	153.1	115.0	38.1
P2 Only(CFP)	C.H.	70.4	72.3	-1.9
	M.W.	117.2	115.0	2.2

Note. Variances are mean within-block variances expressed in $msec^2$. Average difference for the first condition was 34.4 $msec^2$ and for the second the difference was 0.15 $msec^2$. CFP means constant foreperiod.

of control can be accounted for by the additional component of foreperiod variability which is added into the response latency measurements.

As mentioned earlier, the transfer of control to some aspect of **R***i* under the "**P**2 only" condition, entails increasing the effective synchronization interval by an amount equal to the foreperiod duration. The additional delay required for synchronization with a variable foreperiod averages 61 msec while that required with a constant foreperiod is 91 msec. It is interesting to note that, in spite of these increases in the effective synchronization interval, mean response latency was unchanged even during the first block of trials following the transition. Thus, the internally timed delays, presumably underlying synchronization performance, appear to be quickly and accurately adjustable. Earlier work (Kristofferson, 1976) had shown that 10 msec changes in the **P**1**P**2 interval could be accommodated with very little deterioration of performance. In the present instance, however, changes in the synchronization interval of almost 100 msec are easily accommodated as shown in Figure 12 by the similarity in response latency distributions obtained for subject C.H. under baseline and constant foreperiod "**P**2 only" conditions. With the exception of the flattened peak on the baseline (**P**1**P**2) distribution, the two plots are nearly superimposed. The baseline distribution has a mean

of 308.5 msec and a variance of 72.3 msec² relative to a mean of 309.1 msec and variance of 70.4 msec² for the other distribution. Response latencies obtained with "P2 only" and the variable foreperiod are shown in the third distribution. It has more of a spread because it includes the foreperiod variance, as discussed earlier. The mean is 305.8 msec and the variance is 105.4 msec².

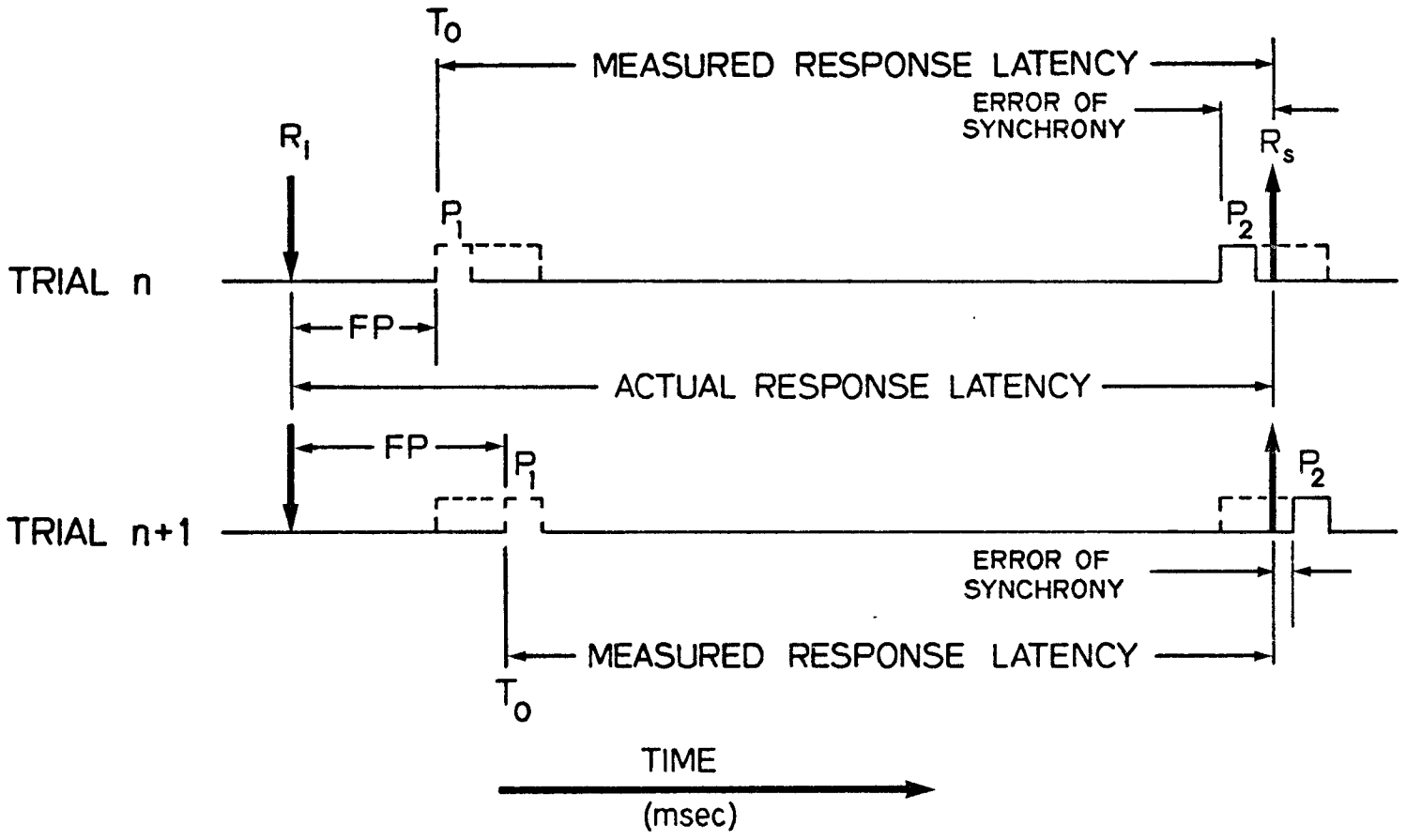
Performance with P1 Only

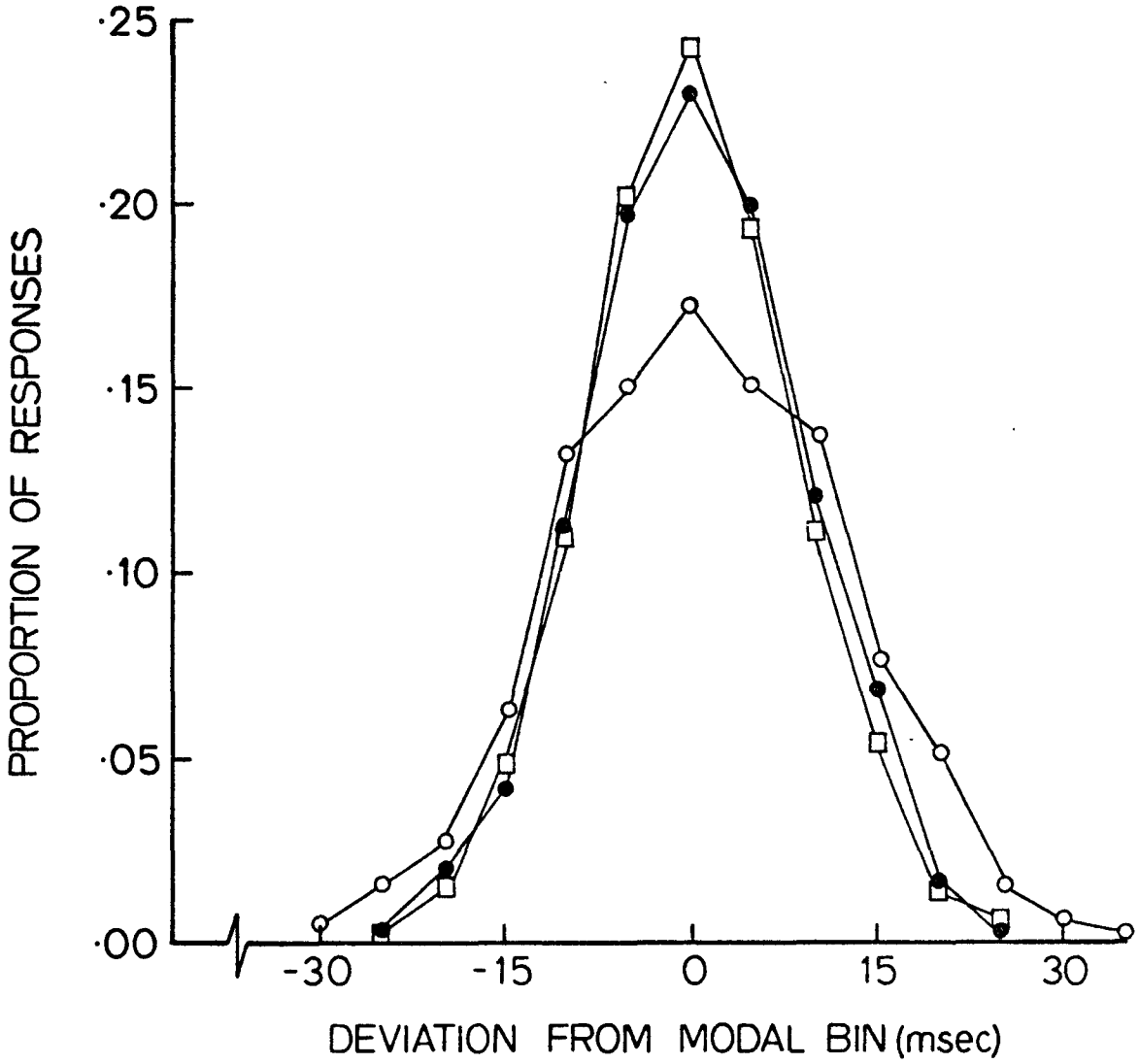
Removal of P1 had little effect on synchronization performance. However, the same was not true when P2 was removed. Even with the delayed feedback still available for response evaluation, performance was severely disrupted for subjects G.H., C.H., and J.B. immediately upon transfer to the "P1 only" condition. Response latency variances increased three to four times over baseline (P1P2) levels. Continued practice under this condition was typically characterized by further degradation of performance followed by gradual improvement. Subject G.T. was the exception and exhibited little difficulty with this condition. After only 20 sessions, his performance was quite stable. Mean response latency variance for the last five sessions under the "P1 only" condition was 92.2 msec² relative to a postcondition baseline variance of 63.3 msec².

Improvements in performance for the other subjects were extremely slow and frustrating. Consequently, only

Figure 11 - Diagram of the sources of ambiguous feedback and experimentally induced increases in measured response latency variance when timing of RS has to be made from R*i* in the absence of P1 (P2 only condition) and the foreperiod is variable from trial to trial. Dashed rectangles indicate range of temporal placements, relative to R*i*, of T*O* and P2. Note that these two events are always time-locked regardless of condition.

Figure 12 - Relative frequency distributions, of 1,500 response latencies each, for subject C.H. under baseline (filled circles), "P2 only - constant foreperiod" (open squares), and "P2 only - variable foreperiod" (open circles) conditions. P1P2 interval is 310 msec. Means and variances are given in the text.


T0
MEASURED RESPONSE LATENCY
ERROR OF SYNCHRONY
R1
Rs
P1
P2
TRIAL n
FP
ACTUAL RESPONSE LATENCY
FP
P1
P2
TRIAL n+1
ERROR OF SYNCHRONY
MEASURED RESPONSE LATENCY
T0
TIME
(msec)


·25
·20
·15
·10
·05
·00
PROPORTION OF RESPONSES
-30
-15
0
15
30
DEVIATION FROM MODAL BIN (msec)

subject G.H. persevered until a stable level of performance was obtained. This required 55 sessions, at which point his mean response latency variance was 92.5 msec^2 relative to a postcondition baseline variance of 51.2 msec^2. The results for both G.H. and G.T. are shown in Table 8. The difference in variances is 41.3 msec^2 for G.H. and 28.9 msec^2 for G.T. Since these values were so close to the 30 to 35 msec^2 range of variance differences observed between baseline and "P2 only" conditions, it was thought that subjects under the "P1 only" condition might simply be ignoring P1, in the absence of P2, such that the additional variance observed could be accounted for by the subject timing from the initiation response with its associated 33.3 msec^2 foreperiod variability. To test this idea, the foreperiod was made constant for subject G.H. No change in performance occurred. Mean response latency variance for the last five sessions with P1 only and a constant foreperiod was 92.4 msec^2. The results from this series of conditions for subject G.H. are shown in Figure 13. Furthermore, when both P1 and P2 were removed from the stimulus sequence for G.T., the difference in observed variance, relative to baseline, increased to 57.8 from 28.9 msec^2 suggesting that the effect of P1 removal is independent and additive to the effect of P2 removal. However, the nature of the effect caused by deletion of P2 is unclear. It may simply serve some immediate, perceptual feedback role which cannot be totally

Table 8

Comparison of Performance with the Baseline (P1P2) Condition vs. Performance with "P1 Only", "P1 Only" with a Constant Foreperiod, and "No P1 or P2" Conditions.

		Variance		
Condition	Subject	Condition	Baseline	Difference
P1 Only	G.H.	92.5	51.2	41.3
	G.T.	92.2	63.3	28.9
P1 Only(CFP)	G.H.	92.4	51.2	41.2
No P1 or P2	G.T.	111.9	54.1	57.8

Note. Variances are mean within-block variances expressed in $msec^2$. Average difference for the first condition was 35.1 $msec^2$. CFP means constant foreperiod.

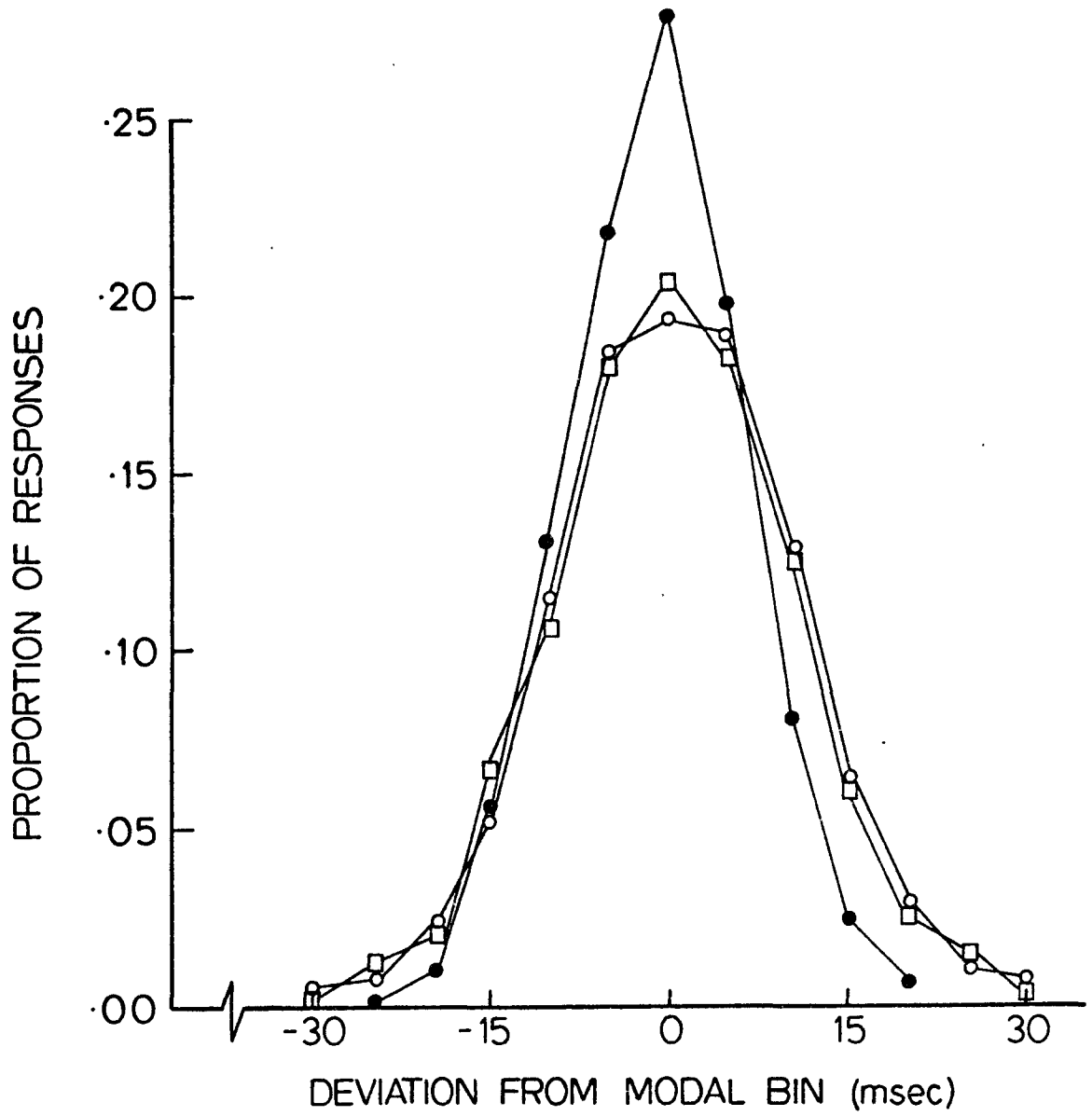
compensated for by the delayed feedback.

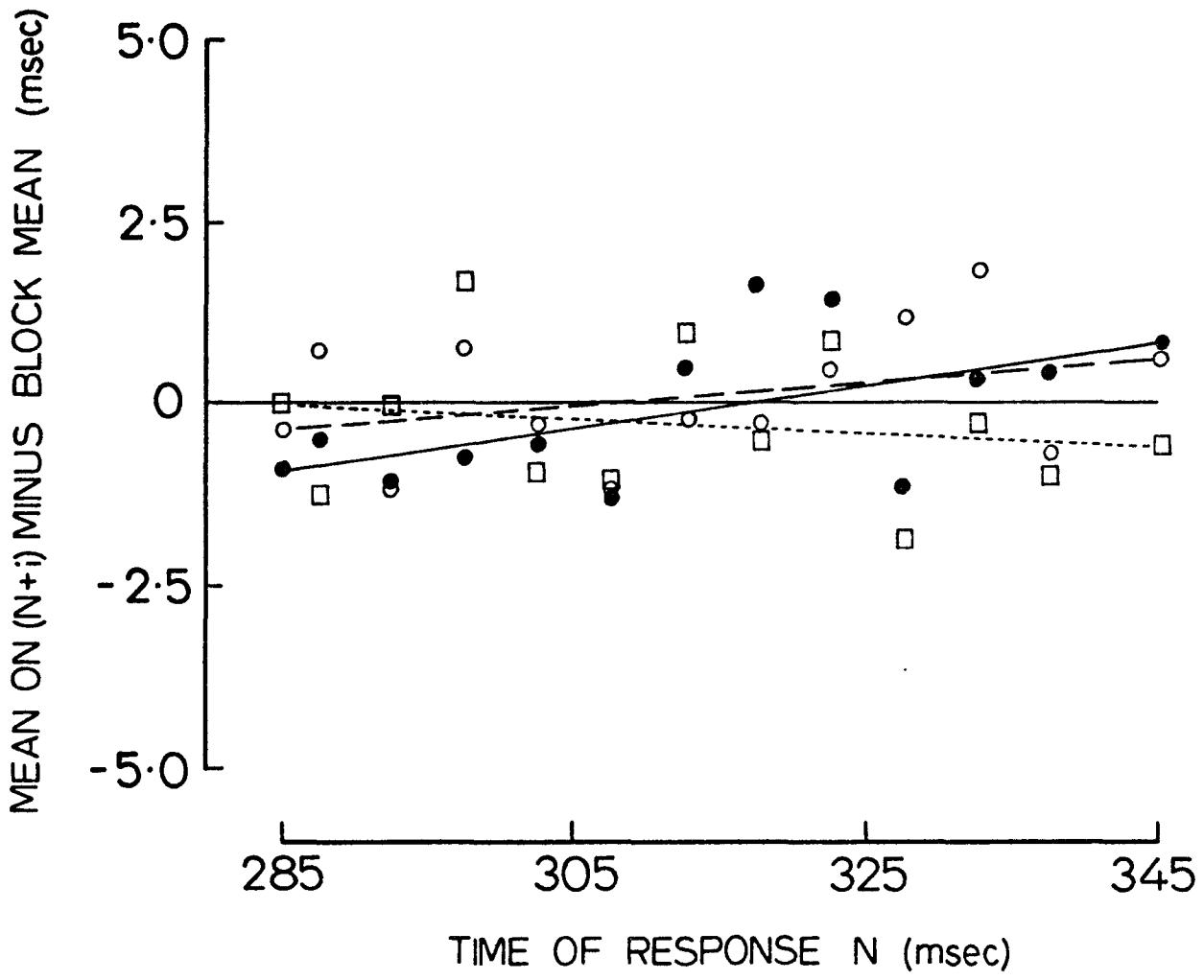
Sequential Dependencies

Early in acquisition, subjects exhibited strong positive dependencies between successive response latencies such that perseveration of early or late responding over several trials was quite common. By sessions 26 to 30, however, this sequential dependency was limited to only immediately succeeding trials. An example of an autocorrelation analysis applied to the data from G.T. at this level of practice, is shown in Figure 14. The straight lines are best-fits to the temporal autocorrelation data obtained at lags 1 to 3. Significance of dependencies was evaluated by constructing a 2x2 contingency table with early-late on trial n versus early-late on trial $n+i$, i = 1, 2, or 3, and calculating the Chi-square value. Only the data points for lag 1 have a significant positive slope (Chi-square = 7.17 on 1 d.f.), indicating a weak positive sequential dependency that has no effect beyond the immediately succeeding response. As practice continued, this positive dependency diminished to the point where no evidence of a temporal autocorrelation between successive response latencies remained. This independence of response latencies is shown in Figures 15 and 16 for four subjects after relatively prolonged practice. The two curves in Figure 15 show the results of lag 1 autocorrelation analyses

Figure 13 - Relative frequency distributions, of 1,500 response latencies each, for subject G.H. under baseline (filled circles), "**P**1 only - constant foreperiod" (open circles), and "**P**1 only - variable foreperiod" (open squares) conditions. **P**1**P**2 interval is 460 msec. Variances for the distributions are given in Table 8.

Figure 14 - Autocorrelation analyses (lags 1 through 3), of 1,500 response latencies each, for subject G.T. relatively early in acquisition (sessions 26-30). Data points show deviation from the block mean on trial $(n+i)$, i = 1, 2, or 3, given that the response on trial n was of the latency on the abscissa. Slopes of best-fitting lines and associated Chi-square values (see text) are: lag 1-filled circles, solid line (slope = .030, Chi-square = 7.17); lag 2-open circles, dashed line (slope = .016, Chi-square = 3.44); lag 3-open squares, dotted line (slope = -0.010, Chi-square = 1.73).


·25
·20
·15
·10
·05
·00
PROPORTION OF RESPONSES
-30
-15
0
15
30
DEVIATION FROM MODAL BIN (msec)

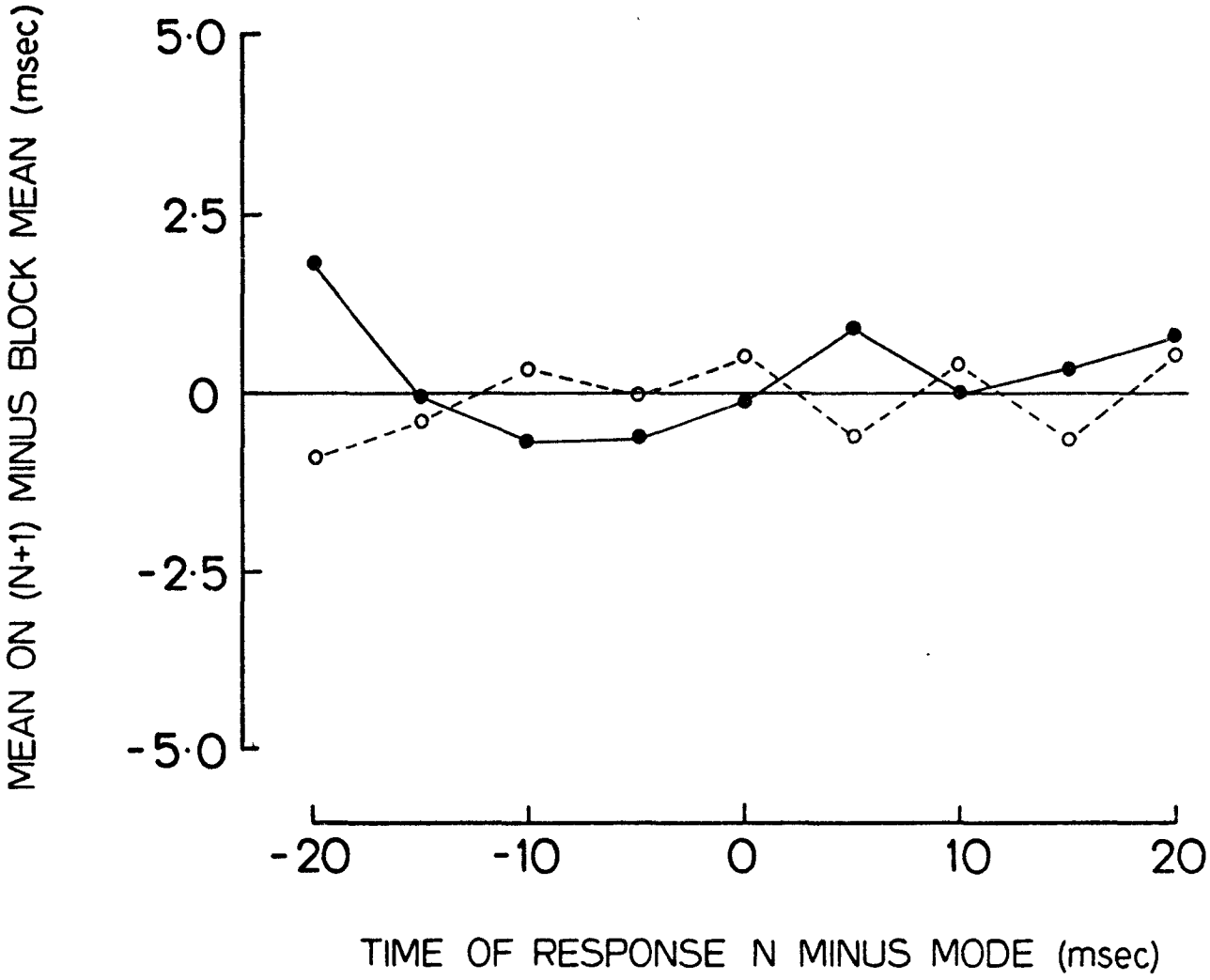

MEAN ON (N+i) MINUS BLOCK MEAN (msec)
5·0
2·5
0
-2·5
-5·0
285
305
325
345
TIME OF RESPONSE N (msec)

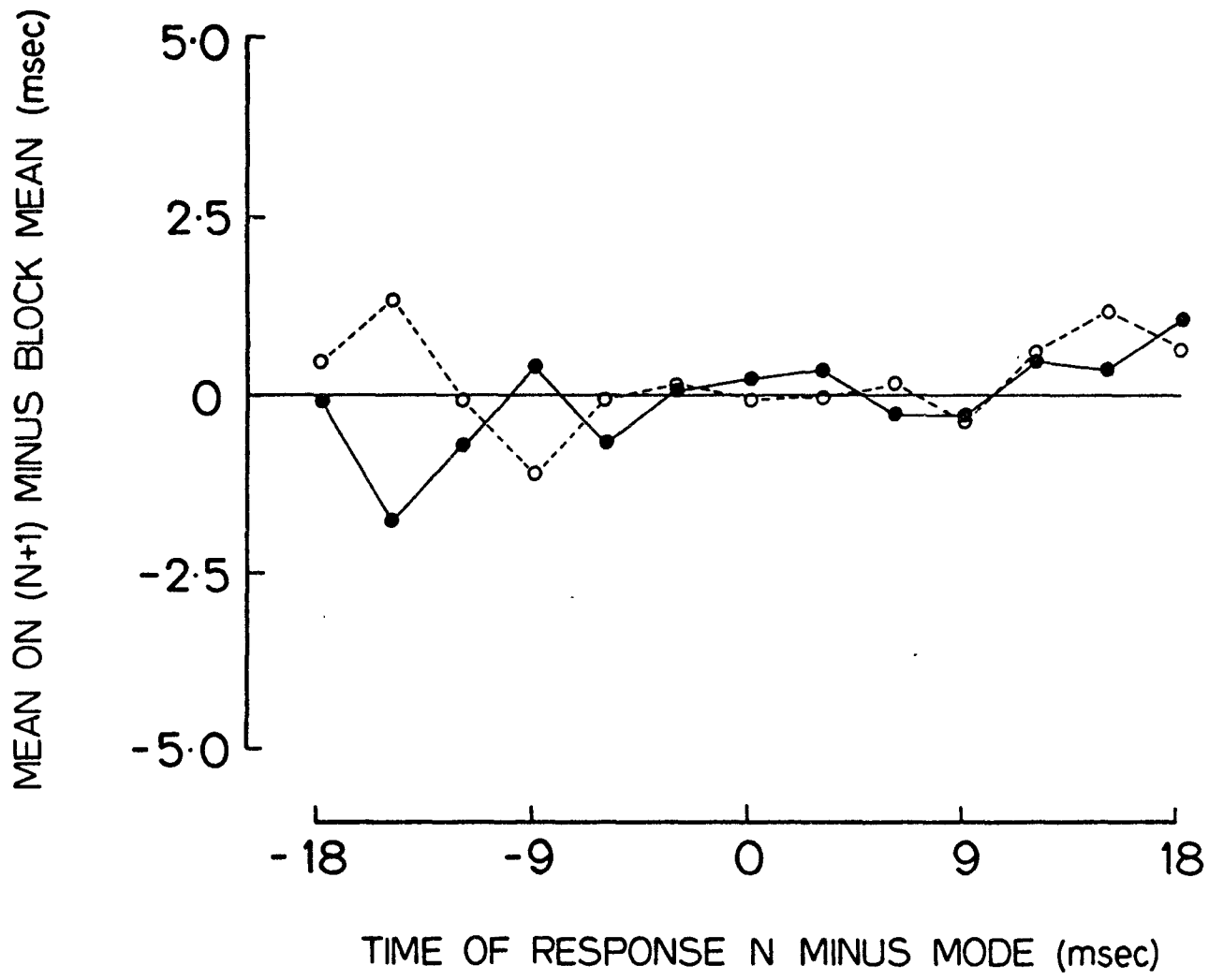
for subjects J.B. and C.H. as applied to the 1,500 responses obtained from each during sessions 121 to 125. Results of similar analyses conducted on the data from sessions 176 to 180 for subject G.T. and sessions 206 to 210 for subject G.H. are shown in Figure 16. In all cases, time of response on trial *n+1* is independent of the response latency on trial *n*, as indicated by the flat autocorrelation plots.

Sequential analyses were also performed on the data from the "P2 only" condition which yielded results indicating a weak autocorrelation only for subject C.H. while the data from the other three subjects on this condition generated autocorrelation coefficients which were not significantly different from zero. Under this condition, with a variable foreperiod, the subject is in effect timing his synchronization response from a time-point that has some temporal variability associated with it relative to the time of P2 onset. This means that feedback under these conditions is not always appropriate. That is, for a given accurately timed, internal delay, the feedback signal will always vary as a function of the foreperiod duration. If such erroneous information were taken into account by the subject on each trial, response latency variances associated with the "P2 only" condition would be expected to be inflated beyond the levels observed, which are simply the addition of baseline and foreperiod variance components. The failure to find this additional increase in

Figure 15 - Lag 1 autocorrelation analyses, of 1,500 response latencies each, for subjects J.B. (filled circles) and C.H. (open circles). Flat plots indicate no sequential dependencies between successive response latencies. Figure shows mean deviation from the block mean of response latencies on trial $(n+1)$ given that the deviation of the preceding response from the mode was of the value given along the abscissa.

Figure 16 - Lag 1 autocorrelation analyses, of 1,500 response latencies each, for subjects G.H. (filled circles) and G.T. (open circles) indicating that successive response latencies are independent of one another. Data points show mean deviation of response $(n+1)$ from the block mean given that response n deviated from the mode by the value given along the abscissa.


MEAN ON (N+1) MINUS BLOCK MEAN (msec)
5·0
2·5
0
-2·5
-5·0
-20
-10
0
10
20
TIME OF RESPONSE N MINUS MODE (msec)


MEAN ON (N+1) MINUS BLOCK MEAN (msec)
5·0
2·5
0
-2·5
-5·0
-18
-9
0
9
18
TIME OF RESPONSE N MINUS MODE (msec)

variance or a consistent autocorrelation, suggests that feedback is being ignored, at least on a trial-to-trial basis, although some averaging of feedback signals may be taking place over many trials. Such averaging could provide reliable information for maintenance of the internally timed delay required for accurate synchronization.

C. Discussion

These results clearly demonstrate that, with special procedures, the upper bound on minimum S-R latency variance can be reduced substantially below levels provided by previous research. Response latency variance values of less than 35 $msec^2$ represent a reduction in variance by a factor of 3 over those values obtained by Kristofferson (1976), indicating that the changes in procedure incorporated into the present experiments were instrumental in reducing variances further. It is not clear, however, which of the several changes were responsible for this improvement.

It is interesting to note that the lowest variance value obtained in the experiment is well within the range of minimum latency variance values predicted by Kristofferson's (1976) synchronization model, with estimates for the central and efferent delay variances derived from duration discrimination and interresponse timing experiments, respectively. The asymptotic performance of G.H. corresponds to a q-value of 12 which agrees well with the

minimum *q*-value of 12.5 obtained by Kristofferson (1980). Whether further practice would have significantly reduced response latency variances beyond those levels observed is not known, but the model suggests that these low variances are at, or very near, the limit.

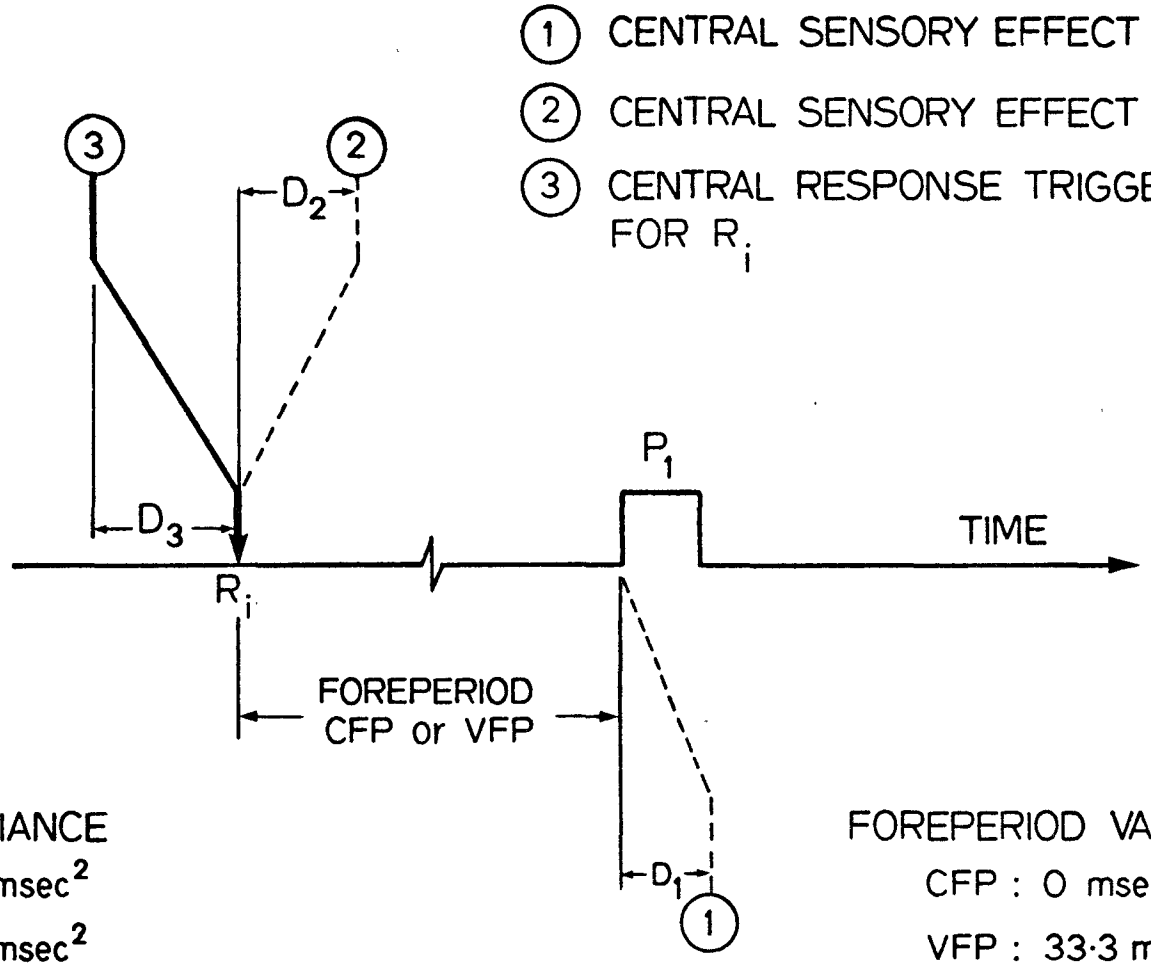
The present results also support Kristofferson's (1976) proposal for the existence of deterministic internal delays; that is, adjustable delays within the processing system which can be inserted into S-R chains without any associated decrease in the temporal certainty of the response. However, not only is the variance of response latencies independent of mean latency, but so is rate of acquisition, which indicates that longer delays are no more difficult to establish than shorter ones. This is true over a range of synchronization intervals from 310 to 550 msec, if the constant foreperiod data, obtained with "P2 only", is taken into account (P1P2 interval of 460 msec plus 91 msec foreperiod duration), which corresponds to the region where Kristofferson observed equivariance performance.

Information regarding the locus of these internally timed delays is not directly forthcoming from this experiment, but some of the data suggest further speculation. Kristofferson (1976) has proposed that deterministic delays are contained in the afferent latency of the stimulus. The "P2 only" results can be interpreted as providing some additional support for this notion, or at

least for the notion of zero-variance afferent latencies. Transfer of timing control for synchronization responding from auditory to tactile stimulation had no effect on variance measures of performance. In order to obtain such a result, if afferent latencies were variable, one would have to assume that the afferent latencies associated with auditory and tactile stimuli have exactly the same variability. Such an assumption does not seem to be as parsimonious as simply assuming zero variance.

Is it possible that the control of timing of the synchronization response could be accomplished by something other than the central sensory effects of the initiation response and, therefore, nullify such reasoning? It does not appear so. As shown in Figure 17, in the absence of P1 there are only two possible strategies for this timing control: use of the time-point associated with central registration of the tactile stimulation following the response (labelled as 2 in the figure) or of that associated with the central response trigger preceding the response (labelled 3). With a constant foreperiod and use of the first strategy, the time-point associated with initiation of central timing is perfectly time-locked (assuming zero-variance afferent latencies) to the electronic switching involved with initiation of the P1P2 interval such that the delay between commencement of objective and subjective timing is constant. However, such is not the

Figure 17 - Diagram of different theoretical possibilities for triggering the timing control of subjects' synchronization responses showing the delays and variabilities associated with each. Under "P2 only" conditions, only possibilities 2 and 3 are available to the subject.



DELAY VARIANCE

D_1 : ≈ 0 msec^2

D_2 : ≈ 0 msec^2

D_3 : 10 – 50 msec^2 (WING & KRISTOFFERSON 1973b)

FOREPERIOD VARIANCE

CFP : 0 msec^2

VFP : 33·3 msec^2

case for the second type of timing control. The time-point associated with the central response trigger has some temporal variability with respect to the time-point of the electronic switching produced by the initiation response due to variability of the efferent delay separating these two events. Thus, use of the response trigger for timing would entail the addition of another variance component into the observed response latency distribution. If one assumes this variance to be of the same order of magnitude as the estimate provided by Wing and Kristofferson (1973b), then the second possibility (strategy number 3 in Figure 17) for timing control must be rejected based on the finding of no increase in variance under the "P2 only" condition.

Once established, synchronization performance is remarkably unaffected by major changes in procedure. Subjects can accommodate alterations in the synchronization interval of nearly 100 msec and are able to transfer the control of synchronization timing from an auditory to a tactile stimulus. The fact that cross-modal transfers of this nature are possible in this task suggests that the internally timed delays underlying synchronization performance might serve a quite general and useful purpose in acts of information processing within the central nervous system.

A problem is: How are these accurate, internally timed delays established and maintained? It is clear that

some form of feedback is being effectively used early in acquisition because mean response latencies centre on the point of veridical synchrony almost right from the start. Yet it is also evident that, later in practice, response latencies are independent of feedback, at least on a trial-to-trial basis. This suggests that the accuracy of the internally timed interval is maintained by an averaging process that integrates information over a large number of trials. If it is further assumed that the sample size increases with practice, such a strategy could explain many of the sequential dependency results obtained. With a small sample size, the standard error of the mean is large. Therefore, early in practice, strong positive dependencies between response latencies would be expected due to a slow wandering of the mean during a session. However, with a large sample of feedback information, the standard error of the mean would be quite small, resulting in stable responding from block-to-block and session-to-session, as observed.

There are two types of feedback information available to the subject: the delayed feedback provided by the experimenter, and the immediate perceptual feedback, derived from the temporal relation between the central sensory effects caused by **P**2 and those of the subject's synchronization response. The question concerning how these sources of information are used, both during acquisition and

in maintenance of steady-state performance, is not answered by this experiment.

In the next experiment, the role of auditory, delayed feedback is examined when it is used in conjunction with visual synchronization interval markers.

III. Experiment 2 - Synchronization Performance with Visual Interval Markers

In spite of the extremely stable results obtained in Experiment 1, it was unclear as to what was being learned in the response-stimulus synchronization task and what role the different types of feedback were playing during acquisition. One approach to gaining some insight into these problem areas is to look at the effects on performance of transferring a well-practiced subject to another stimulus modality. Such a transfer was, in part, accomplished in Experiment 1 by removal of P1 from the stimulus sequence. Under this condition, it was clear that subjects were able to transfer control of their synchronization response to the tactile sensory information, produced by their initiation response, without any associated decrement in performance. However, the question of interest here is whether the modality of P2 can be changed and still assume the important feedback role it played in Experiment 1. A transfer of this kind would also provide information concerning the generality of synchronization training.

There are several things that possibly contribute to the improvements in performance observed during acquisition. These include: (1) processing the stimuli marking the interval more efficiently; (2) interpreting the feedback information with more precision; (3) timing out the internal

delay more accurately; and (4) fine tuning the response mechanism so as to minimize efferent delay variance. Following acquisition, however, a change in stimulus modality of the interval markers should only affect some of these learning factors. Obviously the stimulus markers must be processed differently, which will also affect the interpretation of the immediate perceptual feedback derived from a temporal order discrimination of P2 and the subject's synchronization response. But, if the delayed feedback signal format is kept the same, all other aspects of learning should be constant across experiments. Therefore, any differences in performance following transfer must be attributed to differences in processing the synchronization stimuli. These differences are independent of the subject's abilities to: (1) process the delayed feedback information; (2) generate a precise response sequence; and (3) time out an appropriate internal delay for the response. Remember that even though a change in stimulus marker modality may alter the duration of the internal delay required, as a result of a longer or shorter afferent latency, it is clear from Experiment 1 that such changes can be easily accommodated with no change in performance. Thus, if there is substantial transfer such that the large gains in performance typically observed during initial acquisition do not result, then those gains must be attributed to either refining the response side of the S-R chain or learning how

to interpret the delayed auditory feedback.

The use of a well-practiced subject in the transfer task also afforded the opportunity to examine any processing differences between visual and auditory stimuli because several studies suggest that important and measurable differences do exist. The bulk of this literature points to a difference in the input or afferent latency variability. According to Michon (1967) the ear is the superior sensory receptacle for temporal information and several other studies support this contention (e.g., Efron, 1973; Goldstone & Lhamon, 1971; and Goodfellow, 1934).

Lhamon and Goldstone (1974) report eleven experiments which all indicate a striking difference between the auditory and visual modalities with respect to judgments of short durations. They used the psychophysical methods of paired-comparison and absolute judgment, with amount of information transmitted as their dependent measure, and repeatedly found higher levels of information transmission with auditory durations than with visual durations. This suggests less variability is associated with the auditory system and this intersensory difference remains despite large changes in stimulus properties and other aspects of the psychophysical context.

Tanner, Patton, and Atkinson (1965), using a two-alternative, forced-choice procedure (shorter or longer), recorded number of correct intramodality judgments

of signal duration. The stimulus was either a 1000 Hz tone or bright light from a glow-modulator tube and durations ranged from 0.5 to 1.6 seconds. They found performance to be significantly better in the auditory modality.

More relevant to the present study are data obtained by Bartlett and Bartlett (1959) using a type of synchronization task as described in the introduction. When 2 msec light flashes were substituted for the auditory clicks the standard deviation of the response latencies just about doubled, changing from about 20 msec to nearly 40 msec with the visual markers. This difference was consistently obtained for synchronization intervals ranging from 167 to 500 msec which suggests that deterministic delays are still available with the visual stimulus markers; but that the processing of these markers involves some variability.

Obviously the limit on temporal discrimination is a function of the uncertainty with which the instant of onset of the stimulus marker is detected, as well as that imposed by variability in the timing process itself. As mentioned earlier, afferent latencies in the auditory modality are assumed to have negligible variance based on the work of Divenyi (1976). Similar work by Zacks (1973) with the visual system, however, indicates significant afferent latency variances associated with this modality. He used a two-alternative, forced-choice procedure to examine the ability of human subjects to discriminate which of two

spatially separated flashes of light came first. With careful consideration of the complex interactions that can occur between retinal locations, Zacks measured performance as a function of the onset asynchrony between the flashes needed for a given level of discrimination. Then, using an analysis derived from signal detection theory, the data were interpreted in such a way as to provide an estimate of the standard deviation of visual afferent latencies. These estimates ranged from 7.5 to 13.5 msec and agree well with similar estimates derived from work with cat retinal ganglion cells (Levick, 1973).

In light of the seemingly substantial difference between auditory and visual processing of temporal information, it was hoped that the use of a transfer paradigm, with a subject well-practiced in response-stimulus synchronization, might provide another independent estimate of the magnitude of this difference. This procedure, as pointed out earlier, would also provide cues as to what is being learned during acquisition of the skill. To extend this information, it also seemed desireable to observe the acquisition performance of naive subjects who had never experienced auditory response-stimulus synchronization. These were the primary motivating factors for this experiment.

A. Method

Subjects

Three students (two male and one female) from McMaster University served as subjects. One of these subjects (the author) was well-practiced, having participated in earlier segments of this ongoing research project. The two naive subjects were run to gain information about the nature of initial acquisition in a visual modality, response-stimulus synchronization task to which the veteran subject was transferred.

Procedure

The basic procedure was exactly the same as that outlined in Experiment 1 with the following exceptions: the ready signal was changed to a 2000 Hz tone and the interval markers (P1P2) were changed to brief light flashes. These alterations kept the ready signal and P1P2 markers in different modalities, as in Experiment 1. The delayed feedback, however, was maintained in the auditory modality so as to be consistent with the training of subject G.H. (transfer subject) in the previous experiment. This was done to reduce the number of variables that could be responsible for performance differences following transfer.

Immediate perceptual feedback was different from that in Experiment 1. Since P2 was changed to a visual marker, the temporal order discrimination became one between

the onset of the light flash, P2, and onset of the tactile stimulation produced by the subject's synchronization response, RS.

As before, both stimulus markers could be selectively omitted from the stimulus sequence to ascertain their respective roles in maintaining synchronization performance. To obtain this information, these manipulations also involved the use of both constant and variable foreperiods, in conjunction with the use of subject-paced trials, as in Experiment 1.

The brief light flashes, P1 and P2, were both 10 msec emissions from a diffused, green, light-emitting diode (L.E.D. - Fairchild FLV 360) 5.08 mm in diameter. Light output of the L.E.D. with a current of 20 mA was 3.2 mcd. A small grey box housed the L.E.D. which was located directly in front of the subject (eye level) at a distance of about 1 metre. Subjects were instructed to fixate the centre of the L.E.D. to ensure foveal stimulation. The sound-attenuated chamber was dimly lit such that the ambient illumination was near the threshold for photopic vision. Consequently, despite the low level of light output from the L.E.D., the light flashes appeared relatively bright and were quite salient to the subject.

B. Results

The P1P2 interval remained the same as in Experiment 1 for subject G.H. at a value of 460 msec. For the other two naive subjects, E.A. and V.A., the synchronization interval was fixed at 360 msec. As in the previous experiment, some experimental manipulations were repeated because the subjects' performances were not fully stable on their first exposure to the conditions which made the interpretation of the data difficult. A chronology of experience with the various stimulus manipulations used in the experiment is given in Table 9 for subjects E.A. and G.H. Subject V.A. ran for 95 sessions, all on the baseline (P1P2) condition with a variable foreperiod. (See note in Appendix A)

Initial Acquisition of Synchronization by Naive Subjects

Similar to the acquisition behaviour observed in Experiment 1, the performance of subjects E.A. and V.A. improved rapidly over the first 8 to 10 sessions, followed by increasingly slow improvement. The acquisition curves are shown in Figure 18.

Mean response latency quickly centres around the point of veridical synchrony and there is a general trend for mean error of synchrony to decrease as a function of practice with no indication of preferential responding, early or late, evident. Compared to the acquisition curves for subjects with auditory P1P2 intervals (see Figure 4)

Table 9

Chronology of Experimental Conditions in Experiment 2.

	Subject	
Condition	E.A.	G.H.
P1P2	1- 45	271-290
P2 Only	46- 55	291-295
P2 Only(CFP)	56- 65	296-305
No P1 or P2(CFP)	66 107	306-315
No P1 or P2(CFP) with Noise	--	316-330
P1P2	108-135	--
P1P2(CFP)	136-145	331-375
P1 Only(CFP)	146-165	--
P1 Only	166-175	--
No P1 or P2	176-180	--
P1P2(CFP)	181-185	--

Note. Numbers of the sessions devoted to each experimental condition are given in the body of the table. CFP means constant foreperiod. Subject V.A. ran 95 sessions on the baseline P1P2 condition with the variable foreperiod.

mean errors of synchrony tend to be larger with the visual interval markers and there is less evidence of stability in mean response latency even after 35 sessions of practice.

Decreases in response latency variance also appear to follow similar time courses for the two subjects. The initial differences are not reliable because of the manner in which the computer handles erroneous responses. Extremely aberrant responses are eliminated from analysis, as pointed out in the method section. Therefore, during the period of highly variable responding, early in practice, a subject making many aberrant responses (discarded from analysis) could conceivably obtain a lower calculated variance than another subject who exhibits only moderate variability in responding such that none of the responses is discarded from analysis. Although changes in performance, reflected by decreases in variance as a function of practice, follow a pattern similar to that obtained with the auditory modality, the absolute variance levels are higher for the visual modality throughout acquisition. Mean within-block response latency variance for the three naive subjects from Experiment 1, on sessions 26 to 30 combined, is 183.6 $msec^2$ compared to 243.9 $msec^2$ for the two subjects in this experiment at the same level of practice. Although this difference is substantial, 60.3 $msec^2$, the data are still sufficiently unstable at this point in practice to allow much in the way of an interpretation of this

difference. In spite of higher levels of variance, by session 12 both subjects exhibit response latency variances below the lowest levels previously reported for simple reaction time (Saslow, 1974).

As in Experiment 1, acquisition can be well represented by a power function as shown in Figure 19. This analysis includes the same data points as those in the lower panel of Figure 18 following a log-log transformation. The linear plots fit the data quite well, accounting for between 87 and 90% of the variance. Parameter values for the lines obtained from the regression analysis are provided in Table 10. Similarities in acquisition of the skill for the two subjects is evident from the comparable parameter estimates obtained. It is interesting to note that compared with the corresponding parameter estimates from Experiment 1 (see Table 3) only the intercepts seem to differ. The slope, or rate of acquisition, seems to be independent of stimulus modality while the initial level of performance seems to be worse for the visual modality.

It should be pointed out that this type of analysis does not necessarily indicate that zero variance should be reached at some point in practice. In other words, there is probably some lower limit which cannot be exceeded. The analysis does, however, provide a good representation of the data as it approaches this hypothetical limit and, as yet, no lower limit has been clearly demonstrated.

Table 10

Regression Analysis of Data from Log-Log Plots of Acquisition Performance for the Two Naive Subjects in Experiment 2.

Subject	Slope	Intercept	r	r^2	SE
E.A.	-0.61	7.46	-0.95	0.90	0.18
V.A.	-0.65	7.66	-0.93	0.87	0.22
Overall	-0.63	7.56	-0.94	0.87	0.21

Note. SE refers to standard error of the estimate of y on x and r^2 represents the proportion of variance accounted for by the regression.

Further along in synchronization training, a curious divergence in performance began to emerge, as shown in Figure 20. Subject V.A. exhibits very little, if any, improvement in synchronization performance following session 20 (actually session 13) while subject E.A. continues to decrease his response latency variance with no clear indication that any asymptotic level is ever reached. The results for subject E.A. are consistent with the findings from Experiment 1. For subject V.A., however, the data showed no net gain in performance over a span of 60 sessions. The variability in performance during this period was substantial though; mean within-block, sessional variances ranged from 165 to 393 $msec^2$. As a result of this instability, data collection with subject V.A. ceased at session 95, before she had been exposed to any stimulus manipulations. The existing data, however, are still important and will be considered later in the discussion.

This failure to improve with practice is very atypical in the current context, however, a similar observation has been reported by Kristofferson (1976) in which his performance during his first experiment was stable for 30 sessions. An important difference though was that Kristofferson's data were much more consistent from session to session.

Acquisition Performance of Transferred Veteran Subject

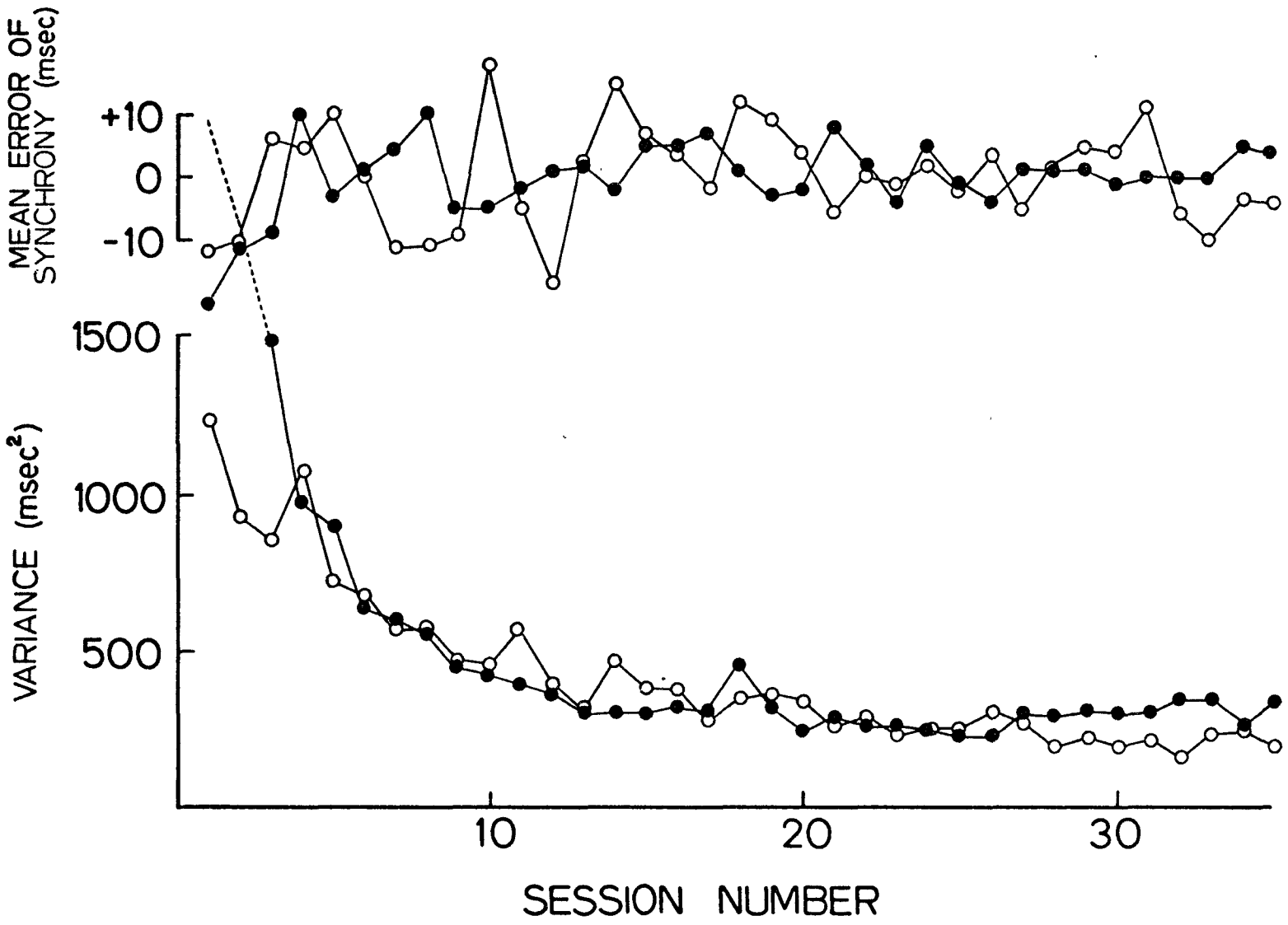
Subject G.H. was transferred to response-stimulus synchronization with visual interval markers after 270 sessions of practice with auditory markers. Prior to transfer his mean within-block variance for the last 5 sessions was 34.3 msec^2. The first session following transfer had a mean within-block variance of 109.5 msec^2 and the variance for the first 5 transfer sessions combined was 110.1 msec^2. These variance levels are higher than those obtained in Experiment 1 during the latter stages of practice. The only condition that comes close to equally degrading performance is the "no P1 or P2 - variable foreperiod" condition.

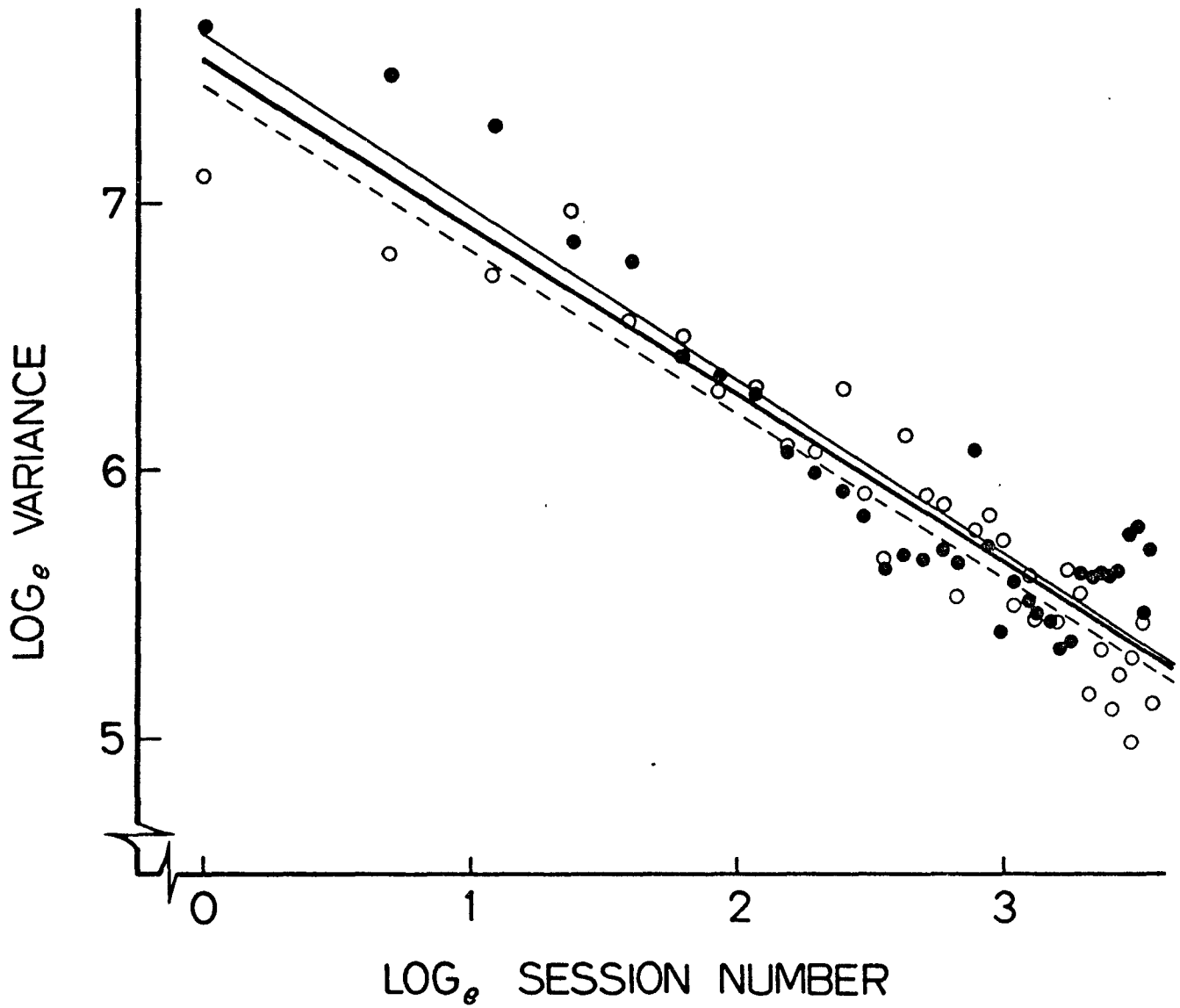
With continued practice, subject G.H. showed slow improvement in performance. Sessions 286 to 290 were used as baseline data for evaluating the effects of subsequent stimulus manipulations. At this point in practice, mean within-block variance was 85.0 msec^2 with an overall variance of 90.3 msec^2. This discrepancy between the two variance measures is much larger than for the baseline data obtained in Experiment 1. This indicates less stability of responding from block-to-block and session-to-session.

The results from an analysis for sequential dependencies using this baseline data set are shown in Figure 21. The graph shows the expected deviation from the block mean of the response latency on trial $n+i$, i= 1, 2, or

Figure 18 - Mean error of synchrony (top) and mean within-block variance (bottom) as a function of practice. Filled circles are for subject V.A. and open circles are for subject E.A. Both subjects had P1P2 intervals of 360 msec with visual interval markers.

Figure 19 - Best linear fits and scattergrams of the acquisition data presented in Figure 18 plotted on log-log coordinates. Solid line and filled circles are for subject V.A. Dashed line and open circles are for subject E.A. The bold solid line represents the best fit to both sets of data combined. P1P2 interval is 360 msec for both subjects.


MEAN ERROR OF SYNCHRONY (msec)
+10
0
-10
VARIANCE (msec²)
1500
1000
500
10
20
30
SESSION NUMBER


7
6
5
0
1
2
3
LOG$_e$ VARIANCE
LOG$_e$ SESSION NUMBER

3, given that the response latency on trial *n* falls within a particular bin range. Therefore, a positive slope indicates some degree of positive dependency between response latencies while a negative slope indicates a negative dependency. Significance of dependencies was tested using the Chi-square statistic described in Experiment 1. Only the data points for lag 1 have a significant positive slope (Chi-square = 5.08 on 1 d.f.) indicating a weak positive sequential dependency between only immediately successive response latencies.

In spite of the poorer performance, in terms of variance, the mean of the overall distribution of response latencies was still centred close to the point of veridical synchrony and highly symmetrical, as shown by the plot of filled circles in Figure 22. Mean error of synchrony is only 0.9 msec and all 1,500 responses fall within a 50 msec time window.

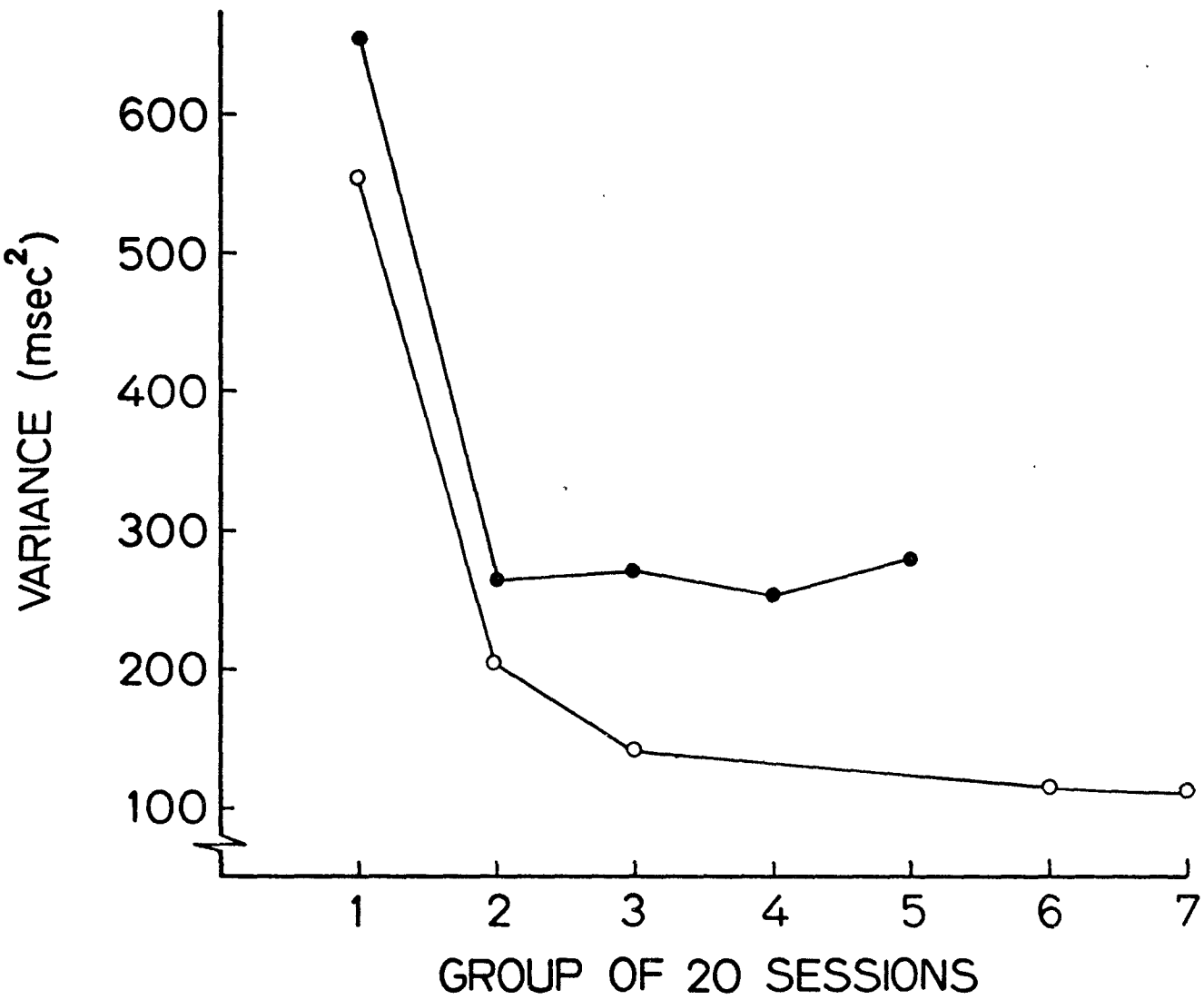
Performance with P2 Only

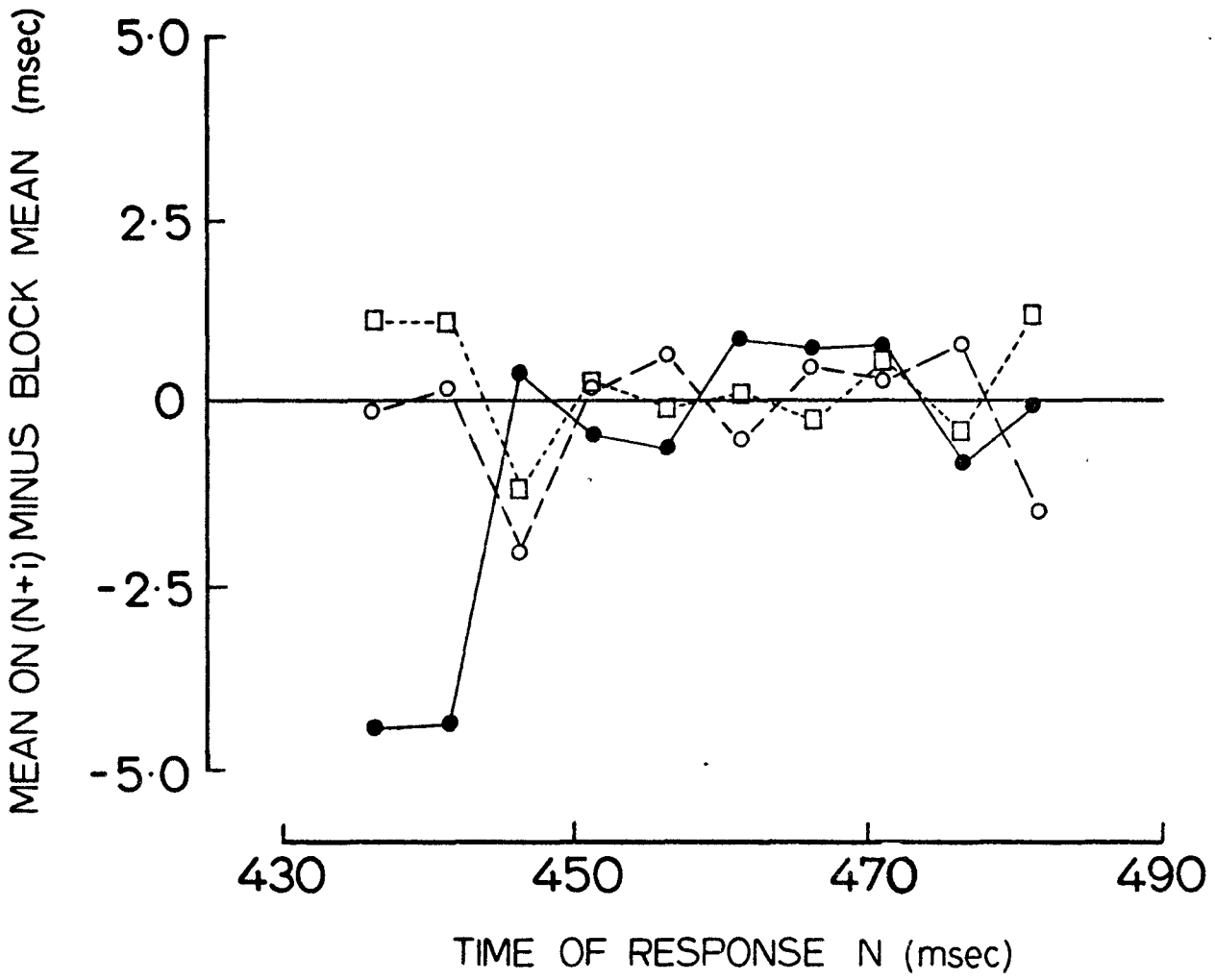
As in Experiment 1, the initial visual flash, P1, was removed from the stimulus sequence to ascertain its role in the control of synchronization timing. Analogous to the auditory situation, the foreperiod interval between R*i* and T*o* (see Figure 1) could be manipulated in such a way as to unambiguously identify whether P1 or the tactile concomitants of R*i* were being used as the source for timing

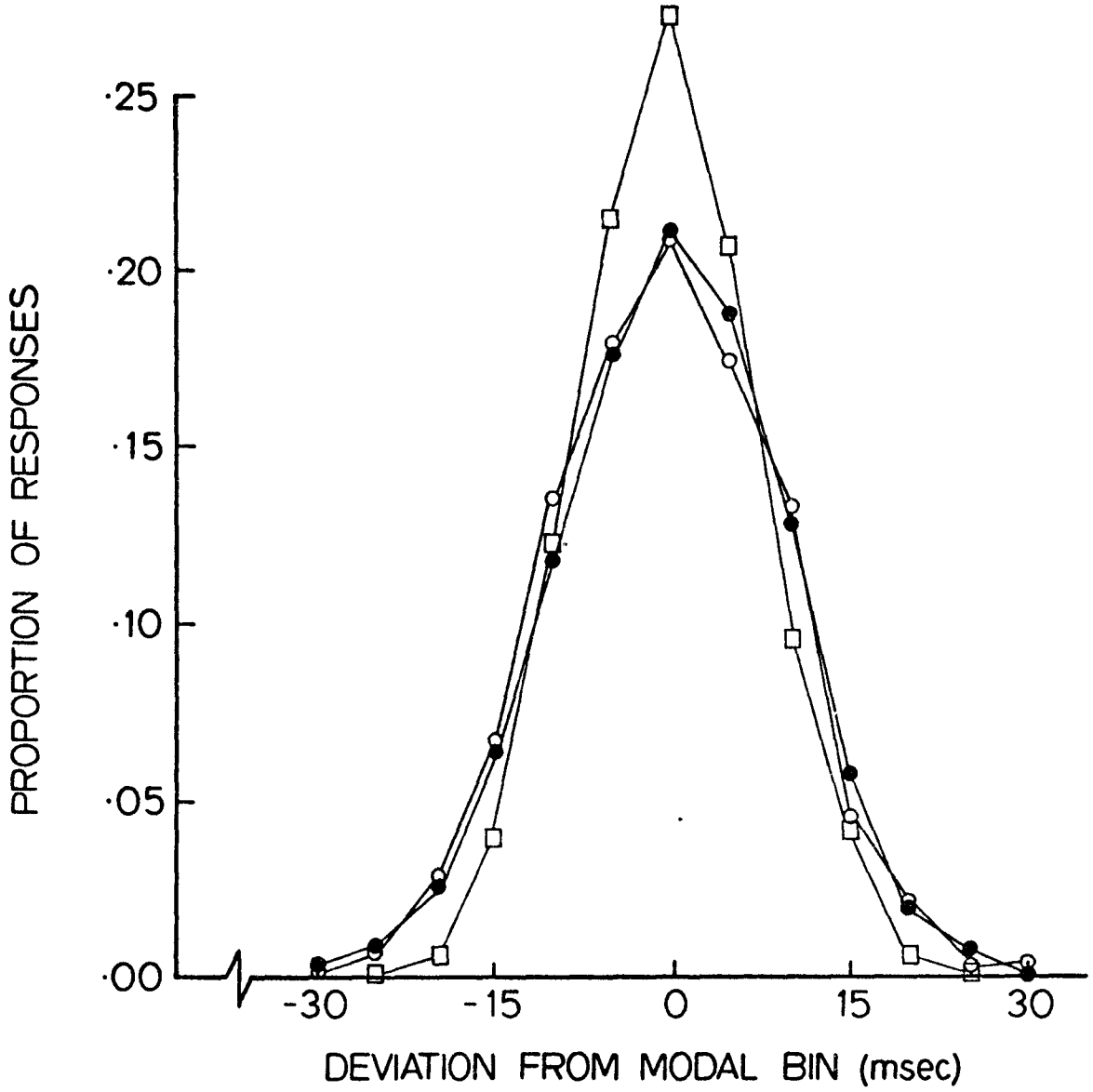
Figure 20 - Decrease in mean within-block variance as a function of practice for two naive subjects with visual interval markers and a P1P2 interval of 360 msec. Filled circles are for subject V.A. and open circles are for subject E.A. Data points are based on only those sessions having both P1 and P2 plus a variable foreperiod. Groups 4 and 5, for subject E.A., were entirely manipulated sessions, hence no data points.

Figure 21 - Autocorrelation analyses (lags 1 through 3), of 1,500 response latencies each, for subject G.H. shortly after transfer from auditory to visual response-stimulus synchronization. Data points show deviation from block mean on trial $(n+i)$, i = 1, 2, or 3, given that the response on trial n was of the latency on the abscissa. Filled circles represent lag 1, open circles represent lag 2, and open squares represent lag 3. Only that data for lag 1 have a significant positive slope (Chi-square = 5.08).

Figure 22 - Relative frequency distributions, of 1,500 response latencies each, for subject G.H. under baseline (filled circles), "P2 only - variable foreperiod" (open circles), and "P2 only - constant foreperiod" (open squares) conditions. P1P2 interval is 460 msec. Means and variances are given in the text.


VARIANCE (msec²)
600
500
400
300
200
100
1
2
3
4
5
6
7
GROUP OF 20 SESSIONS


MEAN ON (N+i) MINUS BLOCK MEAN (msec)
5·0
2·5
0
-2·5
-5·0
430
450
470
490
TIME OF RESPONSE N (msec)


·25
·20
·15
·10
·05
·00
PROPORTION OF RESPONSES
-30
-15
0
15
30
DEVIATION FROM MODAL BIN (msec)

RS.

As a reminder, the data obtained in Experiment 1, following removal of P1, indicated that P1 controlled the timing of RS when present; but, when absent, control was transferred, all but perfectly, to the tactile stimulation produced by R*i*. Since all timing of response latencies and synchronization intervals was made relative to the time-point T*O*, when the subject was using R*i* to time his synchronization response any variability in the foreperiod duration was added into the response latency distribution. This was not true when P1 was present because T*O* corresponded exactly to P1 onset such that if response latencies were controlled by P1 they were independent of foreperiod variability.

When the same manipulations of P1 and foreperiod variability were applied in this experiment with visual markers an entirely different pattern of results was obtained, as shown in Figure 22 for subject G.H. The response latency distribution obtained under the "P2 only" condition is almost identical to the baseline (P1P2 - variable foreperiod) distribution. The means for the two distributions are 461.5 and 460.9 msec with corresponding mean within-block variances of 85.4 and 85.0 $msec^2$ respectively. These data indicate that in the visual modality P1 is ignored by subject G.H. and all timing of response latencies is controlled by R*i*. Further support for

this conclusion comes from the data collected under the "P2 only - constant foreperiod" condition. If the subject is timing from R*i* then the variability of the observed response latency distribution (measured relative to T*O*) should be sensitive to changes in foreperiod variance. It is clear from Figure 22 that such is the case. When the foreperiod variance is reduced from 33.3 to 0.0 msec^2 (constant foreperiod) the performance improves accordingly. Response latency variance drops by 32.3 msec^2 to a level of 52.7 msec^2 with a mean of 461.7 msec. This difference in performance is almost exactly equal to the reduction in foreperiod variance associated with the constant foreperiod condition.

Subject E.A. was also exposed to the same "P2 only" condition and the data are presented in Table 11, along with the data obtained for subject G.H. The pattern of results is the same. With a variable foreperiod, presence or absence of P1 in the stimulus sequence has no effect on performance; however, with a constant foreperiod performance improves. These findings indicate that both subjects are ignoring P1 – the stimulus they are instructed to use for timing their synchronization response – and using R*i* to control the timing instead. It may be noted that the difference in variance between constant and variable foreperiod conditions for subject E.A. is somewhat larger than the 33.3 msec^2 expected. Most of this discrepancy

Table 11

Comparison of Performance with the Baseline (P1P2) Condition vs. Performance with "P2 Only" and the Constant Foreperiod "P2 Only" Conditions.

		Variance		
Condition	Subject	Condition	Baseline	Difference
P2 Only	G.H.	85.4	85.0	0.4
	E.A.	140.1	142.8	-2.7
P2 Only(CFP)	G.H.	52.7	85.0	-32.3
	E.A.	92.7	142.8	-50.1

Note. Variances are mean within-block variances expressed in $msec^2$. Average difference for the first condition was -1.2 $msec^2$ and for the second the difference was -41.2 $msec^2$. CFP means constant foreperiod.

seems to be due to instability in the data. These conditions were run after only relatively few sessions such that the baseline level of variance, based on the data from sessions 41 to 45, is probably an overestimate when used as a comparison with the "P2 only - constant foreperiod" data obtained 20 sessions later.

Later in practice, another baseline level of performance was established for subject E.A. based on data from sessions 131 to 135. Now when the foreperiod was made constant the mean within-block variance level dropped by 43.5 $msec^2$, higher than the value of 33.3 $msec^2$ expected, but consistent with the idea that subject E.A. was still ignoring P1 and using R*i* instead to time his synchronization response. These results are shown in Figure 23. The baseline distribution has a mean of 362.3 msec and mean within-block variance of 110.6 $msec^2$ while the constant foreperiod distribution has a mean of 360.4 msec and variance of 67.1 $msec^2$.

As a final point, it should be noted that both subjects E.A. and G.H thought that when P1 was present that they were using it for timing their synchronization response. The data obtained under the various conditions of stimulus manipulation, however, clearly indicate that, contrary to subjective impressions, both subjects do all their response timing from the tactile stimulation produced by their initiation response, R*i*, whether P1 is present or

not.

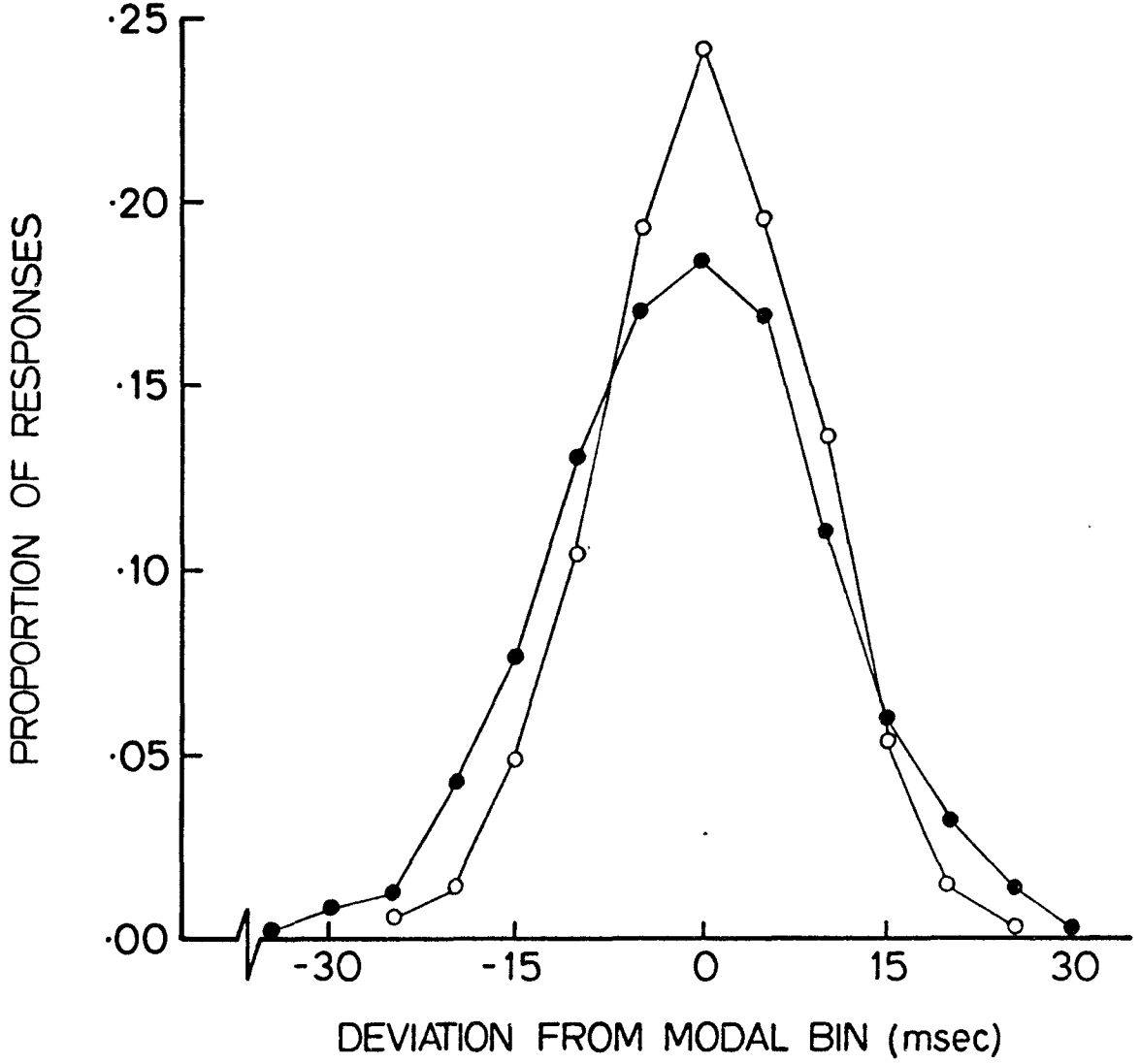
Performance with P1 Only

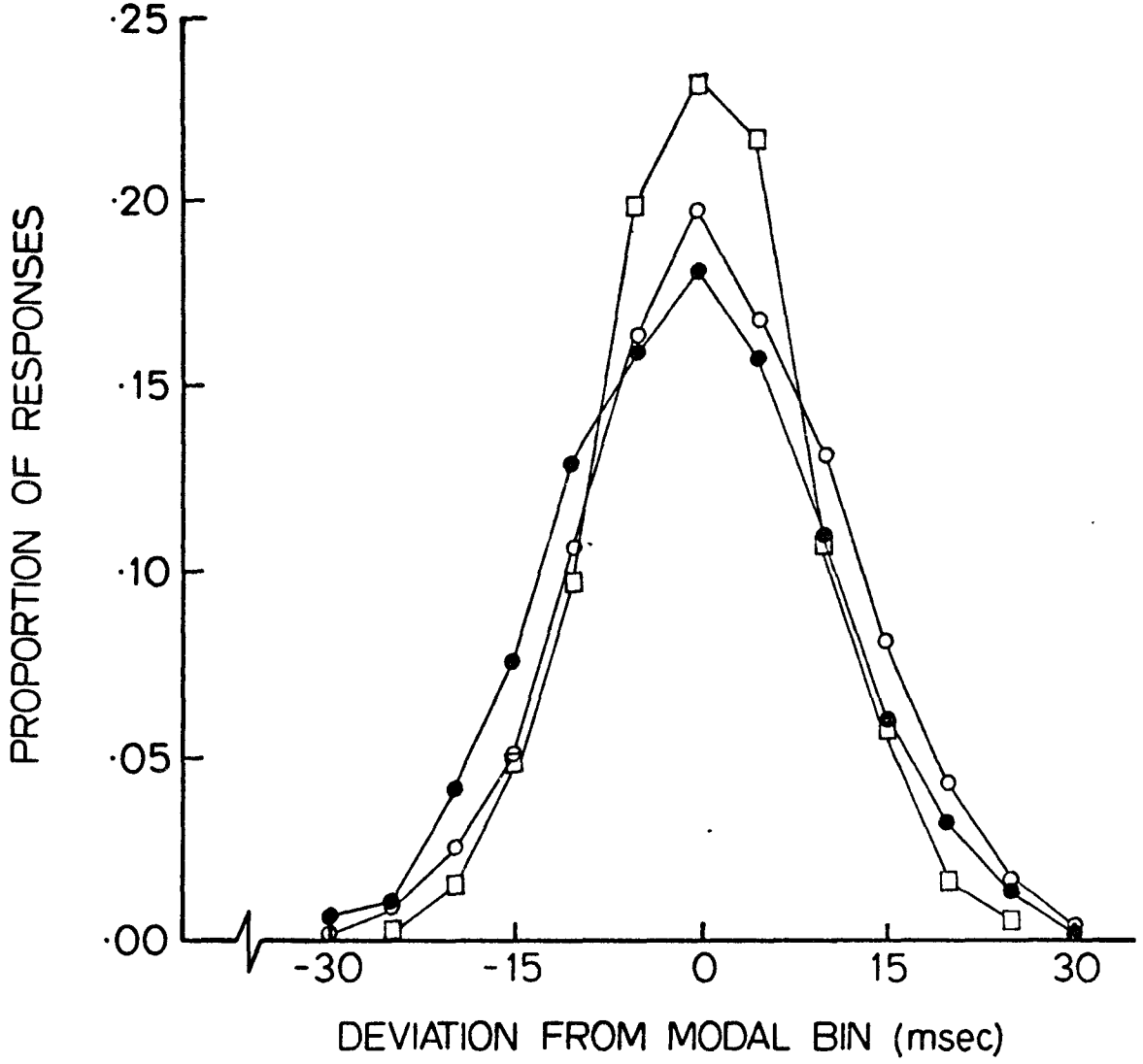
Removal of P2 had little, if any, effect on subject E.A. Again, this is quite a different result when compared with the findings of Experiment 1. Figure 24 shows the results for this condition. The baseline distribution has a mean of 362.3 msec and mean within-block variance of 110.6 $msec^2$. Changing to the "P1 only" condition altered the resulting response latency distribution only marginally. The mean and variance for this distribution are 361.3 msec and 104.6 $msec^2$. Considering the "P1 only" condition distribution was obtained 30 practice sessions following the baseline sessions, it is not surprising that the variance with only P1 is somewhat smaller than the baseline variance.

As with all the other stimulus manipulations discussed so far in this experiment, making the foreperiod duration constant has the effect of improving performance. In this case, subject E.A. exhibited a decrease in variance of 34.3 $msec^2$ which is almost identical to the amount of experimentally induced variance associated with having a variable foreperiod and timing the synchronization response from R*i*. To add to the earlier conclusions regarding subjects' strategies, these data indicate that the subjects seem to be ignoring, not just P1, but both of the interval markers when they are visually presented.

Figure 23 - Relative frequency distributions, of 1,500 response latencies each, for subject E.A. under baseline (filled circles) and "P1P2 - constant foreperiod" (open circles) conditions. P1P2 interval is 360 msec. Mean and variances are given in the text.

Figure 24 - Relative frequency distributions, of 1,500 response latencies each, for subject E.A. under baseline (filled circles), "P1 only - variable foreperiod" (open circles), and "P1 only - constant foreperiod" (open squares) conditions. P1P2 interval is 360 msec. Means and variances are given in the text.


·25
·20
·15
·10
·05
·00
PROPORTION OF RESPONSES
-30
-15
0
15
30
DEVIATION FROM MODAL BIN (msec)


·25
·20
·15
·10
·05
·00
PROPORTION OF RESPONSES
-30
-15
0
15
30
DEVIATION FROM MODAL BIN (msec)

The "P1 only - constant foreperiod" distribution shown in Figure 24 has a mean of 361.5 msec and mean within-block variance of 70.3 msec^2. All 1,500 responses fall within a 50 msec time window and the shape is very symmetrical and sharp-peaked. This general form is the same as that observed in the low variance distributions of Experiment 1.

As in Experiment 1, one manipulation involved omission of both P1 and P2 from the stimulus sequence. For subject E.A. this condition followed the "P1 only -variable foreperiod" condition so the only change was to remove P1 as well. Performance remained virtually the same. Error of synchrony increased slightly from 1.3 to 2.9 msec while mean within-block variance decreased trivially from 104.6 to 102.7 msec^2.

Additional Stimulus Manipulations and Low Variances

Subject G.H. was also exposed to several sessions of response-stimulus synchronization without P1 and P2. In this case the sessions followed the "P2 only - constant foreperiod" condition. Maintaining a constant foreperiod, the only change that was made was the additional deletion of P2 from the stimulus sequence. Consistent with the data for subject E.A., this had no significant effect on performance. Mean within-block variance increased by only 0.1 msec^2 to 52.8 msec^2.

All the results considered in this experiment point to the conclusion that well-practiced subjects do not make use of the visual markers for response timing in the present synchronization paradigm. Instead, all response timing is controlled by the tactile stimulation produced by the subject's initiation response. This is true even with a variable foreperiod, in which case using **R***i* as a cue for anticipating the onset of **P**2 involves some degree of uncertainty. In light of this finding, the appropriate condition for looking for low variance should be a constant foreperiod condition.

In an attempt to get some measure of the low variance for subject E.A., five sessions were run with the full stimulus complement and a constant foreperiod. These data were obtained from sessions 181-185. The mean was 359.2 msec (mean error of synchrony of -0.8 msec) with a mean within-block variance of 61.4 $msec^2$. Overall variance was 66.9 $msec^2$. This discrepancy between the two variance measures is larger than typically observed in Experiment 1 indicating less stability in the data and suggesting the existence of sequential dependencies between successive response latencies. This notion was tested using the Chi-square statistic described in Experiment 1. The results of this analysis showed a significant lag 1 autocorrelation (Chi-square = 15.73 on 1 d.f.). Sequential dependencies beyond lag 1 were all nonsignificant.

Although the synchronization responding of subject E.A. appears to be less consistent when compared with the data of Experiment 1, the absolute level of performance seems to be quite consistent. The value of low variance for subject E.A. of 61.4 msec^2 fits in quite well with the low variances of 54.1 msec^2 for subject G.T. and 71.2 msec^2 for subject C.H. obtained in Experiment 1 when these two subjects were at roughly equivalent levels of practice. All other aspects of the response latency distributions are also similar. They are all highly symmetrical, sharp-peaked, and totally contained within a 50 msec time window. Despite all the data indicating that both of the subjects in this experiment were ignoring the visual markers and using R_i to time their synchronization resonses, there is some indication that this strategy may not have been used by subject E.A. during the earlier stages of acquisition. The "no P1 or P2" data described earlier were obtained after 175 sessions of practice and no difference in performance as a result of omitting P1 and P2 occurred. However, a different pattern of results was obtained earlier on in practice. The same manipulation, administered after 65 sessions, degraded performance from a level of mean within-block variance of 92.7 msec^2 to 103.6 msec^2. Moreover, continued practice on this condition was characterized by further increases in variance (decrements in performance) to a level of 140.1 msec^2 by sessions 101 to 105. This pattern of results is

not unlike those obtained in Experiment 1 during any of the conditions where P2 was absent. This suggests that the light flashes may have assumed some degree of a perceptual feedback role for subject E.A. during the earlier stages of skill acquisition.

In fact, this finding may account for the differences in the acquisition curves for subjects E.A. and V.A. It is possible that V.A. maintained the strategy of using P1 for the timing of her synchronization response and P2 for gaining perceptual feedback while E.A. changed strategies to rely more heavily on R*i* for the purpose of timing his synchronization response. If the former strategy happens to involve more variability than the latter, even though the latter involves the inclusion of the foreperiod variability in the response latency measurements, then the discrepancy in the data for the two naive subjects could be reconciled by attributing the different patterns of acquisition to divergent strategies for the timing of their synchronization responses. In the case of V.A., the increased variability, involved with her strategy, could have made improvements in performance so difficult to make that her motivation fell to a level that prevented further decreases in response latency variance from occurring.

As a final point, there was some worry that subjects might be able to generate an auditory stimulus while making either their initiation or synchronization response by

flicking their finger against or away from the response button in a particular fashion. The presence of an auditory component into the present trial configuration could alter some of the foregoing reasoning about which stimulus was responsible for controlling the timing of the synchronization response. To test this idea, subject G.H. experienced several sessions of practice with neither P1 nor P2, a constant foreperiod, and the inclusion of an ambient "white noise" set at a loudness of 80 dB re .0002 dynes/cm^2. The noise was included to mask any auditory stimulation that might be produced by the subject's responses. Performance deteriorated slightly when the noise was first introduced, but this seemed to be due more to the novelty of the stimulation than to the masking of a vital auditory input generated during responding. In short order, less than 10 sessions, performance was essentially the same as before the noise was introduced. Before and after mean within-block variance levels were 52.8 and 54.8 msec^2, respectively. Thus, even if auditory cues sometimes were available to be used for synchronization responding, it is clear that they were certainly not necessary for performing the skill with the high level of precision observed.

C. Discussion

Initial acquisition of the response-stimulus synchronization skill, using visual P1P2 interval markers, followed a similar pattern to that obtained with auditory stimuli. The absolute levels of response latency variance, however, were consistently higher with the visual markers throughout practice. The regression analysis obtained from fitting a power function to the acquisition data indicated that slope, or rate of acquisition, were comparable across modalities. It was only the intercept that differed. In addition, the data were less stable, both in terms of mean error of synchrony and differences between mean within-block and overall variance measures. Significant autocorrelations were also often found between successive response latencies.

Poorer performance with visual synchronization pulses was not only limited to the naive subjects. The well-practiced subject showed decrements in performance on initial transfer from the auditory situation and never was able to obtain variance levels as low as those obtained in Experiment 1.

Why do these differences exist? All the available evidence, outlined in the results, indicates that the most parsimonious interpretation of the data is that subjects adopt the strategy of ignoring P1 and P2, when they are presented visually, in order to minimize their variance levels. It appears that switching the timing control from

P1 to the tactile stimulation produced by the initiation response allows synchronization responses to be made with less variability. This is true even when R*i* is not perfectly time-locked to the time of P2 onset – during variable foreperiod conditions.

It should be pointed out that the subjects were unaware of which stimulus they were using to control their synchronization timing. They were instructed to use P1 when present and the subjective impression was that they were attending to P1 as the salient stimulus in this task. The data, however, show otherwise. Remember also that subjects are unaware of the foreperiod duration. Therefore, any information obtained from introspection in this situation should be interpreted with caution.

Initially there were large drops in variance for the naive subjects but not for the transfer subject. Indeed, transfer seemed very good. It must be remembered, however, that the visual stimuli, P1 and P2, were being ignored which means that the transfer condition really became the same as the "no P1 or P2 - variable foreperiod" condition of Experiment 1. From that experiment, the estimate of the increase in response latency variance incurred by this condition was about 60 $msec^2$. If this value is added to the 34.3 $msec^2$ minimum baseline variance obtained prior to the transfer, the total is within the range of response latency variances obtained immediately following transfer. The

additional 60 msec² variance was attributed partly to the 33.3 msec² variance associated with the foreperiod plus variability introduced by removing the source of immediate perceptual feedback, P2. In Experiment 1, P2 seemed to play a very important role in this respect throughout acquisition whereas in this experiment P2 played no apparent role in sharpening synchronization performance, consistent with the idea that P2 was being ignored.

What is being learned during acquisition of the response-stimulus synchronization skill? It would appear that the rapid improvement in performance early in practice is largely a function of minimizing efferent delay variance and learning how to interpret the feedback provided. Learning to process the interval markers more efficiently can probably be ruled out because subjects are able to switch easily from P1 to R*i* (different modalities) in Experiment 1. Moreover, in this experiment, although two of the subjects adopted the strategy of ignoring the visual P1P2 markers, the rate of initial acquisition for subject V.A., who appeared to be using the visual stimuli for timing control, was the same as that obtained with the auditory stimuli. In other words, learning of the skill seems to be independent of the stimulus modality used for control of synchronization timing, at least for the three modalities involved in these experiments.

Learning how to time out the appropriate internal delay can also probably be ruled out as a factor contributing to the steepness of the initial learning curve. It is clear from both experiments that subjects are able to change the length of interval to be timed with ease. There is no indication of a learning process. Such changes in the duration of the interval to be timed out are required when switching between the condition in which P1 controls timing and that in which R*i* is the the controlling stimulus and also when foreperiod variabilities are being manipulated and P1 is not available.

Why would a subject adopt a strategy of timing his synchronization response from a time-point event which has temporal variability with respect to the synchronization pulse when a perfectly time-locked event (P1 onset) is available? The only rational explanation is that the afferent latencies have different variabilities such that the internal representations of these two events have reversed variability characteristics; that is, the time-locked visual event, P1 onset, is registered internally with more variability than is the tactile stimulation produced by R*i*, in spite of the foreperiod variability associated with the latter stimulus. According to Zacks (1973), estimates of afferent latency variance for visual flashes range from 56.3 to 182.3 $msec^2$ which is substantially larger than the 33.3 $msec^2$ foreperiod variance

associated with the use of **R***i* for timing control. It may be recalled that the afferent latency associated with the tactile stimulation produced by **R***i* was established as being negligible in Experiment 1. In light of this evidence, the strategy adopted by subjects G.H. and E.A. of using **R***i* rather than **P**1 onset for the timing of their synchronization responses seems quite reasonable. As mentioned before, it appears that subject V.A. failed to make this strategic change because her latency variance remained relatively high compared to the variance of the other naive subject and the difference in performance is well within the range predicted by Zacks (1973) work on measuring visual afferent latencies.

The low variance in this experiment for subject G.H. was 52.7 $msec^2$ which is significantly higher than the level of 34.3 $msec^2$ obtained in Experiment 1. It should be recalled that low variances in this experiment were based on the constant foreperiod condition because timing control was attributed to **R***i*, which preceded the foreperiod. Consequently, with the visual markers being ignored, the "low variance" condition in this experiment becomes analogous to the "no **P**1 or **P**2 - constant foreperiod" condition in Experiment 1. In the latter condition, response latency variances were typically 30 to 40 $msec^2$ greater than the baseline variance at the same level of practice. This difference was attributed to the lack of immediate perceptual feedback due to the absence of **P**2.

Although this spread in performance is larger than the 18.4 $msec^2$ difference obtained when the low variance for this study is compared with the lowest baseline variance obtained in Experiment 1, the discrepancy can probably be accounted for by the fairly substantial difference in amount of practice associated with these two measures. With more practice in the absence of immediate perceptual feedback, the more efficient one should become at using the delayed feedback information.

The synchronization pulse, P2, is present in most of the conditions in this experiment, yet it is clear that to obtain the lowest variance performance the best strategy is to ignore this information. Presumably this is because of the uncertainty associated with this type of information when it is registered internally. In other words, P2 onset is ignored for the same reason P1 onset is ignored – the internal representation of this information is too variable to be used efficiently in this task. The next experiment seeks to establish some measure of the extent of this variability.

IV. Experiment 3 - Visual-Auditory Differences in Simple Reaction Time

As mentioned in the last chapter, several studies point to the difference in the variability of afferent latencies of visual versus auditory stimuli. In these studies the variability measures were estimated from discrimination data (Divenyi, 1976; Lhamon & Goldstone, 1974; and Zacks, 1973) or from neurophysiological data obtained from responses of single, retinal ganglion cells in the cat's retina (Levick & Zacks, 1970). The results of these studies indicate that variability in the afferent latency of detection responses to moderately loud auditory stimuli is basically negligible (variance less than 0.5 $msec^2$), whereas for equally salient visual stimuli afferent latency variances are quite substantial, ranging from 56 to 182 $msec^2$.

In the discussion of Experiment 2, it was argued that these differences in the variability with which visual and auditory signals can be represented internally accounted for the differential results that were obtained. Subject V.A., who appeared to be actually using the visual information from the P1P2 flashes for timing her synchronization responses, exhibited response latency variances which were inflated between 100 and 200 $msec^2$ above the levels obtained with auditory interval markers.

The magnitude of this difference is in good agreement with the estimates derived from the work of Divenyi (1976) and Zacks (1973). For the other two subjects in Experiment 2, it was shown that they were ignoring the visual information, presumably because of the relatively large variability associated with its internal representation, and relying on the tactile information inherent in the task – despite the fact that objectively the tactile information provided a less consistent time-point event for use in synchronization timing. In both cases, the explanation of the differences in performance was purely speculative. In order to provide a direct measure of any differences in the variability with which these auditory and visual stimuli could be represented internally a simple reaction time task was devised, using basically the same stimuli as were used for synchronization.

The literature contains a wealth of research on simple reaction time (RT); but the studies that have looked at reaction time as a function of the sense modality stimulated have been largely concerned with differences in the mean or median response latency (Teichner, 1954). These studies have repeatedly found that RT for audition is 30 to 50 msec faster than for vision, with the mean visual reaction time being near 200 msec. In contrast, however, the author knows of no comparable data which make a clear and direct comparison of response latency variance across stimulus modalities, especially with well-practiced

subjects. Fortunately, the current experimental situation, which involves having a well-trained subject that is experienced with both visual and auditory stimuli, provides a good opportunity to obtain the needed empirical data.

A. Method

Subjects

Only subject G.H. served in this experiment as he was the only one who had had extensive exposure to both the visual and auditory stimuli used for synchronization training in Experiments 1 and 2. Subject G.H. had also had several sessions of RT training prior to his synchronization training.

Procedure

A simple RT task was created using the same response and some of the same stimuli as were used in Experiments 1 and 2 for response-stimulus synchronization. Changes involved the elimination of the delayed feedback signal and P2 from the trial sequence. Also, the foreperiod interval between the subject's initiation response, R*i*, and P1 onset was increased to 1.2 seconds. The foreperiod remained constant but was made quite long to discourage anticipatory responding. The objective for the subject was to respond, by withdrawing his index finger from the touch-sensitive button, as quickly and consistently as possible following

the onset of P1. Thus P1 served as the action stimulus for the reaction time response. When P1 was a tone pulse, the ready signal was a 500 msec visual stimulus and vice versa when P1 was a light. The stimulus characteristics of the P1's were the same as those outlined in Experiments 1 and 2. As before, subjects were instructed to fixate the L.E.D. in order to ensure consistency in foveal stimulation. Trials were still self-paced, with the subject able to respond at any time following offset of the ready signal and, with the increased foreperiod duration compensating for the lack of a P1P2 interval and feedback delay, the average intertrial interval was comparable to that observed during synchronization.

A total of 10 reaction time sessions were run with one session per day, excluding weekends. A session consisted of a set of 200 reaction time trials for each of the two types of stimuli. These 200 trials were subdivided into 4 blocks of 50 trials each. The first block of trials was considered as warm-up and discarded from analysis leaving 150 trials for analysis, per session, for each stimulus condition (auditory-visual). The order in which the visual and auditory reaction time trials were run was alternated each day and the subject was encouraged to minimize both his mean reaction time and latency variance.

B. Results

The results support the hypothesis that reaction time latencies to visual stimuli are more variable than response latencies to auditory stimuli.

Differences in Means and Variances

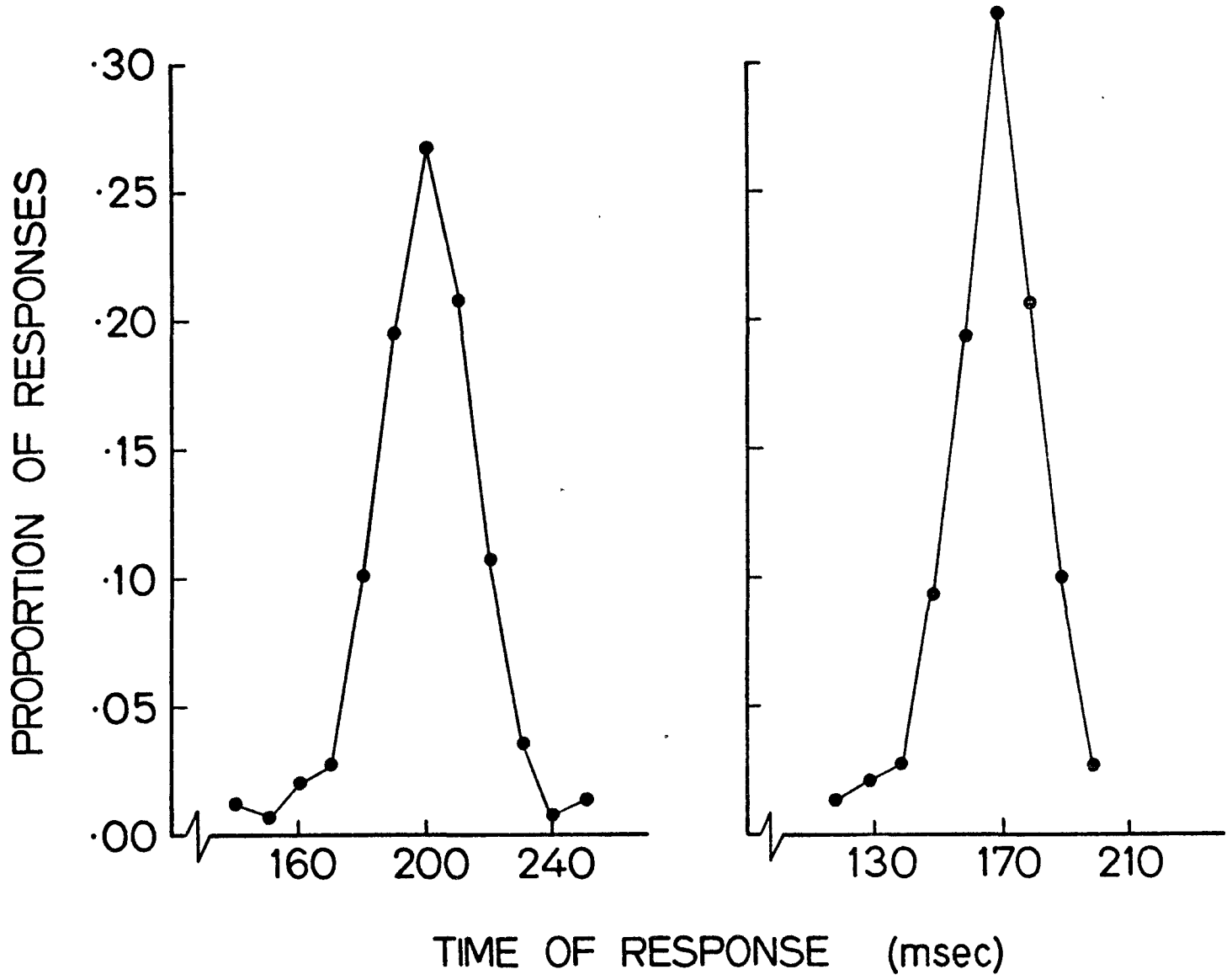
Figure 25 displays relative frequency distributions obtained on the last session of RT practice. Each distribution is based on 150 trials and plotted using 10 msec bins. The left panel shows the response latency distribution for the visual action stimulus. The mean time of response is 199.0 msec with a mean within-block variance of 329.1 $msec^2$ and overall variance of 330.6 $msec^2$. The right panel shows the analogous distribution obtained with the auditory stimulus. The mean is 166.2 msec which is almost 33 msec faster than with the visual stimulus and quite consistent with earlier findings (e.g., Teichner, 1954). The interesting result, however, is the difference in variance measures. Mean within-block variance with the auditory reaction stimulus is 225.4 $msec^2$ with an overall variance of 229.7 $msec^2$. These values are over 100 $msec^2$ less than the comparable variance measures obtained with a visual reaction stimulus. Although the data plotted is only for the last session, the pattern of results was the same for all ten sessions. The mean RT to a flash of light was always greater than the mean RT to an auditory pulse. The

mean difference, across all sessions, was 32.1 msec. Similarly, response latency variances obtained with the visual reaction stimulus were consistently higher than for auditory RT with the mean difference being 176.4 $msec^2$. These differences ranged from 66.9 to 332.0 $msec^2$. Thus, it is clear that significant and large differences are evident in this study with respect to variability of simple RT using auditory versus visual stimuli.

Despite the auditory-visual differences just noted, the response latency distributions shown in Figure 25 do share one common characteristic; namely, they have similar shapes. They are both sharp-peaked and quite symmetrical. Each distribution is based on 150 responses and there are no responses outside the distribution. These findings are consistent with the notion that the only difference between auditory and visual RT is that the latter involves some uncertainty as to the moment of reaction stimulus onset as a result of temporal variability in the central representation of this external, time-point event. Such uncertainty would act to spread the distribution out along the time axis. Finally, the fact that the resulting distribution remains symmetrical suggests that the underlying distribution of the internal representations of action stimuli onsets is symmetrical also.

The fact that these reaction time distributions are symmetrical is somewhat atypical. Generally, simple RT

Figure 25 - Relative frequency distributions, of 150 reaction time latencies each, for subject G.H. The right panel shows the distribution obtained with an auditory RT signal and the left panel shows the analogous distribution obtained with a visual RT stimulus. Bin size is 10 msec. Means and variances are given in the text.


·30
·25
·20
·15
·10
·05
·00
PROPORTION OF RESPONSES
160
200
240
130
170
210
TIME OF RESPONSE (msec)

distributions are skewed right with a long tail of relatively long reaction times (Teichner, 1954). In certain situations, however, symmetrical RT distributions are observed; namely, when the subject is motivated via payoff bands (e.g., Saslow, 1974), or similar means, to respond as consistently as possible. Consistency of responding was of prime concern in this experiment, therefore, obtaining symmetrical RT distributions should not be considered unusual.

Sequential Dependencies

Is it possible that the larger variability associated with visual RT could be simply due to some degree of dependency between response latencies operating to inflate the performance measures? To test for this possibility a simple analysis of sequential dependencies was conducted using the same procedure outlined in Experiment 1. The results of this analysis revealed no significant indication of a dependency between successive response latencies (lag 1), or for any other any lag.

C. Discussion

The results of this experiment provide an additional measure of the difference between visual and auditory temporal information processing which is in good agreement with previous estimates in the literature (cf. Divenyi,

1976 versus Zacks, 1973). These differences are not small either, averaging over 100 msec^2; therefore, input modality must be carefully considered in any theorizing about the temporal characteristics of human information processing.

In Experiment 2, it may be recalled, the data indicated that subjects E.A. and G.H. had developed a strategy of ignoring the P1P2 light flashes and, instead, relied on the tactile information associated with their initiation and synchronization responses for controlling their timing. Such a strategy was observed even when the foreperiod duration between the subject's initiation response and the onset of P1 (start of synchronization timing) was variable. This resulted in an experimentally induced variance of 33.3 msec^2 being added into the response latency variance measures. It was argued, in that chapter, that adoption of such a strategy could only improve performance if, in fact, the "penalty" variance incurred was less than the variability associated with the use of the visual markers P1 and P2 for synchronization timing. The results of the present experiment seem to confirm the validity of this argument. On the average the uncertainty associated with using a visual marker for timing a synchronization response appears to be much greater than that associated with timing the same response from the subject's initiation response (tactile input) despite the presence of a slightly variable foreperiod.

In Experiment 2, it was also argued that the relatively poor performance exhibited by subject V.A. (see Figure 20) could be attributed to her continuing to use the instructed strategy; that is, timing her synchronization response from the onset of the first light flash, P1. It is interesting to note that the variance level where her performance became asymptotic (250 to 300 $msec^2$) is very comparable with the lowest variance level (329 $msec^2$) obtained in the present RT study using visual reaction stimuli. The slightly better performance observed on the synchronization trials compared to the simple RT trials could be due to the presence of delayed auditory feedback in the former paradigm which could be used to gain more consistency in responding. The fact that the difference in performance is so small, however, suggests that this form of feedback, when made available in the synchronization procedure of Experiment 2, contributes little to benefit synchronization with visual markers. The difference could also be due simply to differences in the amount of practice.

The question arises: If response latency variances for visual RT and visual synchronization are quite similar, why is the same not true for the auditory modality? It appears that the answer may lie in terms of feedback utilization. In Experiment 1, the results indicated that P2 provided immediate perceptual feedback that was crucial for maintaining low-variance, synchronization responding. When

P2 was removed, variance levels rose dramatically and prolonged practice was required in order to reduce response latency variance levels to within even twice the variance values obtained with P2 present. The improvements in performance were attributed to relying more heavily on the delayed auditory feedback for maintaining consistent responding. This source of feedback was never removed in Experiment 1; thus, it is not clear that the variance levels, in the absence of all types of feedback, would be similar to those obtained with auditory RT. All indications are, though, that the variance levels would increase even further beyond those levels obtained without P2 present.

The foregoing discussion implies that the relatively poor synchronization performance exhibited by subject V.A. in Experiment 2, with visual markers and delayed auditory feedback, resulted from her not being able to effectively integrate the auditory feedback information with the visual marker information in order to provide useful and accurate evaluative information. This relative inability to extract useful information from the delayed auditory feedback signal, for purposes of refining the timing of the subject's synchronization response, seems quite reasonable given the uncertainty with which the onsets of P1 and P2 seem to be registered in the central nervous system.

It should be pointed out that this experiment is rather weak by itself. However, it was not designed to be a

thorough investigation of differences in auditory and visual simple reaction time. If it had been, obviously much more practice and data with regard to the energy and presentation conditions of the action stimuli would be desirable. Rather, this experiment was simply designed to further our understanding of the differences observed in response-stimulus synchronization performance between Experiments 1 and 2. As a result, only the two types of stimuli (one auditory and one visual) used for interval markers in these earlier experiments were used. Also, the warning signal was always presented in the opposite modality from that of the action stimulus, consistent with the arrangement used during synchronization. The use of a fixed foreperiod with no catch trials might be criticized, but there were no indications of anticipatory responding and the subject was quite experienced at the psychophysical task.

In spite of any weaknesses, the available evidence suggests that the presence and type of feedback, plus the modality in which it is presented, are all of great importance in understanding synchronization performance, how it differs across modalities, and how it differs from reaction time performance. In order to gain more quantitative data relevant to understanding the role feedback plays in the temporal organization of a response, the next experiment will look at what happens under various stimulus manipulations when the delayed auditory feedback

pulse is omitted from the trial sequence.

V. Experiment 4 - Role of Feedback in Response-Stimulus Synchronization

Several studies of temporal perception have shown feedback (FB) to be a very important factor for improving the accuracy of a temporal response. In a duration discrimination task, Jamieson and Petrusic (1976) found that with FB the time-order error, usually observed in this type of study, disappeared producing a very close correspondence with objective, real time.

In a key tapping task (at a specified rate), Chase, Rapin, Gilden, and Guilfoyle (1961) found performance to be directly related to the amount of FB provided. They were able to interfere with auditory FB by varying the level of a masking noise in the subject's headphones. Proprioceptive and tactile FB were also interfered with through the use of vibration and an injection of Xylocaine into the index finger used for tapping.

In tasks more similar to response-stimulus synchronization, the presence of feedback is typically associated with increased accuracy of response timing, in which variability in the time of response is independent of the mean latency over a substantial range of short time intervals from about 160 to over 550 msec (Bartlett & Bartlett, 1959; Naatanen, Muranen, & Merisalo, 1974; and Saslow, 1974). This general finding is consistent with a

deterministic type of process. However, it should be pointed out that none of these researchers ever interpreted their data in these terms. It was Kristofferson (1976) who first suggested that this type of data did, in fact, reflect the operation of a deterministic, internal timing mechanism. With feedback omitted, in these types of paradigms, the results are quite different. The data generated are typically characterized by a relationship between the standard deviation of response latencies and mean latency which is linear with a positive slope (e.g., Snodgrass, 1969).

The question of concern is: What is the exact role and nature of feedback in response-stimulus synchronization and is FB always needed in order to maintain timekeeping integrity? Theories and data from the motor skills literature suggest that feedback (often called knowledge of results) is necessary for learning because subjects must anticipate or predict future stimulus events and can only attain better performance if allowed to compare the actual outcome of their behaviors with the desired outcome (Adams, 1971; Schmidt, 1968, 1975). Viewed in this way, the amount, interpretability, and fidelity of the feedback all become very important. A subject without reliable FB information has no basis for modifications to his response strategy.

Although feedback is crucial during acquisition of a skill, several studies indicate that after a relatively

large amount of practice FB can be withdrawn without any deterioration of performance (Adams, 1971; Schmidt & White, 1972). Removal of FB at earlier points in practice generally results in performance losses which are inversely related to the amount of practice (Wallace, DeOreo, & Roberts, 1976).

The purpose of this experiment is to investigate what role the delayed auditory feedback played in the response-stimulus synchronization paradigm used in Experiments 1 and 2. It is predicted that the absence of the delayed auditory feedback signal will retard progress during acquisition, but will not affect performance following a prolonged training period during which the FB was present. In addition, it is expected that strong sequential dependencies will be evident in those conditions that omit all forms of feedback information. This expectation is based on the results obtained by Snodgrass (1969), using a reaction time paradigm involving payoff bands, which showed stronger lag one autocorrelations and sequential effects for trials with no feedback.

Finally, considering the previous results which indicated that P2 served an important FB role in Experiment 1 (auditory stimuli) but not in Experiment 2 (visual stimuli), it is predicted that removal of the delayed feedback, even after prolonged practice, will be more detrimental to synchronization performance with visual

markers than with auditory markers.

A. Method

Subjects

Three males served as subjects in this part of the research. Two subjects, E.A. and G.H., were both well-practiced, having participated in earlier experiments. Subject G.H., the author, had experience with both visual and auditory synchronization while E.A. had only experienced synchronization with visual, interval markers. The third subject, J.V., was naive with respect to synchronization experiments.

Procedure

All aspects of the procedure and stimulus manipulations were identical to those in Experiments 1 and 2 for auditory and visual synchronization, with the continued use of subject-paced trials. The only difference was the addition of one other manipulation which involved deletion of the delayed, auditory feedback during some sessions.

Subject G.H. was given 20 sessions of practice with a stimulus sequence similar to that used in the baseline condition of Experiment 1 (both P1 and P2 were present and auditory), but with no delayed feedback signal. These sessions were then followed by 15 sessions of practice in which all the stimuli were deleted from the trial sequence

except for the visual ready signal. In spite of the lack of feedback during these sessions the subject was always informed of his performance, in terms of block means and variances, at the end of each session.

With neither P1 nor P2, and no delayed feedback information, the task basically became one of straight temporal production of the interval of time which presumably had some representation in memory after the thousands of trials of experience preceding this condition. The foreperiod, or time between the subject's initiation response (*Ri*) and the start of response timing (To), was made constant for this condition because with P1 absent the subject had to resort to the tactile concomitants of *Ri* for timing his response delay (see Experiment 1). There was no stimulus with which to synchronize, and no feedback of any kind regarding the accuracy of the delayed response; therefore, the system was basically free-running, trying to repeat, as accurately as possible, a stimulus-response chain in a totally "open-loop" fashion.

Subject E.A., the veteran subject of synchronization with visual markers, experienced the analogous conditions to those outlined for subject G.H. In this case, P1 and P2 were light flashes, and the ready signal was aurally presented. The foreperiod was constant throughout because it was evident from Experiment 2 that subject E.A. did all timing of his response delays relative to *Ri* whether P1 was

present or not. Ten sessions of practice were provided with P1 and P2 (no delayed feedback) followed by 15 sessions of free-responding.

The naive subject, J.V., experienced 30 sessions of standard auditory synchronization training, except without the delayed auditory feedback. The interval markers, P1 and P2, were present on all trials and the foreperiod was always variable.

Subject J.V. received all his synchronization training with a P1P2 interval of 310 msec. The veteran subjects continued their no feedback synchronization at the same interval on which they had acquired the skill; namely, 460 msec for subject G.M. and 360 msec for subject E.A.

B. Results

Initial Acquisition

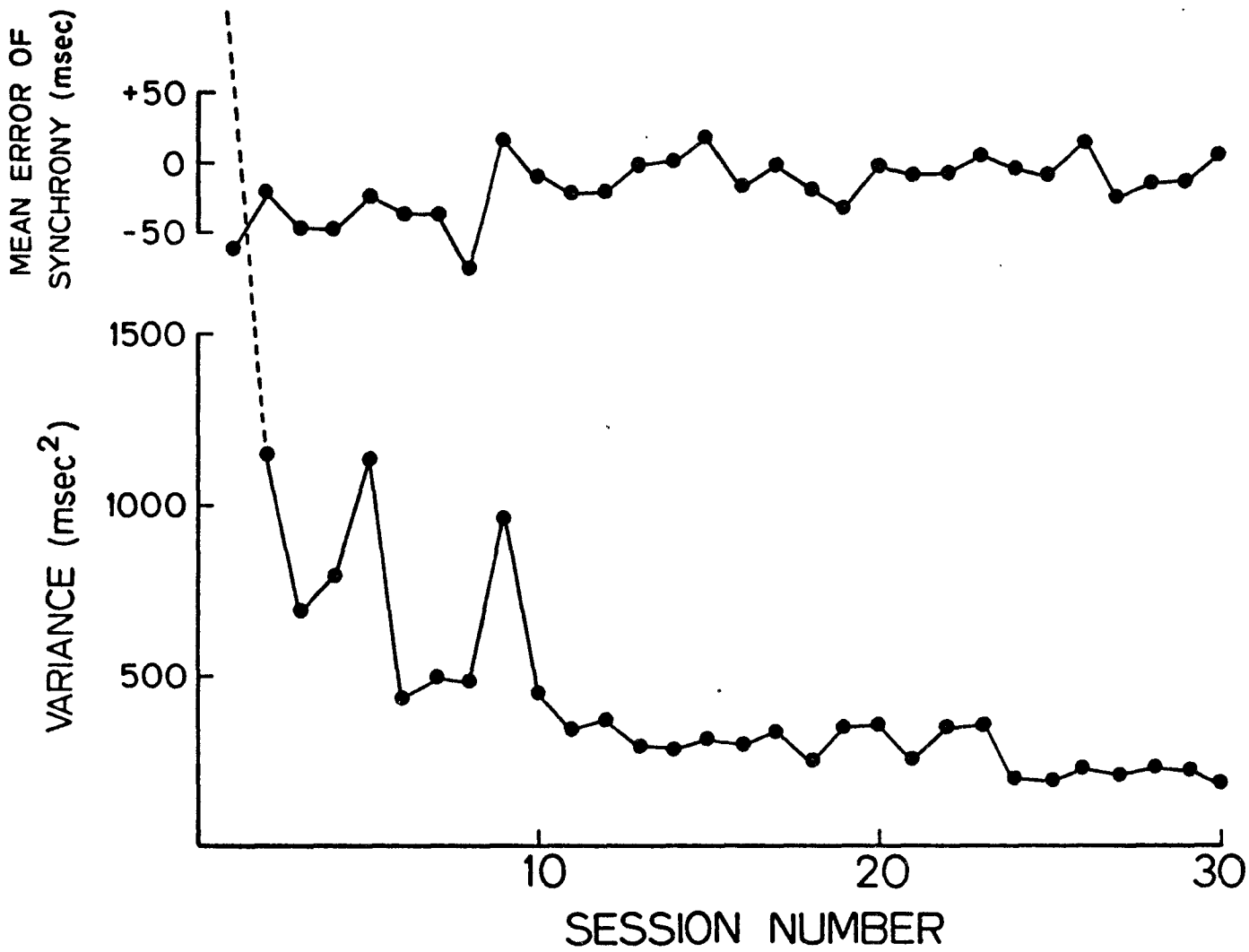
Lack of delayed feedback did have a detrimental effect on the synchronization performance of the naive subject, however, the effect was mostly associated with the error of synchrony and not as much with the variability of response latencies. Acquisition data for subject J.V are shown in Figure 6. If one examines the lower panel, showing decreasing error scores as a function of practice, the effect of not having the additional feedback is not readily apparent. The general shape of the acquisition curve is similar to those seen in Experiment 1, characterized by a rapid decline

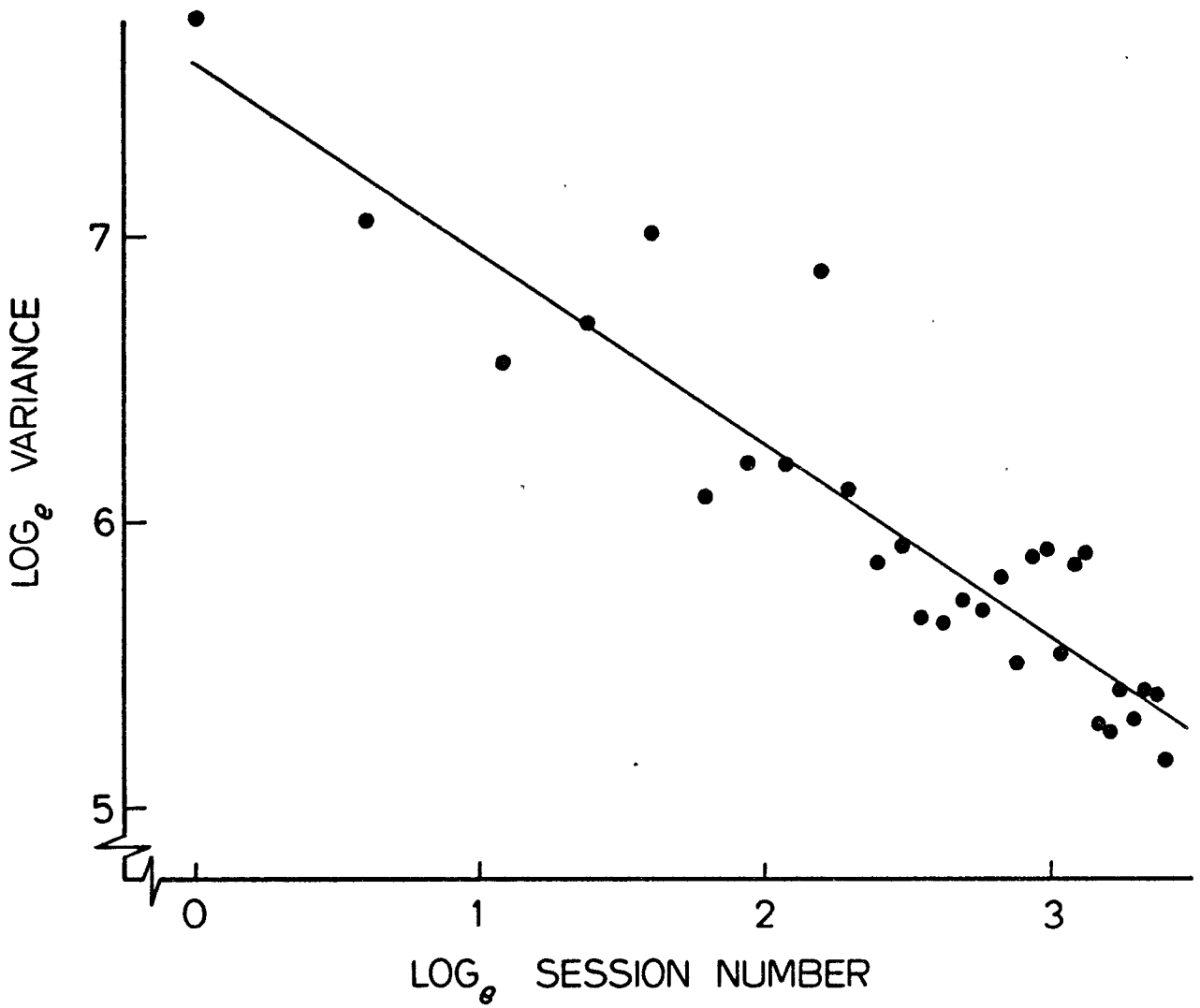
in variance over the first 8 to 10 sessions, followed by much slower improvement. More similarities emerge, with respect to this part of acquisition, when the data points are plotted on log-log coordinates. As in Experiments 1 and 2, the resulting scatterplot, shown in Figure 27, can be well represented with a power function (linear plot in this coordinate system), which accounts for more than 84 percent of the variance. Slope and intercept values are -0.670 and 7.613, respectively, which agree fairly well with the values obtained from subjects under basically the same conditions in Experiment 1, except they had the delayed feedback stimulus. The only difference is that the intercept is slightly higher resulting in an elevated response latency variance throughout acquisition. For subject J.V., the mean within-block variance for the last five sessions, plotted in Figure 26, is 207.4 msec^2 whereas for subjects G.T. and J.B., in Experiment 1, response latency variances obtained at equivalent levels of practice were 119.8 and 135.1 msec^2, respectively.

Although there is some hint of a difference in performance between the feedback and no feedback conditions when comparing mean within-block variances, the difference becomes much clearer when overall variances are compared. Overall variance for subject J.V. is 383.3 msec^2 relative to the mean within-block variance of 207.4 msec^2. This discrepancy between variance measures of over 175 msec^2

Figure 26 - Mean error of synchrony (top) and mean within-block variance (bottom) as a function of practice. Data are for subject J.V. synchronizing at a P1P2 interval of 310 msec with no delayed feedback pulse.

Figure 27 - Best linear fit and scattergram of the acquisition data, presented in Figure 26, plotted on log-log coordinates. The data are for subject J.V. with a P1P2 interval of 310 msec.


MEAN ERROR OF SYNCHRONY (msec)
+50
0
-50
VARIANCE (msec²)
1500
1000
500
10
20
30
SESSION NUMBER


7
6
5
LOG$_e$ VARIANCE
0
1
2
3
LOG$_e$ SESSION NUMBER

indicates very unstable data in which the mean response latency wanders about over time. In contrast, the mean difference between overall and mean within-block variances for subjects G.T. and J.B. was 7.2 $msec^2$ reflecting considerable stability and consistency in the control of their response latencies.

The lack of stability for subject J.V. is most apparent in the upper panel of Figure 26 which plots mean error of synchrony as a function of session number. Note that the scale has been increased by a factor of 5 from that used in the comparable plots presented in Experiment 1. Mean response latencies for this no feedback condition change considerably from session to session compared to those obtained when delayed feedback is provided. The mean absolute error of synchrony, over the first five sessions for subject J.V., is 40.3 msec while for subjects G.T. and J.B. (naive subjects in Experiment 1) the values of the same statistic are only 5.5 and 3.8 msec, respectively. Differences in stability between feedback and no feedback conditions are evident throughout acquisition. For example, mean errors of synchrony for sessions 25 to 30 cover a considerable range from -26.4 to +4.7 msec for subject J.V. while for subjects G.T. and J.B. the errors of synchrony were almost negligible (-1.1 to +1.6 msec for G.T. and +0.8 to +2.9 msec for J.B.).

Despite the instability of the synchronization responses produced by subject J.V., the distribution of responses latencies was similar in shape to those distributions observed in Experiment 1. It was symmetrical, with relatively straight sides, and differed only in its "spread" along the time axis – reflecting the wandering of the mean response latency over time.

An analysis of sequential dependencies (described in Experiment 1), applied to the data for subject J.V., revealed that changes in mean response latency occur very slowly over time. Chi-square values were significant (Chi-square = 205 to 359 on 1 d.f.) to beyond lag 10, indicating a strong positive autocorrelation between response latencies separated by as many as ten other synchronization responses. These results are quite different from those obtained in Experiment 1. With the delayed feedback available, response latencies were typically independent of one another and when sequential dependencies were evident, they were weak and limited to only immediately successive response latencies.

Removal of Feedback for Well-Practiced Subjects

Removal of the delayed feedback from the trial sequence for the subjects well-practiced in response-stimulus synchronization produced several interesting results. For subject G.H., who was synchronizing with auditory interval

markers, removal of the feedback actually resulted in an improvement in performance which was evident even during the first session following transfer to the no feedback condition. The reduction in variance averaged about 6.5 $msec^2$ when compared with performance on the five sessions immediately preceding the removal of feedback. These sessions were conducted under the baseline conditions for auditory synchronization, as described in Experiment 1. As it turns out, these variance levels were slightly higher than had been obtained by subject G.H. earlier. This may have resulted due to some interference caused by switching to visual synchronization for 105 sessions before returning the the auditory markers used in this experiment. Subject G.H. had only had 15 sessions of practice with auditory markers, following his visual synchronization experience, before the delayed feedback was removed. Therefore, a comparison of performance was also made with the low-variance response latency distribution observed in Experiment 1.

Results of this comparison are shown in Figure 28. In the left panel is the frequency distribution obtained during five consecutive sessions of the best baseline performance produced in Experiment 1 (with feedback) and in the right panel is a similar distribution obtained during five consecutive sessions in this experiment, without feedback. The two distributions are almost identical. Mean

response latencies for the distributions on the left and right are 461.1 and 461.8 msec, respectively, with mean within-block variances of 34.3 and 33.5 $msec^2$. Overall variances are only slightly higher, 34.6 and 34.7 $msec^2$, reflecting the remarkable stability in the data represented in these distributions. Both distributions are symmetrical, sharp-peaked, span exactly the same range along the time axis, and have underlying response latencies that are independent of one another.

Results of the analyses of sequential dependencies for the "no feedback" distribution are shown in Figure 29. In the upper panel is a graph of the mean error of synchrony on trial $(n+i)$ as a function of the time of response on trial n for lag i, i = 1, 2, and 3. The essentially flat plots indicate that the response latency on trial n is basically independent of the times of responses on preceding trials. Autocorrelation coefficients, displayed in the lower panel of Figure 29, provide further evidence of independence of response latencies for lags of up to 10. The only exception seems to be with lag 2 in which both forms of analysis indicate some slight degree of sequential dependency, however, this finding was not consistently observed in analyzing the other "no feedback" data, so it should not be considered reliable.

Based on the results just discussed, it is clear that, after a subject is well practiced in response-stimulus

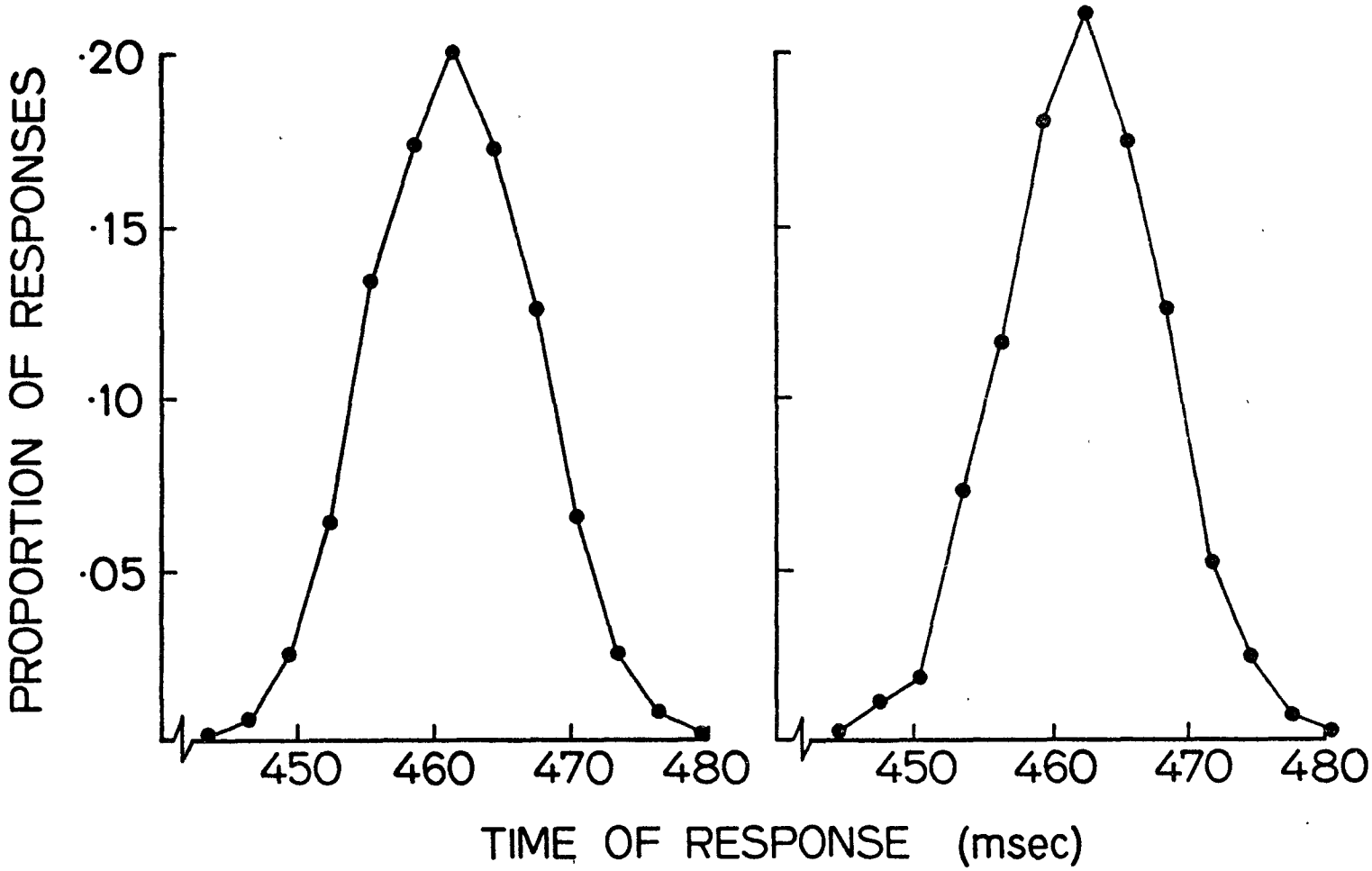
synchronization with auditory interval markers, the delayed feedback can be removed from the stimulus sequence without degrading performance in any way. There is even some evidence to suggest that performance improves in the absence of this type of feedback information.

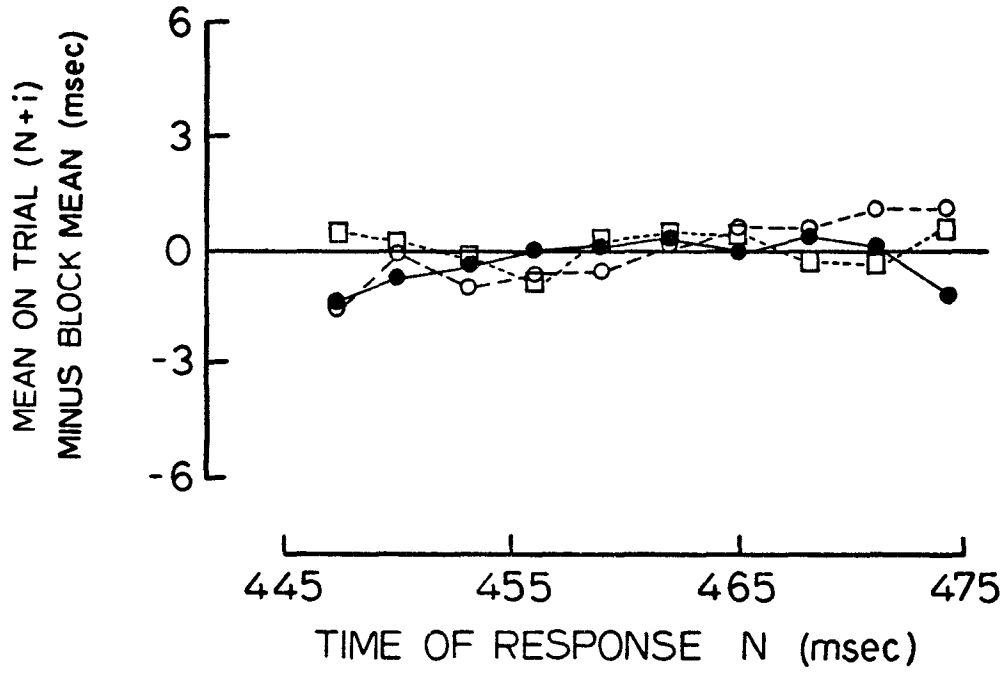
The same conclusion, however, is not true when synchronization has only been practiced with visual interval markers. For subject E.A., removal of feedback had an immediate detrimental effect which seemed to worsen the longer he was exposed to the "no feedback" condition. Both means and variances were affected. Prior to removal of the feedback, for five baseline sessions, the mean of the response latency distribution was 359.2 msec (error of synchrony of -0.8 msec) with a mean within-block variance of 61.4 $msec^2$ and an overall variance of 66.9 $msec^2$. After feedback removal, the error of synchrony increased substantially producing a mean response latency of 384.0 msec (error of synchrony of +24.0 msec). The mean within-block variance rose to 89.1 $msec^2$ while the overall variance was drastically inflated to 263.3 $msec^2$. Severe wandering of the mean from block to block is reflected in this large difference between the two variance measures. Block means ranged from 356.5 to 408.7 msec with the **P**1**P**2 interval fixed at 360 msec throughout.

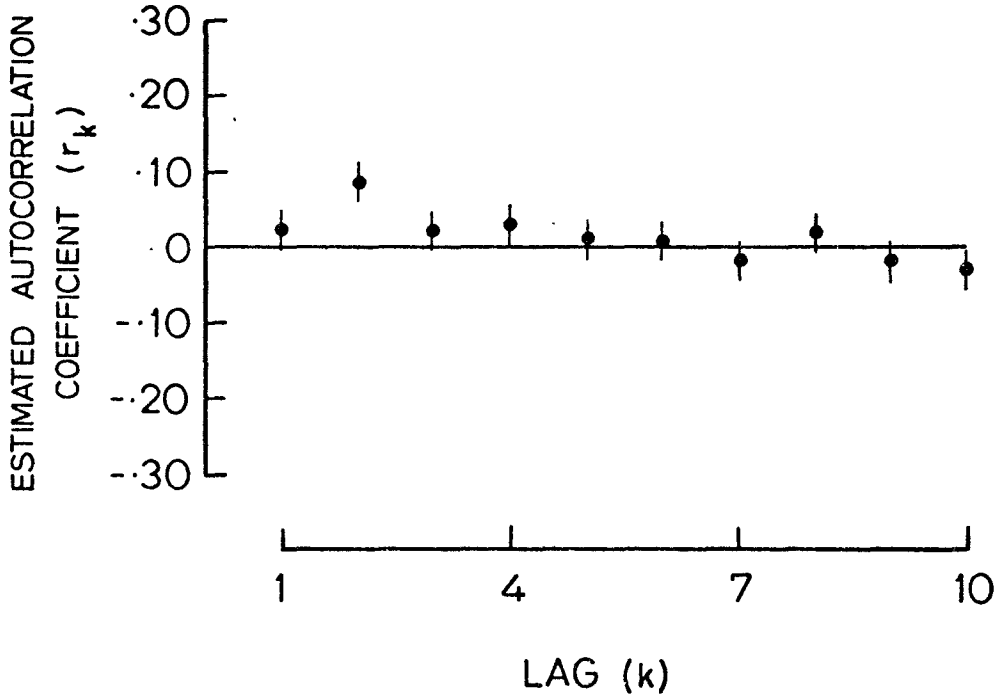
Unlike the case for subject G.H., the response latencies for subject E.A. were not independent of one

Figure 28 - Relative frequency distributions for two different feedback conditions. Left panel is for five sessions (266-270) of baseline synchronization with the delayed feedback signal present and the right panel shows analogous distribution (sessions 391-395) obtained with no delayed feedback. Both distributions consist of 1,500 response latencies each with a bin size of 3 msec. The data are for subject G.H. with a P1P2 interval of 460 msec. Means and variances are given in the text.

Figure 29 - Two types of sequential dependency analyses showing effects on lags 1 through 3 (top panel) and autocorrelation coefficients through to lag 10 (bottom panel) for subject G.H., on sessions 391-395, synchronizing at a P1P2 interval of 460 msec with no delayed feedback signal.


PROPORTION OF RESPONSES
·20
·15
·10
·05
450
460
470
480
450
460
470
480
TIME OF RESPONSE (msec)


MEAN ON TRIAL (N+i)
MINUS BLOCK MEAN (msec)
6
3
0
-3
-6
445
455
465
475
TIME OF RESPONSE N (msec)


ESTIMATED AUTOCORRELATION
COEFFICIENT (r_k)
·30
·20
·10
·0
-·10
-·20
-·30
1
4
7
10
LAG (k)

another, as shown in Figure 30. The plots in the upper panel indicate a strong positive dependency between response latencies up to lag 3 and the lower panel shows that sequential dependencies are indeed evident well beyond lag 3. Not until lag 9 does the value of the autocorrelation coefficient become nonsignificant. Obviously the effect of feedback removal is quite different depending on the modality of P1 and P2. Again, this points to an important difference between auditory and visual stimuli in this experimental paradigm. When the delayed feedback information is absent, the subjects must rely on the rather difficult judgment of the temporal relationship between the onset of P2 and the moment when their finger breaks contact with the response button, for providing information about how accurately they are responding. But, the fidelity and usefulness of this information is largely determined by the modality of P2. Removal of delayed feedback has basically no effect on auditory synchronization whereas this same condition produces a substantial decrement in performance when encountered during visual synchronization.

Interval Production by Well-Practiced Subjects

If P1 and P2 are also removed from the typical stimulus sequence for response-stimulus synchronization (delayed feedback has already been removed), the task basically becomes one of interval production, based on a memorial

representation of the synchronization interval which has presumably been built up over thousands of trials of practice. When subjects G.H. and E.A. experienced this condition both subjects exhibited rather large performance decrements relative to their baseline levels.

For subject G.H., who had been responding in the absence of the delayed feedback with very small errors of synchrony and variances below 35 $msec^2$, the effect of removing the interval markers was quite pronounced. The mean error of synchrony for a group of five consecutive sessions rose drastically to 30.1 msec, producing a mean of 490.1 msec, with a range of block means from 479.2 to 500.5 msec. Mean within-block variance increased by a factor of 10 to 314.5 $msec^2$ with an overall variance of 348.1 $msec^2$ and response latencies exhibited strong sequential dependencies up to lag 8, as shown in Figure 31.

Subject E.A. had been performing with relatively small errors of synchrony and variances in the 60 $msec^2$ range on the baseline conditions conducted just prior to this experiment. When the delayed feedback was removed both his mean error of synchrony and variance increased dramatically. Removal of P1 and P2 simply continued the trend of progressively worse performance. The mean response latency for a group of five sessions, without interval markers or feedback, was 360.1 msec, which is very close to the 360 msec interval required, however, the value of the

block means varied considerably, ranging from 325.3 to 385.0 msec. Mean within-block variance was 110.0 $msec^2$ and the overall variance was 302.5 $msec^2$. As was the case for subject G.H., strong sequential dependencies up to lag 8 were also present in the data for subject E.A.

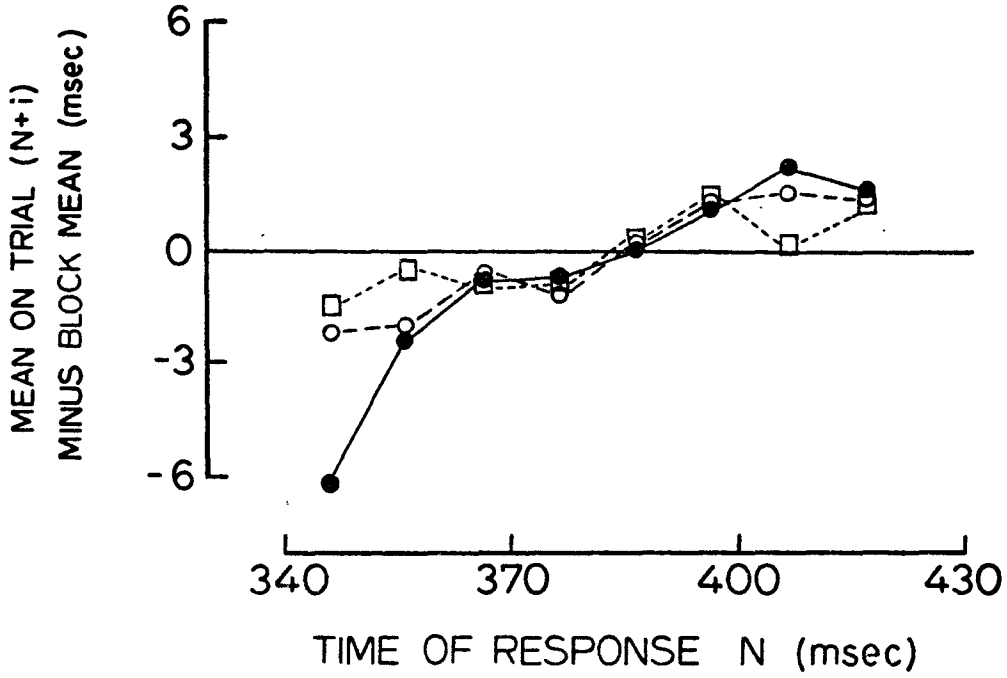
C. Discussion

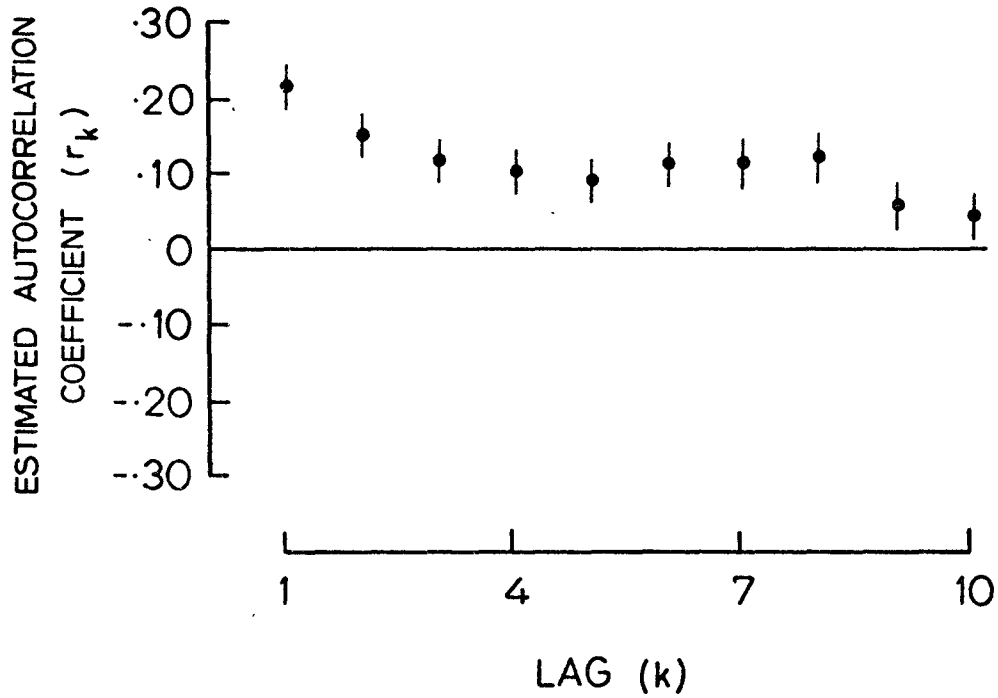
Clearly, the role played by the delayed feedback in the response-stimulus synchronization paradigm used in this series of experiments depends on several factors, including amount of practice and modality of interval markers. During initial acquisition, the delayed feedback information appears to be crucial for quickly centring the distribution of response latencies about the time-point corresponding to veridical synchrony. Yet, later in practice, this information can sometimes be withheld without altering either the mean or variance of the distribution of response latencies. This result, however, depends wholly on the modality of the interval markers to which the subject is used to responding. If P1 and P2 are auditory, then performance is unaffected and may even improve in the absence of delayed feedback; whereas, if P1 and P2 are visually presented performance decrements are quite severe.

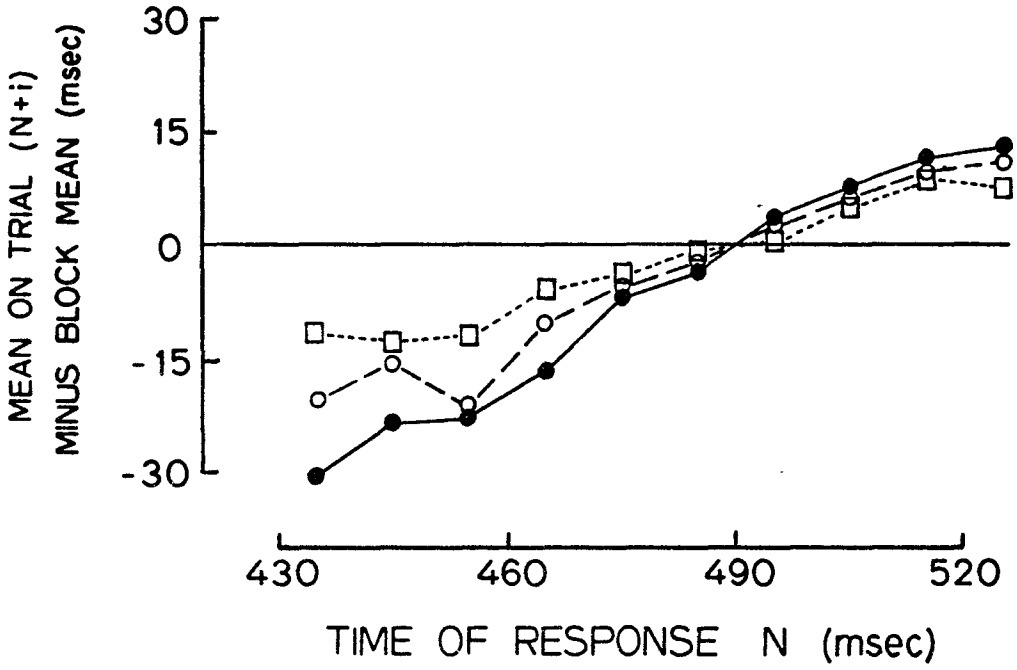
At this point in the discussion, it is probably useful to make clear the distinction between the different sources of feedback information that are available to the

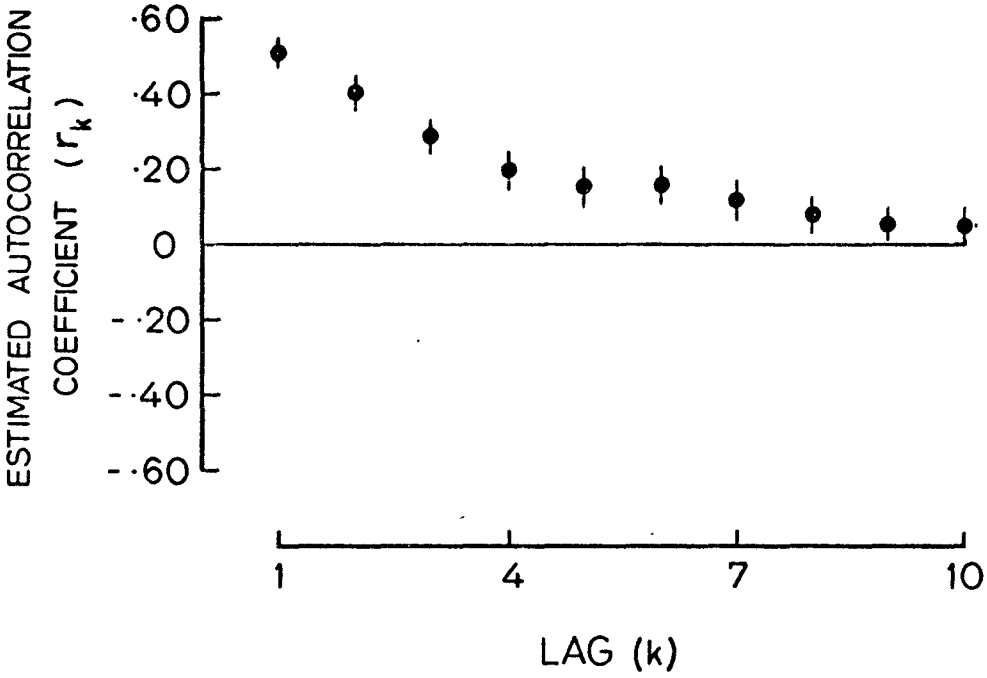
Figure 30 - Two types of sequential dependency analyses showing effects on lags 1 through 3 (top panel) and autocorrelation coefficients through to lag 10 (bottom panel) for subject E.A., on sessions 186-190, synchronizing with visual interval markers (360 msec interval) and no delayed feedback.

Figure 31 - Two types of sequential dependency analyses showing effects on lags 1 through 3 (top panel) and autocorrelation coefficients through to lag 10 (bottom panel) for subject G.H. on sessions 416 to 420, free-responding without interval markers or delayed feedback.


MEAN ON TRIAL (N+i)
MINUS BLOCK MEAN (msec)
6
3
0
-3
-6
340
370
400
430
TIME OF RESPONSE N (msec)


ESTIMATED AUTOCORRELATION
COEFFICIENT (r_k)
·30
·20
·10
0
-·10
-·20
-·30
1
4
7
10
LAG (k)


MEAN ON TRIAL (N+i)
MINUS BLOCK MEAN (msec)
30
15
0
-15
-30
430
460
490
520
TIME OF RESPONSE N (msec)


ESTIMATED AUTOCORRELATION
COEFFICIENT (r_k)
·60
·40
·20
0
-·20
-·40
-·60
1
4
7
10
LAG (k)

subject during response-stimulus synchronization. First, there is the obvious source of information provided by the delayed feedback signal. It consists of a tone which sounds for a duration equal to the magnitude of the error of synchrony. The presence or absence of a light accompanying this feedback pulse serves to indicate the direction of error. This form of feedback information was chosen for its saliency, fidelity, and ease of interpretability. It was quite clear to the subjects whether their response was early or late and they could easily distinguish between small errors of synchrony, such as 1 versus 5 msec, simply due to the large qualitative differences produced by the tones when presented at these two, short pulse durations. Furthermore, a few verbal instructions were all that were required for the subjects to be able to accurately interpret the meaning of the information provided by the delayed feedback pulse.

The other source of feedback is derived from the temporal relationship between the central registrations of two time-point events; namely, the onset of P2 and the moment of finger withdrawal from the response button. This type of information is much more difficult for the subjects to interpret. Hirsh and Sherrick (1961) have shown that stimulus onset asynchronies in excess of 20 msec are needed for accurate temporal order judgments. Yet, for well-practiced subjects, the errors of synchrony from trial to trial are typically less than 20 msec. This raises the

question concerning how this information can be utilized to provide the degree of accuracy observed in auditory synchronization when the delayed feedback is absent. The answer seems to involve a learning process. Although subjects may be relatively poor at making absolute temporal order judgements (being able to tell which stimulus came first), Efron (1973) has shown that some type of temporal information is still processed by our perceptual systems at temporal asynchronies well below the threshold for making temporal order judgments.

Efron presented pairs of brief, discriminably different auditory stimuli with onset asynchronies much smaller than that required for temporal order discrimination. These brief stimulus complexes were "experienced as a unitary perceptual event" and referred to as "micropatterns". But, when two such micropatterns were presented with the temporal order of the elements reversed, subjects were able to distinguish between the two events with stimulus onset asynchronies as small as 2 msec with 95% accuracy even though they could not tell which stimulus came first. The discrimination was based on a qualitative difference which seemed to emphasize the second stimulus element of each micropattern. Experiments were also conducted in the visual and vibratory modalities, with stimulus intensities adjusted to produce comparable levels of sensation. These results showed that discrimination was

far superior in the auditory modality then in the other two. Unfortunately, Efron did not examine any cross-modality discrimination abilities which would have been more directly relevant to the current problem of understanding how subjects doing response-stimulus synchronization make use of the information arising from the temporal relationship between their response and P2 onset for deriving knowledge of results.

Given the available data, though, it seems reasonable to suggest that some form of temporal information could be available to the subject, regarding the accuracy of his response, even with extremely small errors of synchrony. It also seems, however, that the nature of this information would be insufficient, by itself, to allow temporal order judgments to be made. But, if this somewhat obscure, immediate feedback information were combined repeatedly with the salient, easily interpreted, delayed feedback information, it seems reasonable that subjects might learn the meaning associated with the stimulus "micropatterns" produced by the close temporal proximity of P2 onset and the subjects's synchronization response. Such a learning process could account for the results which show that removal of delayed feedback after sufficient practice does not hinder performance and yet removal during the initial stages of acquisition produces very poor synchronization accuracy. Recall, however, that the former finding was only

obtained with auditory synchronization. In this modality, the source of feedback seems to transfer from the delayed signal to the immediate stimulation, accompanying the synchronization response, over the course of practice. In fact, the immediate perceptual feedback eventually comes to equal the combination of feedback sources. That is, well-practiced performance in the absence of the delayed feedback is better than performance in the absence of P2, which effectively removes the immediate feedback source.

The same type of transfer does not appear to develop when the visual modality is used for delimiting the synchronization interval. Presumably this is due to the relatively large temporal uncertainty associated with the central representation of the onset of a visual stimulus, as was discussed in the previous chapter. This notion is further supported by Efron's (1973) findings that discrimination of visual "micropatterns" required substantially larger stimulus onset asynchronies than did discrimination of auditory "micropatterns".

The import of this is that some type of feedback, or knowledge of results, is necessary to maintain accurate synchronization performance. For, when all sources of feedback are removed performance deteriorates immediately and continues to worsen over time. Whatever memory there is for the synchronization interval seems to be very volatile with respect to allowing the type of precision observed

during synchronization to continue during "free-responding". The reason for this is unclear, but perhaps it is due to random, or possibly non-random, variations in the rate of central information processing that we are not able to compensate for during subsequent stimulus-response chains unless we receive feedback from the external world. That is, we are capable of synchronizing responses with events in the "real world" very accurately, but only if we are provided useable feedback confirming the appropriateness of our actions. In the event of any minor discrepancy in synchronization, the system appears to be able to use the information to quickly and accurately engineer slight corrections into the timing of the stimulus-response chain, before any significant errors can be accrued, preventing any autocorrelation from occurring. When such information is unavailable, however, the subject becomes slave to variations in the internal state of his central nervous system and this variability is then added into any measurements of response timing consistency. This is an interesting concept because it suggests the possibility of measuring the extent and nature of fluctuations in the internal state of an organism, over time, by simply comparing the mean and variance obtained during standard response-stimulus synchronization with the performance data observed in a free-responding situation.

Although the data from the present experiment are insufficient for drawing any definite conclusions, one could speculate as to what several of the results would mean in the conceptual context just described. For example, if corrections can be made to the timing of a stimulus-response chain, based on feedback, in order to maintain synchronization with events in the environment, despite changes in the internal state of the organism, then one would expect any individual differences to be minimized by the provision of feedback. This is exactly what has been observed throughout the series of experiments discussed so far. When salient, precise feedback information is available, differences in response-stimulus synchronization performance across individuals are almost negligible. When the feedback is removed, however, much larger individual differences emerge.

In the case of the "free running" (no form of feedback) data for subjects E.A. and G.H., the largest differences seemed to be in the ratio of overall variance to mean within-block variance. For subject G.H. this ratio was near 1 – the spread between the two variances measures was quite small – while for subject E.A. the difference was much larger, producing a ratio of nearly 3. These findings suggest that fluctuations in the internal state of information processing may occur more slowly for subject E.A. than for subject G.H. With sufficient data and some

extensive time-series analyses one might be able to extract the nature of these fluctuations; that is, whether they exhibit any cyclic variations or periodicities.

How is the feedback information used to alter stimulus-response timing in order to compensate for changes in central information processing characteristics? Well, it is clear that it is not on a trial to trial basis because no autocorrelation between response latencies is evident in the data from well-practiced subjects. Rather, it seems quite clear that some strategy which calculates a running average of errors of synchrony is involved in the process of making compensatory timing adjustments. The exact number of trials that are averaged together or the weighting that is given to each error of synchrony is not known, but the results indicate that the information from several preceding trials is probably taken into account.

Michaels (1977) introduced perturbations of ±15 msec into the timing of **P**1 relative to **P**2 in a synchronization paradigm and found that response latencies were time-locked to **P**1 and that latency variances were unaffected by the manipulations. In the case of **P**1 being delayed 15 msec, the **P**1**P**2 interval was, in effect, 15 msec shorter such that the synchronization responses on these trials occurred about 15 msec late on the average. Similarly, when **P**1 was presented 15 msec earlier than usual, responses averaged 15 msec early, relative to **P**2. The two types of perturbed trials

occurred equally often and accounted for one-half of the number of trials presented during a session. On all trials, both the perceptual feedback and a delayed visual feedback signal indicated the subject's errors of synchrony reliably. Yet, regardless of the temporal position of **P**1, and despite being presented with feedback representing rather large errors of synchrony on perturbed trials, subjects consistently produced response latency distributions which were relatively constant with respect to **P**1 onset with stable latency variance. There was certainly no indication of error correction on a trial-to-trial basis, and in fact, subjects were unaware of the manipulations. This strongly suggests that the subjects were mediating their response latencies by averaging the errors of synchrony over several preceding trials. Since the distribution of temporal perturbations was symmetrical, the rather large errors of synchrony accrued on perturbed trials would cancel with one another maintaining consistent responding from trial to trial despite the perturbations. However, this only happens if the distribution of temporal perturbations is symmetrical, as it was in this case. If the perturbations had been asymmetrically distributed then one would expect the mean response latency to migrate towards the more frequently ocurring, perturbed, **P**1**P**2 interval. Michon (1968) has also provided evidence for such an averaging process based on data from a tapping task.

The foregoing chapters have provided a mass of empirical data on response-stimulus synchronization and the discussions of these data have led to some theorizing concerning timing and information processing within the central nervous system. In the next chapter, the theoretical implications of these data will be extended in order to formulate a model that can be mathematically represented.

VI. Mathematical Model of Response-Stimulus Synchronization

Several models have been proposed in the literature to account for the various timing capacities of the human central nervous system. Many of the mechanisms incorporated into these models hypothesize a "time base" of some sort which generates a succession of temporal cues that can be used by the central nervous system for response timing (e.g.,, Michon, 1967) and for controlling the gating of information flow from one central stage to another (Kristofferson, 1967).

In some models, the internal clock varies somewhat in rate causing successive temporal judgments to be variable. The mechanism for timing in these models involves accumulating clock pulses, during the duration to be judged, from a source with identically distributed interpulse delays. In Creelman's (1962) model this source is assumed to be Poisson distributed, whereas for Triesman (1963) the nature of the distribution is not specified. In both cases, however, the models predict increasing variance in temporal judgments as a function of the mean interval to be represented. In some experiments the results suggest a monotonically increasing linear function between the variance and mean (e.g., Creelman, 1962) whereas other results suggest a similar relationship, but between the standard deviation and mean (e.g., Getty, 1975; Wing &

Kristofferson, 1973b). In any event, neither model is appropriate for describing the current response stimulus synchronization data in which the variance and mean of the response latencies are independent over a substantial range of temporal intervals, suggesting some kind of deterministic timing mechanism.

There are a few references in the literature to a fixed and constant time base or "time quantum" (viz., Kristofferson, 1967; McGill, 1962; Michon, 1967) but convergent evidence for the existence of a universal value for such a temporal unit has not been found. Kristofferson (1967) provides evidence of a correlation between the so-called "time quantum" and the half-period of "alpha rhythm" activity in the brain, suggesting a quantum value near 50 msec. In subsequent studies (Kristofferson, 1976, 1977), however, it became apparent that the underlying "time quantum" governing a subject's performance did not always seem to be a fixed value. The results from duration discrimination experiments (Kristofferson, 1980) indicated that under certain conditions the "time base" being used involved a "doubling" or "halving" of the "time quantum" value associated with the half-period of the subject's alpha rhythm.

A deterministic type of model was proposed by Kristofferson (1976) to account for the response-stimulus synchronization performance he observed in his experiments.

Latency distributions revealed a simple, homogeneous stimulus-response unit that was the same whether the mean was at 160 or 550 msec. It was proposed that the elementary response latency distributions observed resulted from the convolution of three independent sources of variance associated with producing the stimulus-response chain. One of these component distributions is normally distributed and represents variability in the efferent delay between the time when the response is triggered internally and when the overt response is produced. Afferent latencies, on the other hand, are assumed deterministic or non-variable. Support for this assumption comes from temporal order discrimination (Allan, 1975) and duration discrimination data (Kristofferson, 1977). The other two sources of variance are assumed to be identical and independent uniform distributions spanning a range of one time quantum. When convoluted, these produce a triangular distribution spanning two time quantum units. These delays, according to the model, represent variable delays in the processing of the stimulus-response chain, but the exact nature or locus of these delays is left unspecified. Also, there is no mechanism proposed to account for the assumption of independence between the quantal units. If these delays result from the operation of a single central mechanism, which gates information through the central information processor, then it is difficult to explain how the two

delays, assumed quantal in nature, and their associated variances can be considered independent because they presumably are both dependent upon the same, non-random, underlying process.

A. Empirical Considerations and Development of the Model

In spite of these criticisms, Kristofferson's model does provide quite accurate predictions of the asymptotic response latency variance obtained in Experiment 1. These predictions were based on an estimate of a minimum time quantum of 12 msec, suggested by some duration discrimination work (Kristofferson, 1977, 1980), and a minimum efferent delay variance of 10 $msec^2$, based on interresponse interval timing experiments (Wing, 1973b). The model, however, never tested the fit of the hypothetical convolution of the two uniform distributions with a normal component. Thus, it is the aim of this chapter to further examine some of the empirical data relevant to specifying the characteristics for a revised model of anticipatory timing, outline the resulting model in detail, and provide mathematical support for the model's ability to accurately represent the data.

First of all, it is probably advisable to briefly review the basic task to be modelled. In response-stimulus synchronization, the subject must perform anticipatory timing, which involves the timing of a stimulus-response

chain relative to environmental stimuli presented in "real time". These stimuli consist of two brief auditory pulses which are always separated by a fixed time interval, such that the time of occurrence of the second stimulus, P2, can be predicted based on the time of onset of the first pulse, P1. In order for the subject to be successful in synchronizing an overt response with the onset of P2, the subject must anticipate the occurrence of P2, timing from P1 onset, and initiate the desired response prior to the actual occurrence of P2. If timing of the response triggering is correct the resulting effect is a perfectly timed S-R chain in which the overt response occurs simultaneous, or in close temporal coincidence, with the occurrence of P2.

Obviously, variability on this temporal axis can arise from various sources. These include inconsistency in the afferent delay between P1 onset and its registration with the timing mechanism, variability associated with the timekeeping process itself, and variance in the efferent delay between response trigger and overt response. As was mentioned earlier, however, the data from several experiments indicate that afferent latencies are deterministic for auditory and tactile stimulation (see Chapter 2) which means that although the afferent delay has some non-zero value associated with it, the delay involved has negligible variance. This leaves only variance in the timing process and in output of the response to be dealt

with.

B. Independence of Central and Motor Components

The question arises whether it is possible to further reduce the number of sources of variance contributing to the shape of the response latency distributions. Is it possible that all the necessary timing for delaying a response for a few hundred milliseconds could be handled within the motor system? In other words, is it conceiveable that a sequence of motor commands, or motor program, could be operating, using a series of unobservable responses to delay an S-R chain which ultimately ends with elicitation of the overt response? Consideration of the relevant research findings allows rejection of such a possibility. These findings indicate that both central and motor components are involved in timed responses which are elicited by stimuli and, in addition, that these two components are independent.

If sequencing of motor components were responsible for the timing of delayed responses, then one would expect such a process to be manifest most clearly in an interresponse timing paradigm in which a repetitive sequence of similar responses, spread out over time, is required. However, this does not appear to be the case. An analysis of carefully collected interresponse timing data, conducted by Wing (1973), indicated that response latency variance was basically a constant – independent of the mean interresponse

interval being produced. Total variance increased with interresponse interval, but this was attributed to increases in the variability of some central processing component which appeared to be responsible for triggering of the overt responses at the appropriate times.

In simple and delayed reaction time paradigms, several experiments have measured EMG and response time, then looked at the correlation between pre-motor time (time from onset of the action stimulus to EMG-onset) and response time versus the correlation between motor time (time from EMG-onset to the overt response) and response time. The results revealed a correlation close to zero between motor time and response time, indicating independence (Botwinick & Thompson, 1966). In the delayed RT situation, the electromyography activation preceded the overt response by a relatively constant interval regardless of the actual response latency produced (Saslow, 1968).

A similar result was obtained by Michaels (1977) using a response-stimulus synchronization paradigm and a countermanding procedure. The countermand signal consisted of a third, brief auditory stimulus that was randomly presented, on one half of the trials, during the P1P2 synchronization interval. When the countermand signal was presented, the subject was required to try and withhold his synchronization response. The data consisted of the proportion of correctly countermanded responses as a

function of the time between countermand signal and P2 onsets. Michaels argued that the 50% point on this psychophysical curve should provide an estimate of the time when the response is triggered. The underlying rationale is that if the countermand signal occurs prior to the trigger, the initiation of the motor program can be successfully suspended, whereas if the signal is too late, the response sequence is already initiated and cannot be retroactively cancelled. The estimates of response trigger timing suggested that the trigger always precedes P2 onset by a fixed time period and is independent of the P1P2 interval being used. These findings, plus those discussed earlier, lead to the conclusion that manipulations affecting anticipation affect only the central, pre-motor component of a stimulus-response chain and argue against efferent stages having any major participation in timekeeping.

It is important to distinguish anticipatory timing, which involves central timing of a particular interval followed by triggering of a very simple, elementary movement, from the other types of timing required in more complex chained movements. It may be that similar central mechanisms are used in timing both simple anticipatory responses and complex, pre-planned motor acts, but much of the motor movement literature suggests that some, so-called "timing" is simply a by-product of the delay required to execute several motor behaviour components chained together

(i.e., Adams & Creamer, 1962; Jones, 1972; Keele, 1973; Schmidt & Christina, 1969; and Tyldesley & Whiting, 1975). Consequently, the current discussion will not address itself to the problem of timing more complex motor acts sequenced in time.

The last piece of evidence to be offered in support of the conclusion that the efferent stages are not involved in the precise timing and timing adjustments observed in anticipatory timing comes from the response-synchronization data itself. In fact, the finding of independence between response latency variance and mean over a wide range of P1P2 intervals strongly suggests the existence of an adjustable, non-variable, central delay mechanism. For if the delay was accomplished by lengthening the duration of the response sequence, then this strategy would involve adding a number of motor components into the simple S-R chain, each with some inherent variability. Thus, when all these components were combined to generate the particular timed response desired, one would expect a commensurate increase in variance as well. This is not the case. Consequently, all the available evidence indicates that efferent delays are distributed with a constant mean and standard deviation despite relatively large changes in the overall mean response latencies produced during synchronization with different P1P2 intervals.

C. Efferent Delay Variance

The next point to be discussed, before formulation of the model, concerns the accuracy and variance associated with the motor component of the response latencies. The overt response itself is just a simple finger withdrawal, but within the motor system there are actually several elements involved in producing the movement. The interesting point is that each of these underlying elements, when measured separately, exhibits a rather large temporal jitter, yet the outcome of their joint action produces a response which is well defined in time.

Meijers and Eijkman (1974) have examined this apparent paradox and offer an explanation based on the macro-activity required in the motor system before an elementary movement can be initiated. In their analysis, both the firing pattern of the efferent neurons and contraction of the muscle fibres are assumed to be non-deterministic processes such that the time elapsing between spike generation and activation of a particular motor element varies on successive innervations. Different elements may also vary, amongst themselves, in their time course of activation even though initiated at the same time. This produces the rather large time jitter associated with these element activities. Execution of the overt response, however, requires the joint effort of many elements. It is this requirement of joint activity that allows the

remarkably small stochastic variation associated with the timing accuracy of the observed, ballistic movements to be obtained. This is accomplished by summation of element activities, thereby providing better time definition than that produced by each of the individual activities. In other words, it is the averaging of the behaviour of several independent elements that is responsible for cancelling out the effect of their individual temporal inaccuracies (Meijers & Eijkman, 1974).

A significant change in temporal accuracy can be accomplished with relatively few elements. The pyramidal tract neurons, which innervate the motor system, have a rather large time jitter with standard deviations close to 20 msec (Meijers & Eijkman, 1974). On the other hand, accuracy for the timing of well-practiced, ballistic movements is estimated to have a standard deviation close to 3 msec (Wing & Kristofferson, 1973b). If these estimates are valid, then participation of less than 50 units would be required to produce a standard error of the mean consistent with the 3 msec value. Thus we can treat the response system as being very accurate, with efferent delays adding as little as 9 $msec^2$ variance into the response latency distributions obtained during response-stimulus synchronization.

Some research (e.g., Wing, 1977) suggests that lower efferent delay variances are obtained when larger muscle

groups are employed, but this finding is related to repetitive movement in a tapping task in which muscle fatigue becomes a factor. One would expect the larger the muscle group involved, the smaller the effect of fatigue would be. However, in the current context, with discrete trials, fatigue is probably not an important factor.

D. Description of the Model

From the foregoing discussion, it is obvious that several factors must be taken into account in the formulation of a mathematical model that will not only provide a good representation of the data, but will also have parameters which are psychologically relevant. The latter stipulation is very important because without it, the model would have little utility in generating testable predictions both for furthering our understanding of the internal mechanisms underlying response-stimulus synchronization behaviour and for extending the existing theory and model.

Consequently, a rather traditional approach was taken in formulating the model. It was based on the notion that if the data are stable, then any stochastic processes associated with the time involved in each of a series of independent processing stages would reflect themselves in the overall response latency distribution given by the convolution of all of the component stage distributions.

The model for response-stimulus synchronization is diagramatically presented in Figure 32. Basically, it is just a modification of Kristofferson's (1976) model which attempts to provide a locus for the central delays, incurred in processing the stimulus-response chain, as well as account for the assumption of independence made between the central delay components. It should be noted that in the process of describing the model in detail, some terms will be borrowed from the discipline of computer science to facilitate an understanding of the mechanisms involved. However, this is purely eclectic and should not be construed as implying any type of direct analogy. It should also be noted that this discussion assumes a steady-state condition exists in the central information processor. Violation of this assumption will be considered in subsequent discussions.

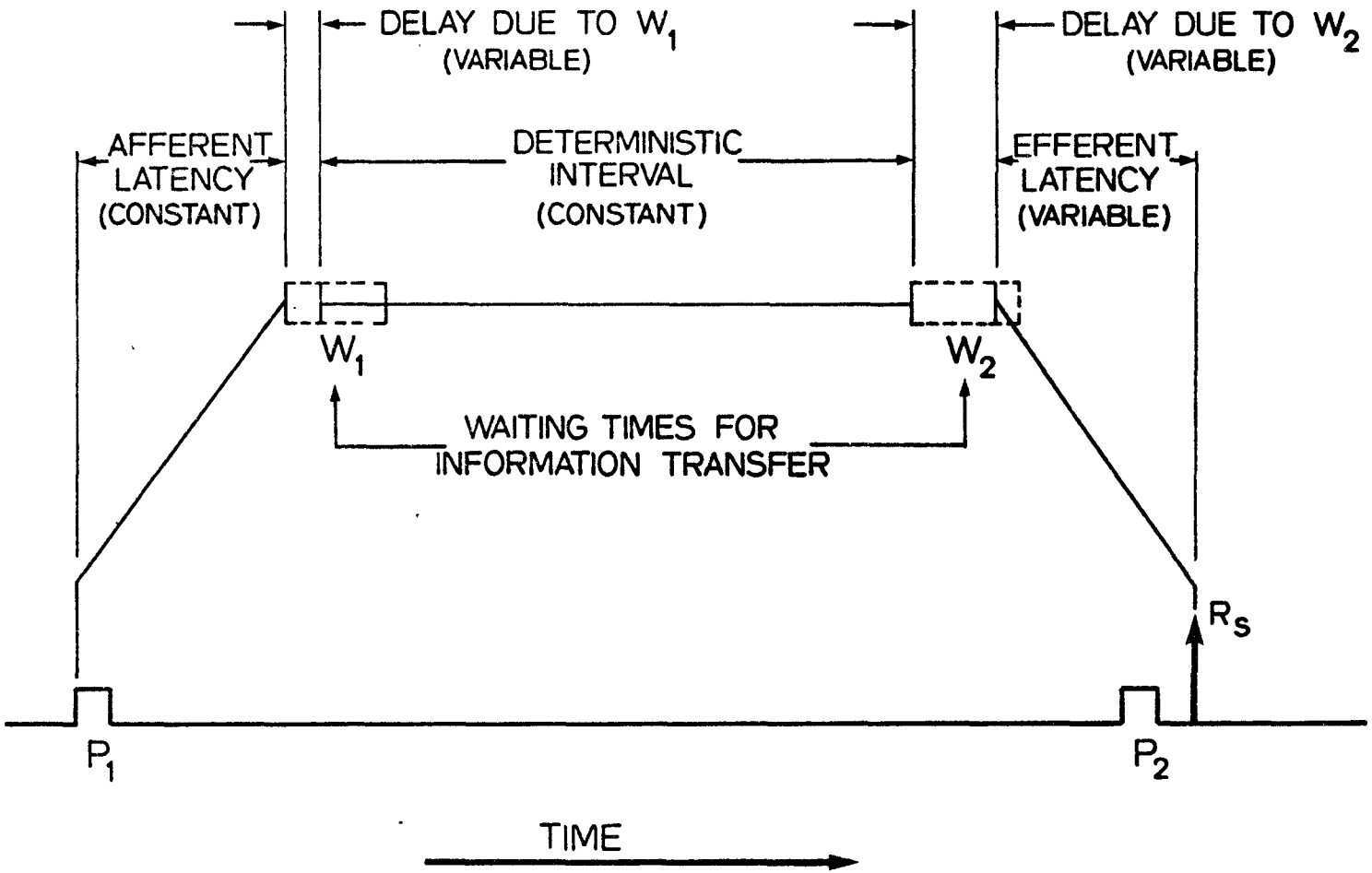
The onset of P1 is a sharply defined external event, however, its sensory effect is extended over time, as shown by the interval labelled afferent latency. This latency refers to the time from onset of the peripheral stimulus until an internal state has developed, as a result of P1 stimulation, which is sufficient to exceed some criterion and cause triggering of the next stage in the information processing chain. This process is similar to filling an input buffer and setting a flag which indicates that information is available for further processing. In this

context, the afferent latency can be considered as a combination of the time required for transduction of the stimulus information at the peripheral receptor, the conduction time from periphery to the central system, plus the time required to represent this information in one of the registers of the central processing unit.

Although the afferent latency certainly has some non-zero value, its variability is assumed to be negligible. This assumption is based on several pieces of evidence discussed earlier. Therefore, the afferent latency can be considered a constant, contributing nothing to the variance and shape of the observed distribution of response latencies.

Once the stimulation produced by **P**1 onset is registered internally, in a buffer, the information must wait for a period represented by **W**1, before gaining access to subsequent processing stages. This waiting time for information transfer from the input buffer to the deterministic timekeeping mechanism results because the contents of the input buffer are only accessed periodically. The concept is similar to that involved with the cycle time of a computer. Every *n* units of time the central processor reads the contents of the input buffer and then performs operations based on this information. (For the purposes of the current discussion, based on a biological processing system, the most appropriate units of time are milliseconds,

Figure 32 - Diagram of response-stimulus synchronization model. **P**1, **P**2, and **R**S are external, observable events while **W**1 and **W**2 are hypothesized internal events. The diagram represents the time course of various components of a stimulus-response chain on a typical trial.


DELAY DUE TO W_1
(VARIABLE)
DELAY DUE TO W_2
(VARIABLE)
AFFERENT
LATENCY
(CONSTANT)
DETERMINISTIC
INTERVAL
(CONSTANT)
EFFERENT
LATENCY
(VARIABLE)
W_1
W_2
WAITING TIMES FOR
INFORMATION TRANSFER
R_S
P_1
P_2
TIME

however, in computer systems the time units are typically substantially smaller.) Since this cycle time, or scheduling of access time-points, is independent of peripheral stimulation, the delay due to **W**1 is variable and uniformly distributed over a range from zero to **W**1*max* milliseconds. For example, sometimes stimulus information will reach the input buffer just before the start of a new internal cycle so this information will gain access to the central processor with very little delay. On the other hand, stimulus information loaded into the input buffer just after the start of a cycle will have to wait almost an entire cycle period, **W**1*max*, before gaining access to the central processor.

Following access of the stimulus information to the central processor, or in this case, the deterministic timekeeper mechanism, a delay appropriate to the synchronization interval and state of the organism is assumed to be generated. This delay has negligible variance associated with it, hence the use of the term – deterministic interval. Physiological mechanisms capable of producing such delays in the processing of a stimulus-response chain are not forthcoming from the current literature, but the rationale for assuming the existence of such mechanisms is clear from the preceding discussion and experiments.

The termination of this deterministic delay produces information which is then loaded into an output buffer and generates a flag in a fashion similar to that described for the input buffer. This time, however, the information is waiting to gain access to the response processor. This processor also has a fixed cycle time, **W2***max*, which is similar to that of the central processor, **W1***max*, thus generating uniformly distributed delays over a range from zero to **W2***max*. Although **W1***max* and **W2***max* are similar, they are not exactly the same. As a result, the two processors cycle in and out of phase relatively frequently during the synchronization interval. Consequently, the two waiting times can be considered independent, since the initiation of a synchronization interval (onset of **P1**) is totally independent of any phase relationship that exists between central and response processor cycle times.

At this point in the description of the model, it might be worthwhile to further clarify this proposed mechanism, which allows the assumption of independence between **W1** and **W2** to be made. Suppose **W1***max* is 12 msec and **W2***max* is 13 msec. These values can be considered the periods, or cycle times, of the periodic processes responsible for the waiting times **W1** and **W2**, respectively. Thus, these two processes will pass in and out of phase every 156 msec (12x13). Now, consider how **W1**, **W2**, and their phase relationship is related to the stimulus sequence used

in response-stimulus synchronization. The occurrence of P1 is assumed to be independent of W1 for several reasons. First, the trials are subject-paced such that the intertrial interval varies greatly with respect to the cycle time responsible for W1. Second, there is no evidence to suggest that the conscious decision to elicit the initiation response, R*i*, is dependent, in any way, on the central process responsible for W1. And third, even if one postulated a relationship existing between the W1 process and R*i* triggering, its characteristics would be lost due to two sources of temporal variability interposed between triggering of R*i* and the occurrence of P1. One source of variance is due to the efferent delay variance between the central response trigger and the overt response, R*i*, and the other is due to the experimentally introduced, variable foreperiod between R*i* and P1 onset. Both sources are random and relatively large with respect to the cycle time of W1. Therefore, the occurrence of P1 onset can be considered independent and random with respect to any time-point in the W1 cycle, resulting in a uniform distribution of W1 waiting times.

How is W2 independent of W1? Well, the best way to explain the independence assumption involves visualizing two continuous sine waves, one representing the periodic process underlying W1, with a period of 12 msec, and the other representing the process underlying W2, with a period of 13

msec. At some random point in time, P1 onset is registered centrally in the input buffer and must wait for a new cycle to begin (W1 delay) before this information receives further processing. Following W1, an appropriate deterministic interval is generated before the information is registered in the output buffer, but because this interval is a constant, it need not be considered in the determination of the relationship between W1 and W2. Thus, the problem can be simplified to taking a hypothetical "slice in time" through both sine waves and looking at the phase relationship that exists between the two. The key lies with the fact that P1 onset occurs randomly with respect to the W1 cycle. Since the processes responsible for W1 and W2 slide in and out of phase relatively frequently during the intertrial interval, knowing at what point in the cycle of W1 was intersected at some random slice in time provides no information about what part of the W2 cycle will be intersected at that same point in time. Consequently, W1 and W2 can both be considered independent, and uniformly distributed. As a result, the convolution of the distributions of these two waiting times will generate a unit of central temporal variability which is basically triangular (see Appendix E), as long as the values of W1*max* and W2*max* are not too dissimilar. A slight difference in periodicities, as discussed, only produces a slight bluntness in the peak of the triangular distribution.

Finally, since P1 onset, W1, and W2 can all be considered independent, because P1 occurs randomly with respect to the other events, no autocorrelation should exist between trials either, which is consistent with the data.

Having finished examining the characteristics of the central processing of response-stimulus synchronization information, only the characteristics of the output stage need yet be considered to complete the processing chain which culminates in the subject's synchronization response. Thus, following information transfer from the output buffer of the central stage to the response processor, the appropriate action (finger withdrawal) is triggered and, after an efferent latency, the overt response (RS) is produced. As discussed earlier, these efferent latencies are assumed to be normally distributed with a relatively small variance (Meijers & Eijkman, 1974).

The ultimate goal of this sequence of processing stages is to produce a response which is perfectly synchronous with the onset of P2. Due to variability incurred at various stages, however, the best performance that can be realized involves centring the response latency distribution about the time-point corresponding to P2 onset and minimizing the variance of the various stochastic components.

As discussed earlier, the overall response latency distribution is given by the convolution of the component

distributions. In the model, this involves convoluting the distributions associated with **W**1, **W**2, and the efferent latency. Although **W**1*max* and **W**2*max* are not exactly the same, for purposes of modelling they will be considered as identical. Thus, the convolution of the distributions of waiting times for information transfer will produce a triangular distribution spanning a range of 2x**W***max*. Further convolution with a normally distributed component will produce a distribution which is basically triangular, with short tails and a slightly blunted peak, if the normal component has relatively small variance. The complete mathematical derivation for the convolution of these component distributions and the formulae for the resulting density and distribution functions are provided in Appendix E. For purposes of simplifying the derivation, a logistic distribution was substituted as an approximation to the normal.

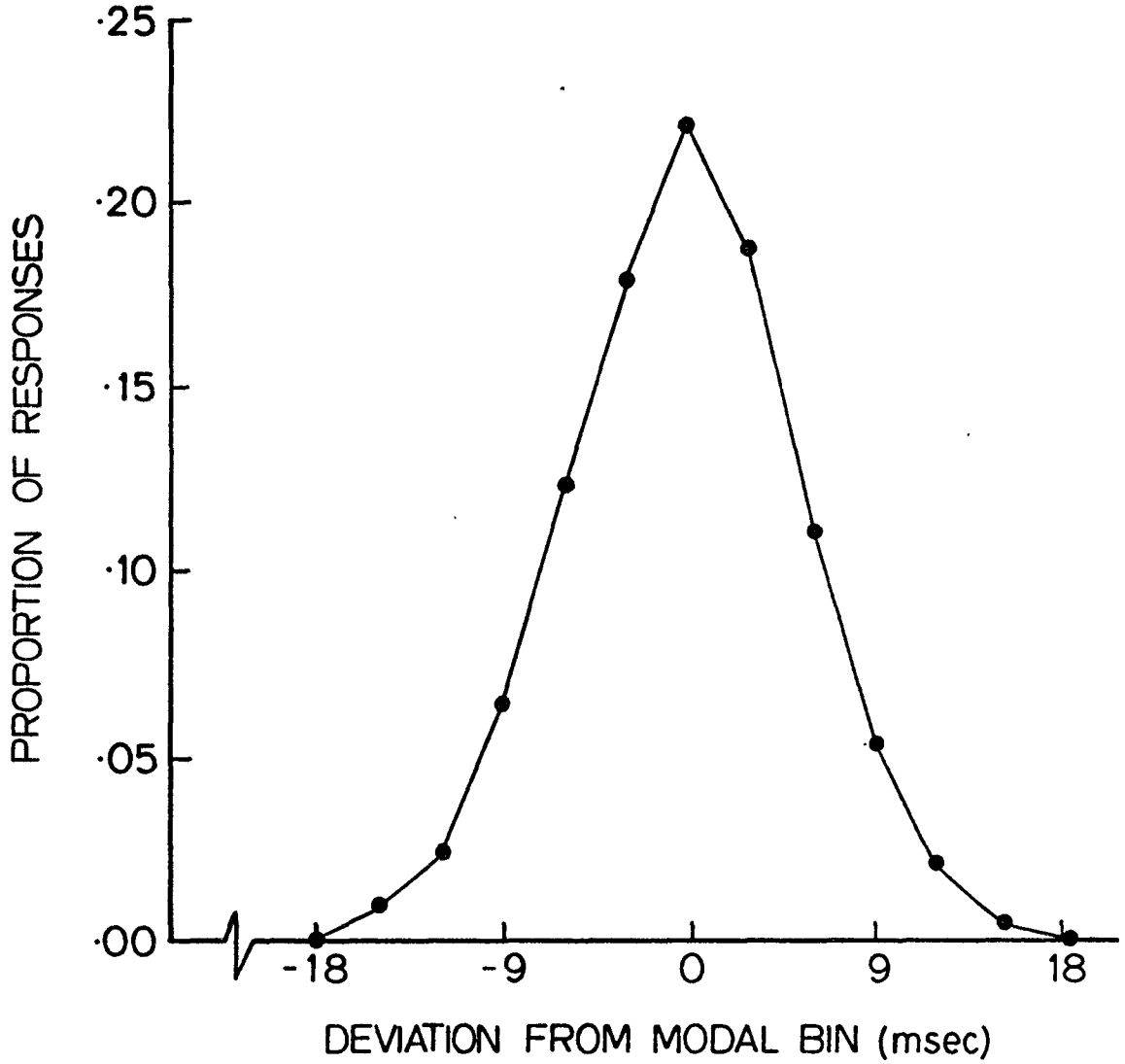
E. Goodness-of-Fit Testing

The general description of the shape of distribution that is generated by the model seems to characterize the data quite well. Figure 33 shows a response latency distribution composed of 1,500 response latencies gathered during 5 sessions of very stable performance from subject G.H. Its shape seems to be perfectly described by the model, however, a more rigorous test of the model's ability

to represent the data was obtained by mathematically testing the goodness-of-fit between the distribution function generated by the model and the cumulative probability of response distribution derived from the data.

Results of such comparisons, conducted with baseline data, are shown in Figures 34 through 37 for different subjects and for different levels of practice. Actual values used for the plots are presented in Appendix C. The procedure used for making these comparisons involved selecting a mean for the theoretical distribution, based on interpolation of the point in the data where the cumulative probability of a response was equal to 0.5, and then determining the corresponding time of response. Parameters of the model, representing the variable components, were estimated by allowing them to vary over a calculated range while repeatedly testing for goodness-of-fit via the minimizing Chi-square technique. The two parameters estimated consisted of **W**, which represented the average of $\mathbf{W}1_{max}$ and $\mathbf{W}2_{max}$ (the maximum times required for information transfer), and **b**, which represents the standard deviation of the efferent response latency distribution. Values for **W** and **b** covaried and were constrained by the overall variance of the observed distribution. Variance of the triangular distribution is $\mathbf{W}^2/6$ and variance of the normal distribution is approximated by $\mathbf{b}^2$. Thus, the equation for the overall response latency variance $V(total)$ is:

Figure 33 - Relative frequency distribution composed of 1,500 response latencies obtained from five of the best (lowest variance) sessions from those sessions numbered 391 to 410 for subject G.H. P1P2 interval is 460 msec which is also the lower bound on the modal bin. Bin size is 3 msec.


·25
·20
·15
·10
·05
·00
PROPORTION OF RESPONSES
-18
-9
0
9
18
DEVIATION FROM MODAL BIN (msec)

$$V(total) = W^2/6 + b^2$$

Therefore, the constraints are clear. If b=0 then:

$$Wmax = \sqrt{6 \times V(total)}$$

and conversely, if W=0 then:

$$bmax = \sqrt{V(total)}$$

Moreover, for any W chosen in the range from 0.0 to *Wmax* the value of b is fixed by the following equation:

$$b = \sqrt{V(total) - W^2/6}$$

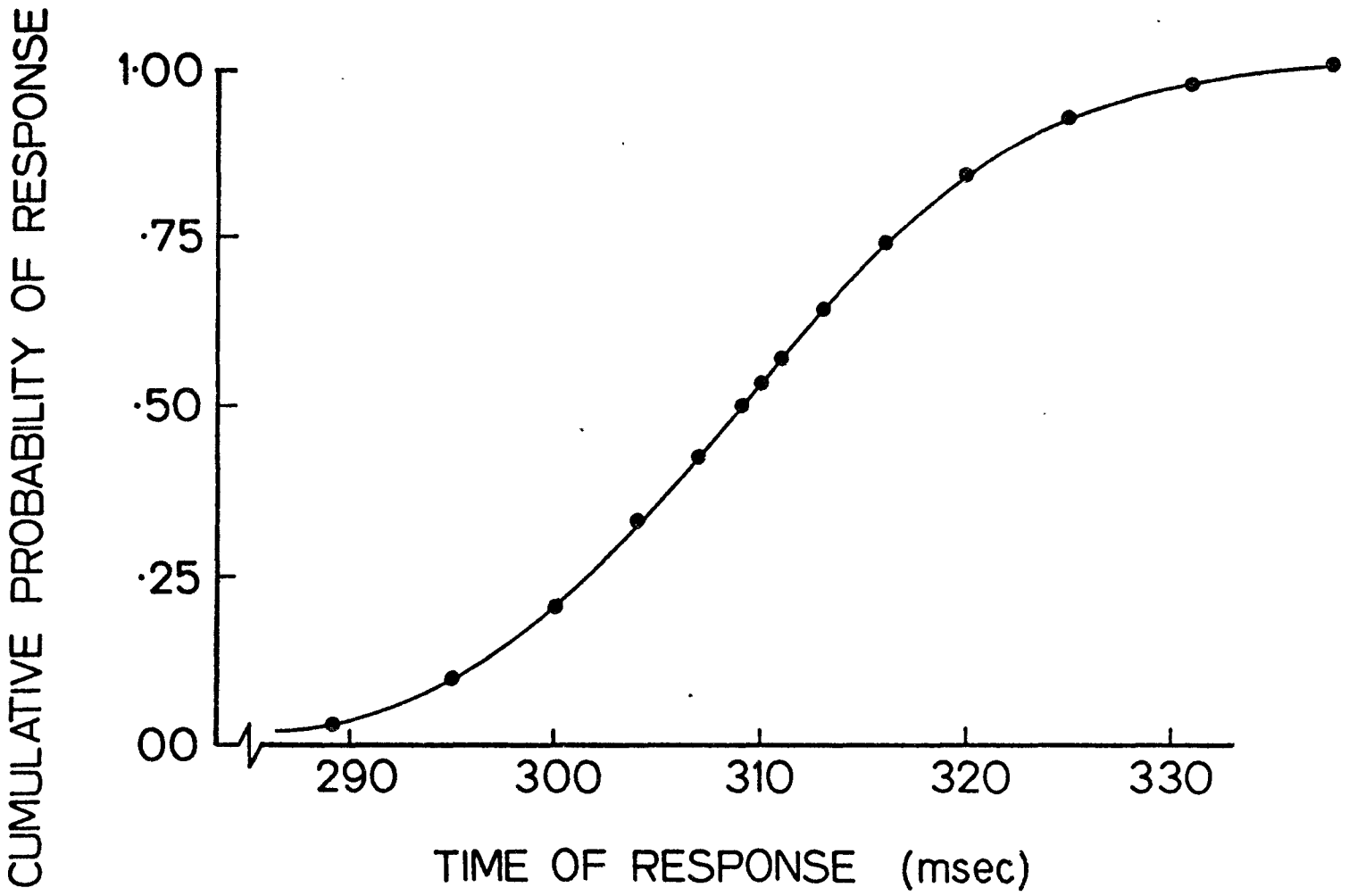
In actual practice, however, a small range of b-values, centred about the calculated value, were used for the purposes of curve fitting.

Figure 34 shows the fit of the model to the data from sessions 26 to 30 for subject G.T. (Chi-square = 6.83 on 13 d.f.). This best fitting curve produced estimates for W and b of 16.6 and 9.12 respectively. The overall response latency variance calculated using these estimates is 129 relative to 125.5 msec^2 obtained from the data. In general, the fit is very good, with no indication of any systematic discrepancies between model and data.

Later in practice, overall response latency variance for subject G.T., on sessions 176 to 180, decreased substantially from 125.5 to 56.8 msec^2. The fit of the model to this more stable data is even more striking (Chi-square = 3.34 on 11 d.f.) as shown in Figure 35. Parameter estimates for W and b are 14.8 and 4.4 msec respectively. These provide an estimate of the overall

Figure 34 - Graph showing goodness-of-fit of model to the data from sessions 26 to 30 for subject G.T. The filled circles are the data points and the line is the psychophysical function predicted by the model. Parameter values are given in the text. P1P2 interval is 310 msec.

Figure 35 - Graph showing goodness-of-fit of model to the data from sessions 176 to 180 for subject G.T. The filled circles are the data points and the line is the psychophysical function predicted by the model. Parameter values are given in the text. P1P2 interval is 310 msec.


CUMULATIVE PROBABILITY OF RESPONSE
1·00
·75
·50
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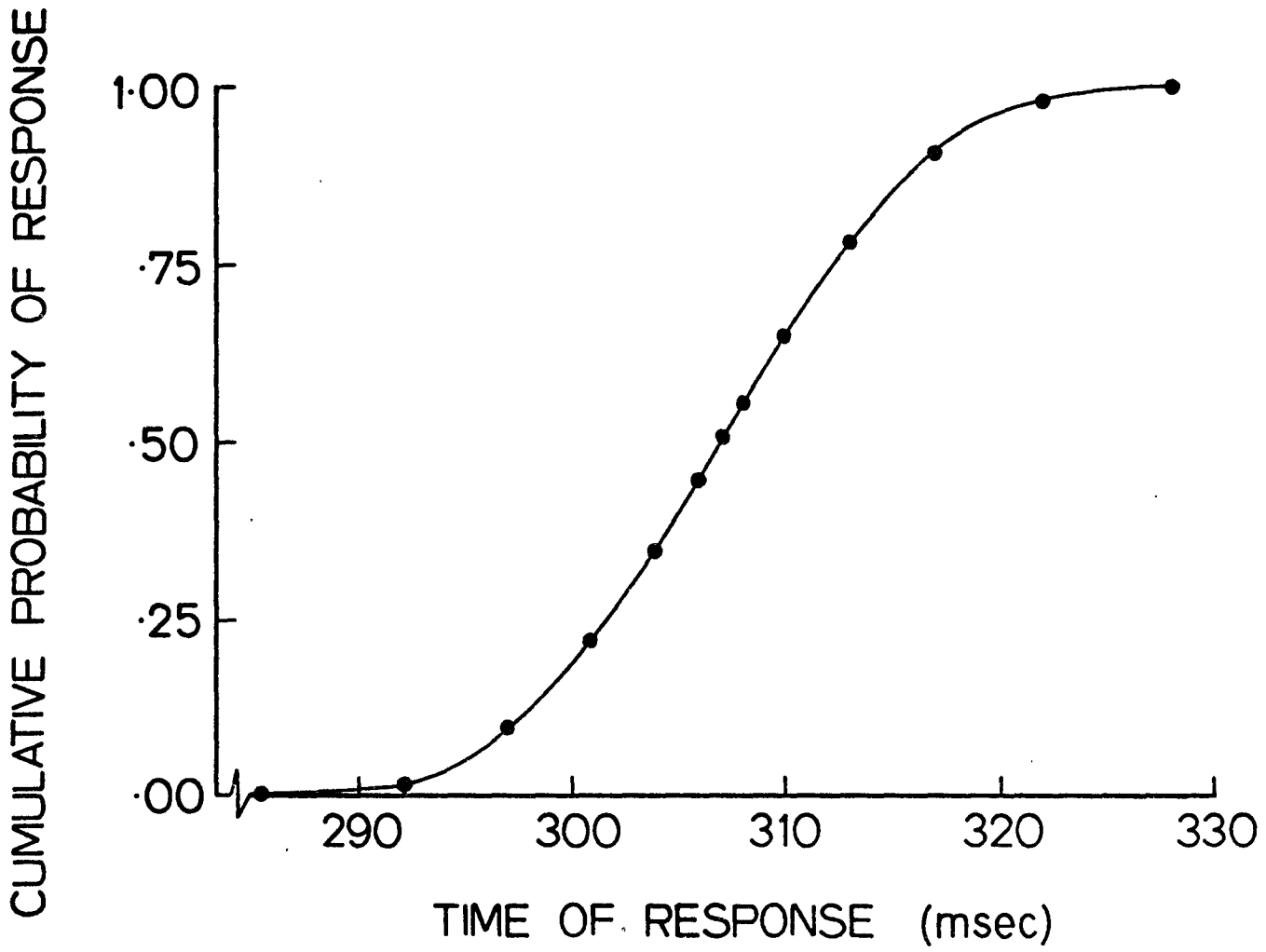
00
290
300
310
320
330
TIME OF RESPONSE (msec)


CUMULATIVE PROBABILITY OF RESPONSE
1·00
·75
·50
·25
·00
290
300
310
320
330
TIME OF RESPONSE (msec)

variance of 55.9 msec^2 which is very close to the obtained value.

As practice continues and the total variance decreases to near asymptotic levels, the model still provides a very good representation of the data, as shown in Figure 36 for subject G.H. on sessions 266 to 270 (Chi-square = 4.81 on 9 d.f.). In this case, the estimates for **W** and **b** are 11.4 and 3.6 msec respectively. This produces an estimate of overall variance of 34.7 relative to 34.6 msec^2 obtained from the data.

The final graph in this series, Figure 37, shows the best fitting curve to data obtained for subject G.H. by combining the five best session performances from sessions numbered 376 to 395. Not only does the model describe the data exceedingly well (Chi-square = 4.11 on 9 d.f.), but the plot also serves to graphically emphasize the amazing accuracy with which humans can perform response-stimulus synchronization. Mean response latency is only 0.3 msec longer than the **P1P2** interval (460 msec) to be synchronized with and the spread of the distribution of response latencies is small with 50% of all responses falling within a 7.3 msec time window. Expanding the time window to 18.7 msec accounts for over 90% of the responses and, in fact, all of the responses in this analysis fall within ±16 msec of the mean. The estimates for **W** and **b** for this set of data are 9.1 and 4.18 respectively. These values provide an

estimate of overall variance of 31.3 msec^2 which is exactly what was obtained from the data.

F. Accuracy of Parameter Estimates

It is evident from the preceding series of analyses that **W** and **b** do not always represent the same proportion of the total variance. For example, during sessions 26 to 30, for subject G.T., variance attributed to **W** accounted for only 36% of the total variance whereas during sessions 176 to 180 its contribution to the total variance rose to 65%. If the actual **W**- and **b**-values are examined, it is clear that most of the reduction in overall variance results from a substantial decrease in the value of **b** while **W**-values decrease more slowly. Similar trends were found across subjects tested. This suggests that fine tuning of the response system, to minimize efferent latency variance, may be responsible for much of the improvement observed during the first couple of hundred sessions. After this level of practice, the value of **b** seems to stabilize while **W**-values continue to decrease slowly, indicating increasing efficiency in the central processing of the relevant information. This finding, however, was not as clear-cut across individuals. As a result, this notion should be considered highly speculative. Other findings were even more equivocal.

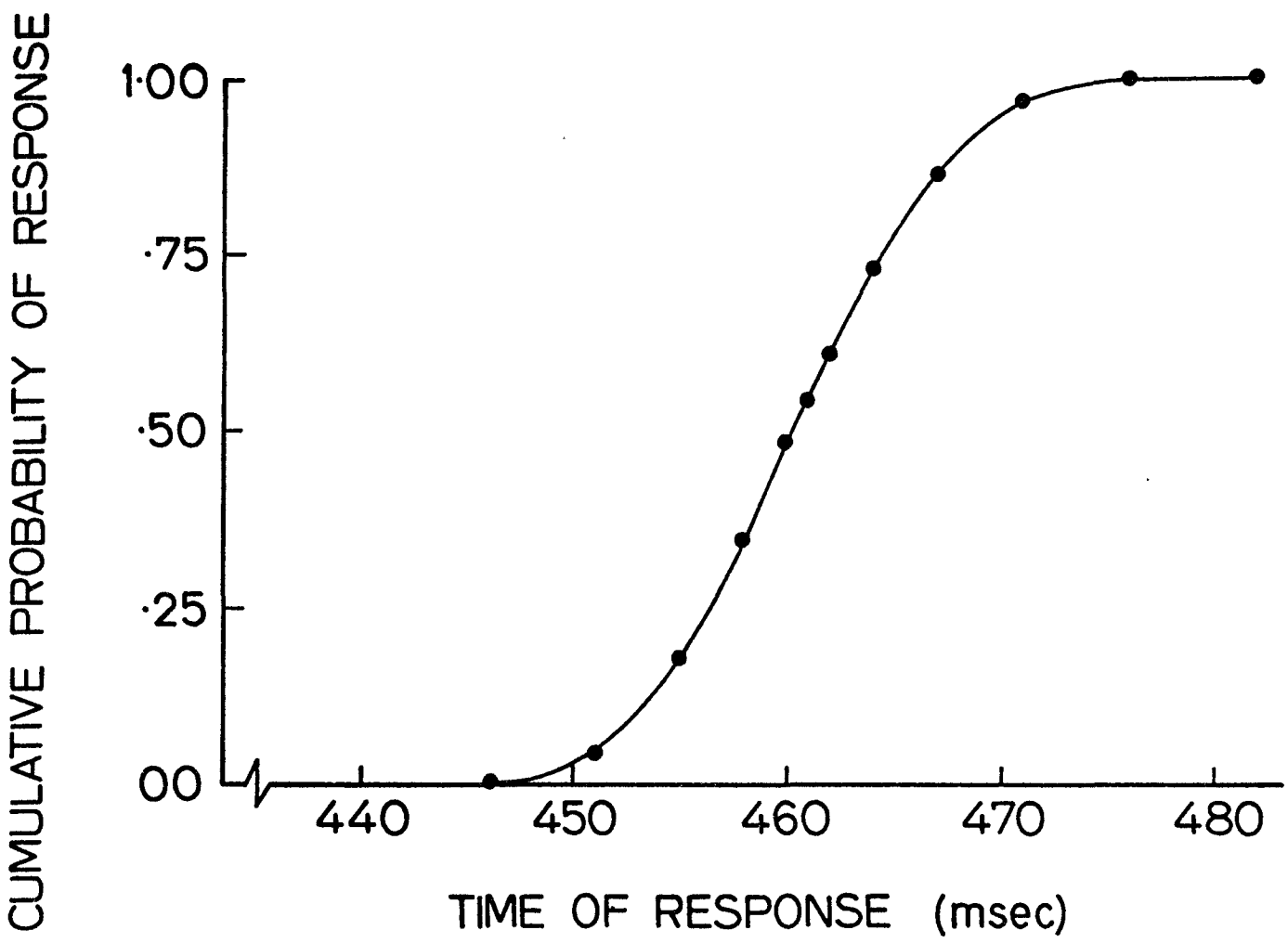
Ideally, parameter estimates derived from the various sets of data gathered in this series of experiments could expand our understanding of what stages in processing are most affected by the various manipulations that were employed. Unfortunately, the procedure used for estimating the parameters lacks the power to provide such an ideal condition. In spite of the fact that the data are remarkably stable and the model provides an excellent fit, there are actually several sets of values for W and b which provide, for a single set of data, fits which are indistinguishable. This situation makes it difficult, if not impossible, to draw any firm conclusions as to which variance component is being most affected by a particular manipulation.

An example of the problem encountered is shown in Figure 38. This graph was plotted from the parameter estimation analysis conducted on the data from sessions 176 to 180 for subject G.T. (see Figure 35). It shows how Chi-square values vary as a function of various combinations of W- and b-values. As a reminder, the best fit to this data (minimum Chi-square) occurred when W and b had values of 14.8 and 4.4 respectively. But, it is clear from Figure 38 that near optimal fits to the data can be obtained with b-values ranging from 3 to 5.5 msec, and even beyond. Such changes in b produce large changes in the relative proportion of total variance that is attributed to each of

Figure 36 - Graph showing goodness-of-fit of model to the data from sessions 266 to 270 for subject G.H. The filled circles are the data points and the line is the psychophysical function predicted by the model. Parameter values are given in the text. P1P2 interval is 460 msec.

Figure 37 - Goodness-of-fit of model to the data presented in Figure 33 for subject G.H. The filled circles are the data points and the line is the psychophysical function predicted by the model. P1P2 interval is 460 msec. Dotted lines indicate the quartile response times.

Figure 38 - Graph showing lack of power of minimizing Chi-square technique. Solid lines represent Chi-square values obtained with various values of **W** and **b** (standard deviation of logistic). Dotted line is the envelope encompassing minimum Chi-square values. The analysis was applied to the data from sessions 176 to 180 for subject G.T.


CUMULATIVE PROBABILITY OF RESPONSE
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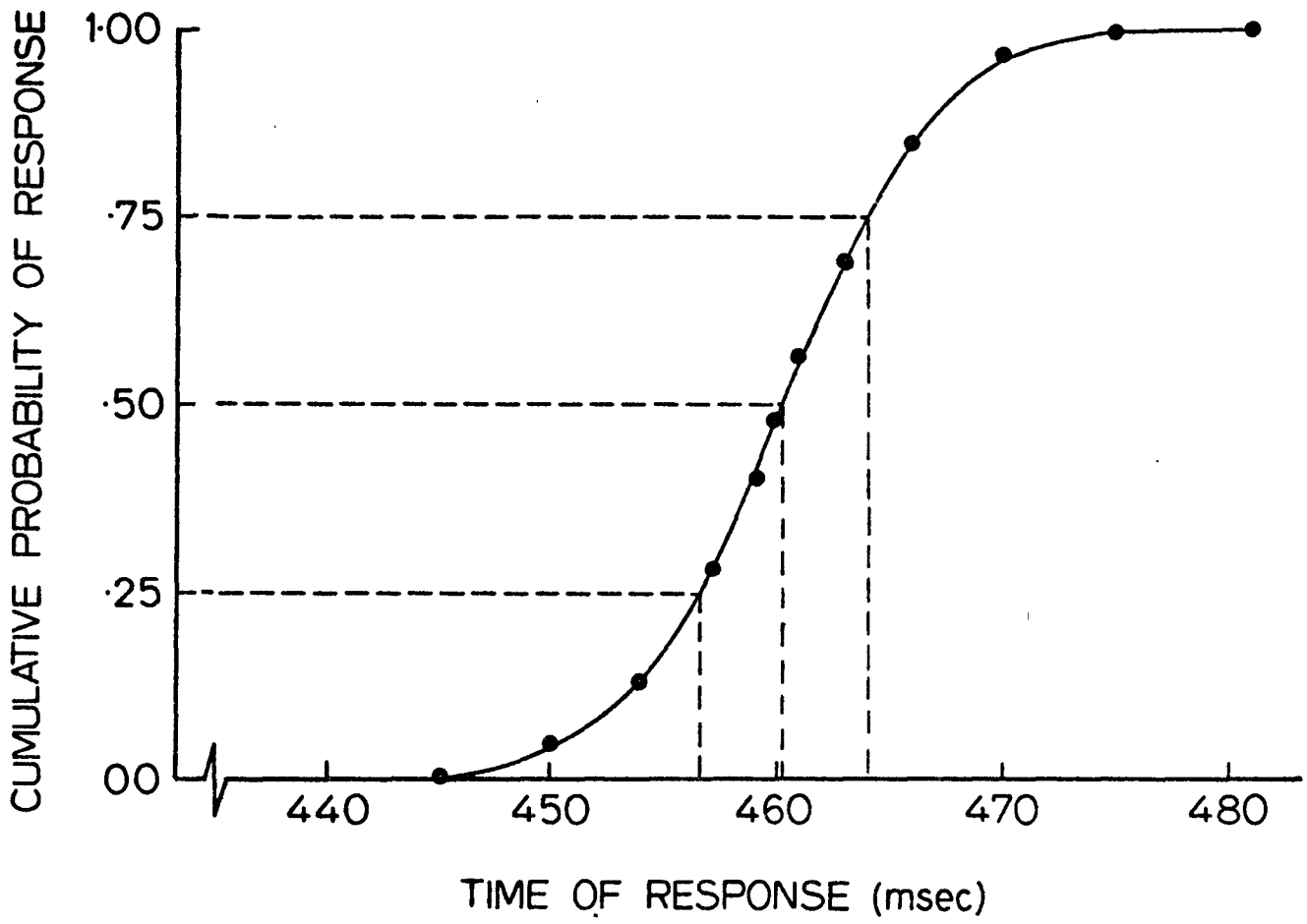
460
470
480
TIME OF RESPONSE (msec)


CUMULATIVE PROBABILITY OF RESPONSE
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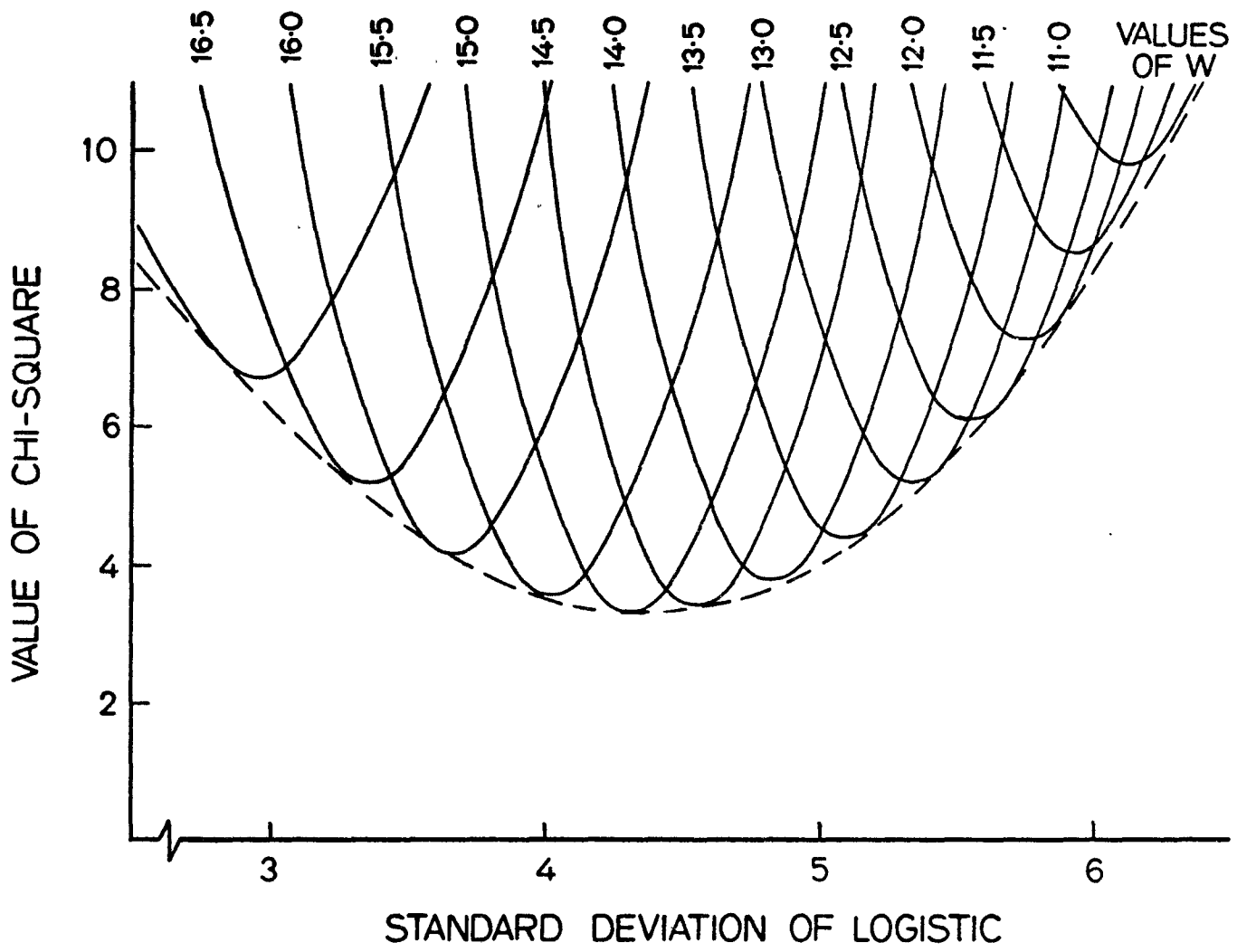
TIME OF RESPONSE (msec)


16·5
16·0
15·5
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14·5
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13·5
13·0
12·5
12·0
11·5
11·0
VALUES OF W
VALUE OF CHI-SQUARE
10
8
6
4
2
3
4
5
6
STANDARD DEVIATION OF LOGISTIC

the component stochastic processes, making theorizing about the effects of the various manipulations highly risky. Most of this problem stems from the great similarity between triangular and normal distributions. Consequently, any "noise" in the obtained response latency distributions greatly decreases the confidence associated with the parameter estimates derived from this data. As a result, no estimates of **W** and **b** were attempted with the reaction time data because it was relatively "noisy" and not much data was available for analysis.

In spite of "noise" problems, it is interesting to note that the estimates of **b** that were calculated, where $\mathbf{b}^2$ represents the efferent delay variance, agree well with those estimates provided by Wing and Kristofferson (1973b). The actual values for four well-practiced subjects are presented in Table 12. It is clear that the value of $\mathbf{b}^2$ decreases with practice, but the minimum level, near 10 $msec^2$, is exactly the same as that estimated by Wing and Kristofferson and the range of values is similar also.

Out of the many parameter estimation analyses conducted, only the one finding mentioned earlier was consistent. That is, the variance of the efferent latency component, estimated by the parameter **b**, tends to contribute proportionately less to the total variance as a function of practice. In terms of the response latency distribution, it means that the shape should become more triangular as

Table 12

Estimates of b^2 for Several Well-Practiced Subjects Synchronizing Under Baseline Conditions.

Subject	Sessions	Variance	b^2
J.B.	121-125	84.7	35.5
C.H.	170-174	72.8	33.4
G.T.	176-180	56.8	19.4
G.H.	386-390	40.6	10.2

Note. b^2 refers to the estimate of efferent delay variance derived from the model. The variance column in the table is mean within-block variance.

practice continues and, to some extent, this can be seen in the data. Table 13 presents a shape parameter analysis for several response latency distributions obtained in Experiments 1 and 4. The coefficient of kurtosis for a normal distribution is 3.0 and for a triangular distribution the value is 2.4. Thus, the trend towards a reduction in the coefficient of kurtosis, as shown in Table 13 is consistent with the notion of a transition in shape from normal to triangular. This information, however, should only be taken as corroborating evidence because the change in coefficient values is rather small and inconsistent in a few instances.

The other information provided in Table 13 is the coefficient of skew. This statistic quantifies the symmetry of the response latency distributions. It may be noted that all values are close to zero, indicating almost perfect symmetry. The average of all tabled values is .025. This finding supports the assumption of symmetry made in formulating the model and also justifies the use of mean and variance as the primary descriptive statistics used throughout the experiments.

Table 13

Shape Parameter Analysis of Response Latency Distributions Obtained in Experiments 1 and 4.

Subject	Sessions	Skew Coeff.	Kurtosis Coeff.
G.H.	26- 30	-.046	3.025
	96-100	.006	3.073
	176-180	-.048	2.997
	206-210	-.006	2.926
	266-270	.140	2.916
	386-390	-.003	2.865
G.T.	26- 30	.117	3.069
	131-135	.200	3.292
	176-180	-.081	2.954
C.H.	31- 35	.069	3.329
	121-125	-.069	2.824
	170-174	.024	2.875
J.B.	31- 35	.086	3.185
	121-125	-.039	3.295

Note. All sessions tabled were obtained under baseline (P1P2) conditions. Average skew for all sets of data combined is .025.

G. Alternatives to the Model

As mentioned earlier, there are two requirements of a good model: (1) being able to provide a good mathematical representation of the data; and (2) having parameters with psychological relevance. The model described in the preceding discussions fulfills both of these requirements. In searching for alternatives to this model, several different distributions were entertained. Of these, however, only three were found which reasonably fulfilled the first requirement of accurate representation of the data, but none fulfilled the second requirement very well, when compared with the proposed model.

The three alternatives that were found to fit the response-stimulus synchronization data best include the normal, logistic, and Tikhonov distribution. (The latter is a specialized distribution borrowed from electrical engineering). Other distributions that were considered, but rejected because of their relative inaccuracy in characterizing the data, included some that have been previously proposed in temporal information processing research: the ordinary gamma distribution (McGill, 1963), the general Erlang or generalized gamma (McGill & Gibbon, 1965), the Poisson (Creelman, 1962), the negative binomial (LaBerge, 1962), and the double monomial, formed by putting two power functions back to back (Luce & Galanter, 1963).

Comparisons of how well some of these distributions represent the data, compared to the proposed model, are shown in Figures 39 and 40. In both cases, the ranking of the four distributions, in terms of their goodness-of-fit, is the same.

The statistic used for computing this ranking consists of subdividing the data into 3 msec bins and simply calculating the deviation between the observed relative frequency, in each bin, and that predicted by a particular distribution. These deviations are then added together for each distribution and used as the basis of comparison. Of the four tested, the logistic is the least successful in representing the data. Deviations totalled .095 and .091 when the logistic was fit to the data for subjects G.T. and G.H., respectively.

The other three distributions fit the data much better, but the proposed model still provides the best representation, followed by the normal, and then the Tikhonov. This ranking was the same for both subjects. Mean total deviation for the proposed model is .038, relative to .041 for the normal and .042 for the Tikhonov. It is obvious that the differences between these three distributions is minimal when the proper parameters are selected. This similarity can be seen in Figure 41 which plots the probability density for each of the distributions tested, using the parameters that provided the best fits to

the data for subject G.T. All, except for the logistic, are nearly superimposed. Thus, based on the first requirement of good representation of the data, it would be difficult to select among these distributions for purposes of modelling.

Consideration of the second requirement of a model, however, of psychological relevance allows the alternatives to be rejected in favour of the model proposed. For purposes of this discussion, the logistic will not even be considered because of its relatively poor fit to the data. In addition, the logic behind its rejection is analogous to that to be discussed for the normal.

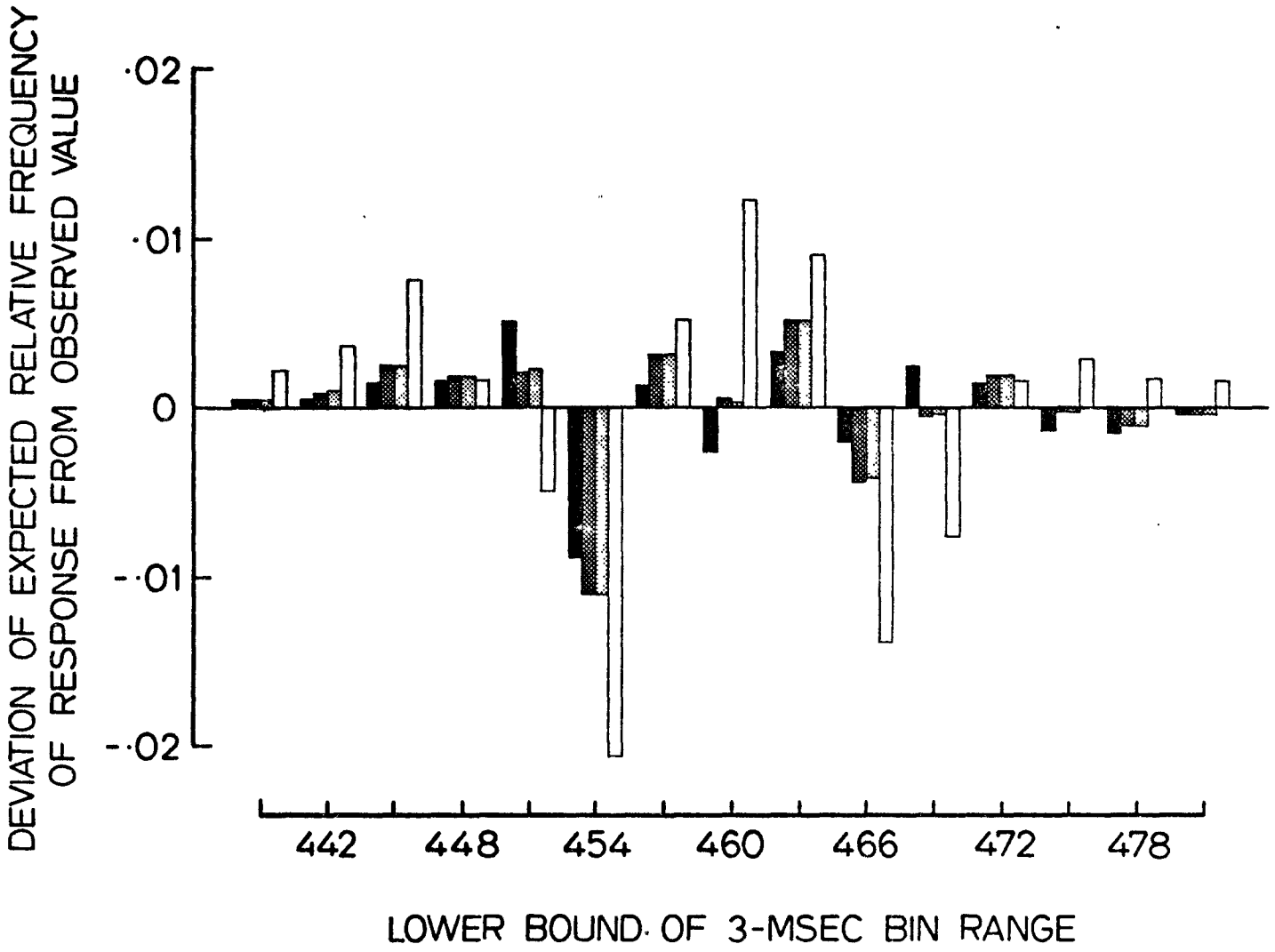
There are a few ways in which a model of response-stimulus synchronization could be formulated, based on a normal distribution, but none provides a more complete theory about the underlying mechanisms and how they manifest themselves in the data than that provided by the model proposed. First of all, the model must account for the independence that exists between the mean and variance of the response latency distributions obtained at different P1P2 intervals. This rules out almost any type of notion that postulates counting pulses from some internal, biological clock that is stochastic in nature simply because larger counts (increased mean) would also produce more variance.

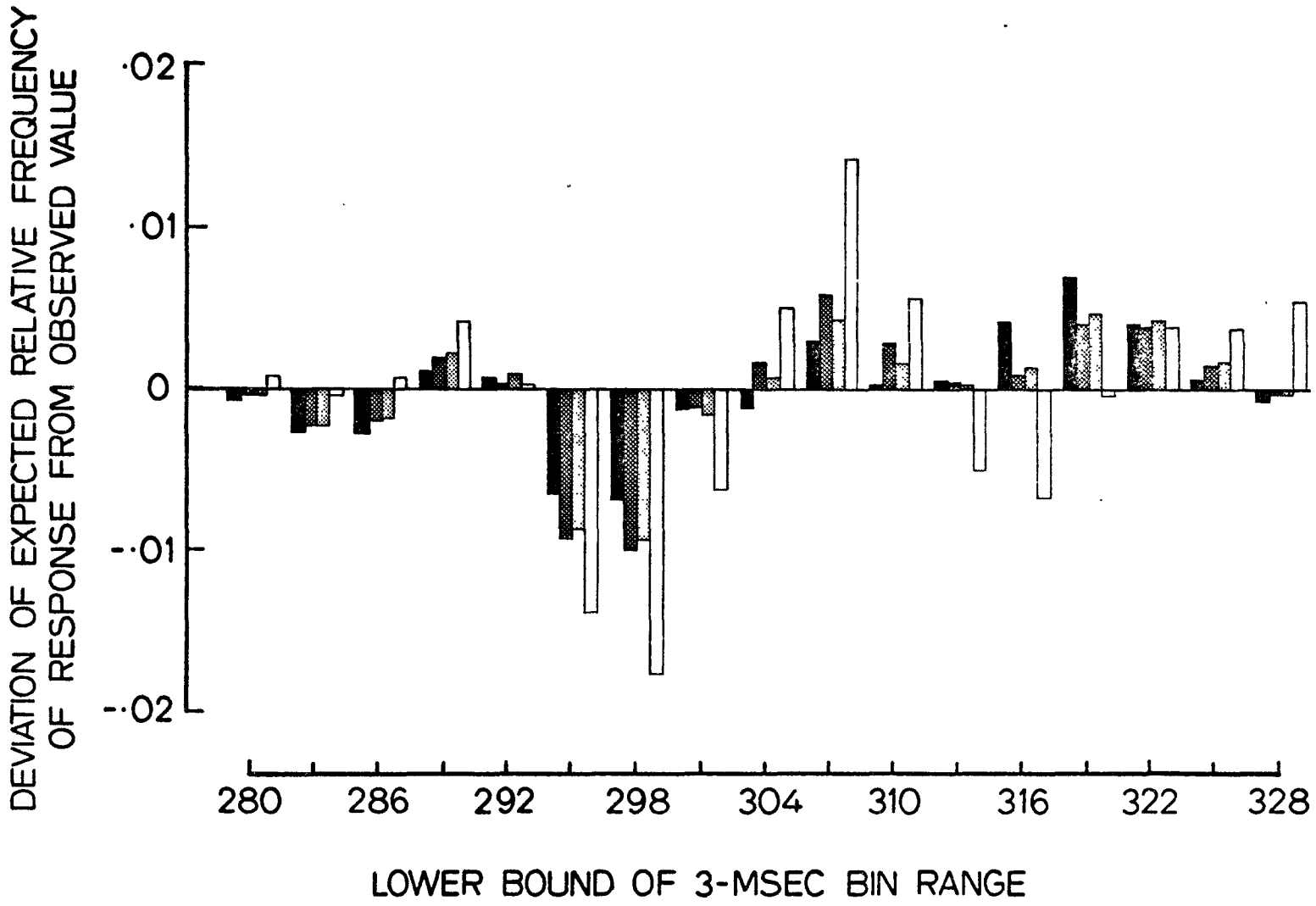
The only way in which such a notion could be entertained is to assume a complex correlation exists

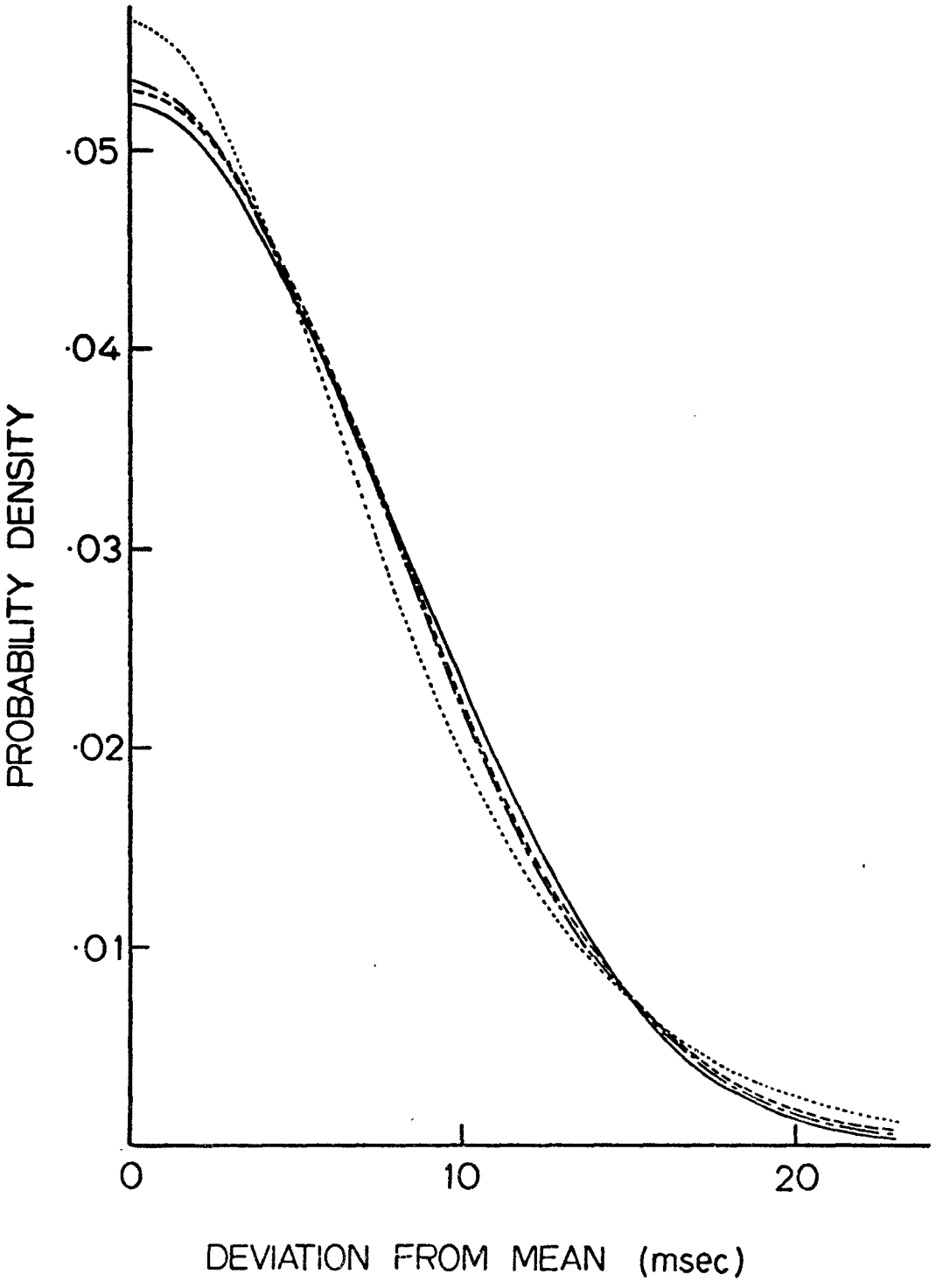
Figure 39 - Comparison of different models in terms of their ability to quantitatively represent the data from sessions 266 to 270 for subject G.H.. Black bars are for the proposed model, dark gray bars are for the Tikhonov distribution, light gray bars are for the normal distribution, and white bars are for the logistic distribution.

Figure 40 - Comparison of different models in terms of their ability to quantitatively represent the data from sessions 176 to 180 for subject G.T.. Black bars are for the proposed model, dark gray bars are for the Tikhonov distribution, light gray bars are for the normal distribution, and white bars are for the logistic distribution.

Figure 41 - Graph showing the similarities of four different density distributions that were tested for goodness-of-fit to the data from sessions 176 to 180 for subject G.T. Solid line is the proposed model, broken line is the Tikhonov distribution, dashed line is the normal distribution, and the dotted line is the logistic distribution.


DEVIATION OF EXPECTED RELATIVE FREQUENCY OF RESPONSE FROM OBSERVED VALUE
·02
·01
0
-·01
-·02
442
448
454
460
466
472
478
LOWER BOUND OF 3-MSEC BIN RANGE


DEVIATION OF EXPECTED RELATIVE FREQUENCY OF RESPONSE FROM OBSERVED VALUE
·02
·01
0
-·01
-·02
280
286
292
298
304
310
316
322
328
LOWER BOUND OF 3-MSEC BIN RANGE


·05
·04
·03
·02
·01
PROBABILITY DENSITY
0
10
20
DEVIATION FROM MEAN (msec)

between the central and efferent stages of processing which changes as a function of the central delay required. In other words, one could propose a system in which as the central delay increased it became more negatively correlated with the efferent delay. Thus, any increase in variance, caused by increasing the central delay component, could be exactly compensated for by a correspondingly higher negative correlation with the efferent delay stage. This could produce what would appear to be independence between mean and variance in the data, however, there is no evidence for the existence of such a relationship that the author is aware of. Moreover, the estimates of efferent delay variance provided by several independent measurements in the literature indicate that the variability in this processing stage is simply not sufficiently large enough to compensate for any type of linear increase in the ratio of standard deviation to the mean (Weber ratio) over the range of P1P2 intervals that produce equi-variance performance.

Another possibility for use of the normal is to substitute normal distributions for the two uniforms in the model presented earlier. This substitution provides a model that predicts independence of mean and variance, however, it has two drawbacks relative to the proposed model. First, the use of normal distributions, for each of the theoretical, stochastic components, produces a response latency distribution which is also normal with a fixed

coefficient of kurtosis. In other words, the shape of the distribution is the same regardless of the relative contributions of each of the variable components. This is inconsistent with the data. The analysis presented in Table 13 indicates that the shape of the response latency distributions changes as a function of practice. Coefficients of kurtosis generally decrease.

Secondly, it is difficult to postulate a mechanism for generating a normal distribution of information transfer delays. Furthermore, such a notion, in which each variance component is assumed normally distributed, is less theoretically appealing because it does not allow for deconvolution of the response latency distribution into its component processes. Finally, this idea ignores the growing literature indicating the involvement of at least quasi-quantal mechanisms in the processing of temporal information (e.g., Kristofferson, 1980). Thus, based on both theoretical and quantitative grounds, an alternative model characterized by a normal distribution must be tentatively rejected in favour of the proposed model.

This leaves only the model characterized by the Tikhonov distribution to be considered. Some background information will help to decide whether the parameters of this model have any psychological relevance to stimulus-response timing mechanisms in the human central nervous system. In electrical engineering, the Tikhonov

distribution is used to represent the distribution of phase errors accruing during steady-state performance of a phase-locked loop circuit. This circuit is simply an electronic servo-loop that is designed to synchronize an internal oscillator with an incoming signal. Any phase changes, indicating changes in the incoming frequency, are detected and cause the internal oscillator to change accordingly, thereby maintaining a "locked" condition (Lindsey, 1972). Without going into all the mathematics of the model, the equation for the Tikhonov in an unbiased, steady-state condition is:

$$f(\phi) = \exp(\alpha\cos\phi)/(2pi I_0(\alpha))$$

where α is a parameter representing the signal-to-noise ratio or sensitivity of the circuit and ϕ is a random variable taking on values from $-pi$ to $+pi$. The term $I_0(\alpha)$ represents the value of the zero-order Bessel function for α. The frequency of the signal is related to pi. As the frequency to be synchronized with decreases, the amount of time encompassed in the range from $-pi$ to $+pi$ increases. Thus, by decreasing frequency, to spread the distribution, while simultaneously increasing α, to compress the density of the distribution, it is possible to get a whole family of different shaped distributions with the bulk of their density distributed within a relatively constant time window.

In testing for goodness-of-fit with this model, both frequency, which controls the amount of time spanned by *pi*, and α were allowed to covary. The best fits were always obtained with very low frequency values (below 0.2 Hz) paired with very high α values.

Although accurate characterizations of the data could be obtained using this model there are several theoretical problems with applying it to response-stimulus synchronization performance. First of all, the use of extremely low frequencies in the context of synchronization timing does not make any intuitive sense. The lowest frequency that could be reasonably entertained is one in which the P1P2 interval corresponded to one-half wavelength. In this case, the lowest frequency expected with an interval of 460 msec would be about 2.2 Hz. If such a constraint is put on the lower limit for frequency, the difference in goodness-of-fit is rather minor but it does give a greater edge to the fit provided by the proposed model.

Another problem arises in analyzing the psychological meaning of changing α values. If one assumes that the frequency must be related in some manner to the P1P2 interval then it is necessary for α (signal-to-noise ratio) to increase as the synchronization interval increases in order to maintain equi-variance distributions. The rationale for such a relationship is not intuitively obvious but one could propose an explanation based on some kind of

psychological refractory period which operates to reduce efficiency in the system when two stimuli occur in close temporal succession. The major problem with this model, however, stems from the fact that it is based on a continuous periodic process whereas in the current context it is being applied to discrete behaviour. It is clear, especially in the self-paced procedure used throughout this series of experiments, that P1 onsets, from trial to trial, are not occurring with any regular periodicity. Such a violation of a central assumption of this model rules out the Tikhonov distribution as a theoretically valid alternative to the model proposed.

H. Theoretical Extensions to the Synchronization Model

In the earlier discussion, during formulation of the model, the theory surrounding the model was presented in a simplified form because the discussion assumed the central processor to be in a steady-state condition. In actual practice, however, this assumption is probably violated because the state of the central stage, which governs timing of the response trigger, slowly changes over time, being affected by other cognitive activity and changes in the physiological state of the organism. Thus, internal conditions should be viewed as if they were in a state of continual flux. This necessitates a dynamic process for fine tuning, or updating, the central timing stage based on

feedback about recent successes and/or failures in synchronization.

The result of this continual updating of the system introduces a new source of variance that was not considered earlier. As the internal state of the organism changes, timing will be affected in such a way that the response triggers will begin to occur too early or too late on the average. This information, provided by the feedback available, allows the central information processor stage to alter the timing process accordingly in order to maintain accurate synchronization.

Obviously, the accuracy of such a feedback loop is determined, in part, by the amount of information that is considered in the determination of the extent of alterations to be made to the central timing stage. If only the information from the preceding trial were considered it would not be very reliable. For example, production of a relatively short response latency may simply result from the combination of several short delays required to get through each of the stochastic components in the stimulus-response chain. This information provides almost no indication of what the mean response latency is. On the other hand, if the feedback information from several preceding trials is considered and it indicates that response latencies tend to be a little short, on the average, then this provides much more reliable information that some slight adjustment should

be made to increase the timed delay. Consequently, the amount of variance that will be added into the observed response latency distribution will depend on the extent to which the internal state of the organism varies during a session and on how much feedback information is integrated in the process of updating the internal system to maintain synchronization with the external stimulation.

In the chapter on feedback, it was clear from the sequential dependency analyses that averaging of the feedback information does occur in response-stimulus synchronization. Those analyses also indicated that feedback information became more reliable with practice, suggesting the experienced subject integrates more and more information into the updating decision process. Some of the increased accuracy in the updating process also probably stems from the subject learning to interpret the feedback information better.

What happens without the feedback? Well, the data show that performance deteriorates rapidly and strong sequential dependencies begin to develop. In terms of the model, lack of feedback prevents the updating process from operating and causes the mean response latency to vary over time as a function of changes in the internal state of the organism. This produces a slow wandering of the mean which inflates overall response latency variance and introduces autocorrelations between responses occurring in close

temporal succession. Decrements in performance are probably further enhanced by degradation of the memorial representation of the P1P2 interval. Without feedback, there is no way to refresh the memory for the synchronization interval.

If this interpretation of what causes decrements in performance in the absence of feedback is valid, then use of a response-stimulus synchronization paradigm, in conjunction with a "feedback - no feedback" manipulation might provide some insight into individual differences in central processing variability. It should be recalled that throughout this series of experiments individual differences have been notably lacking except in cases where some aspect of the feedback was manipulated.

When neither P2 nor the delayed feedback pulse were available large differences in performance were observed between subjects G.H. and E.A. For subject G.H., mean error of synchrony increased from near zero to over +30 msec and variance increased almost tenfold. The mean within-block variance was 314.5 with an overall variance of 348.1 $msec^2$. The spread between these two variance measures, combined with the computed autocorrelation coefficients for lags 1 to 10, indicated that the mean response latency drifted relatively quickly over time.

The data for subject E.A. were quite different. His mean error of synchrony did not change much in the absence

of feedback and his response latency distributions were somewhat less variable. Mean within-block variance was 110.0 relative to an overall variance of 302.5 $msec^2$. In this case, the large difference in variance values combined with weaker autocorrelations suggest that internal fluctuations in the state of the organism were occurring at a slower rate for subject E.A. compared to subject G.H.

A similar type of finding was found when P2 was removed from the stimulus sequence. This removal of one of the primary sources of feedback produced a range of effects on performance for the four subjects exposed to this condition. It may be recalled that three of the subjects experienced varying degrees of performance decrement while one subject (G.T.) continued to respond with little change in the distribution of response latencies. When this data was reported, it was suggested that the individual differences that appeared were due to different degrees of reliance on the delayed feedback versus the feedback provided by the presentation of P2. This argument is still sound, but some of the effect may have also been due to different rates and degrees of fluctuation in the internal state of these subjects.

If this idea, that the central delay timer is continually being updated to compensate for changes in the internal state of the organism, is incorporated into the model, then some changes have to be made to the earlier

interpretations of the model's parameters. An updating process based on averaging feedback information over several trials would generate a variance component which is normally distributed. Consequently, in the parameter estimations performed earlier this component of variance would be reflected in the value of the parameter **b**, being combined with the efferent delay variance. This suggests that the relatively large changes that occur in **b**, over the course of practice, may be more due to changes in the efficiency of feedback utilization than to continued improvements in fine tuning the motor system. After all, it is difficult to imagine much room for improving on a simple finger withdrawal response after more than 100,000 responses. At asymptotic levels of performance, however, any variability contributed by the updating process must be considered very small such that the value of **b** can be considered as providing a fairly reliable estimate of efferent delay variance.

To review the process of response-stimulus acquisition, in terms of the model, there appear to be three factors affecting performance which change as a function of practice. First, there are improvements produced by consolidating an efficient motor program which utilizes a maximum number of efferent and muscle fibres in order to reduce any effects of fatigue and minimize temporal uncertainty of the response. This process probably occurs

quite quickly and may, in part, be responsible for the rapid decline in variance over the first 3,000 to 4,000 trials. Its contribution to improvements in performance past this point must be considered minimal. The second factor concerns minimizing the variability attributed to the updating of the central delay mechanism which compensates for variations in the internal state of the organism. As just discussed, this factor appears to be influenced by the quantity and interpretability of feedback information. Such a factor could be responsible for large increases in performance during the initial stages of practice followed by slower improvements over time because discrimination of the feedback information would become progressively more difficult as the size of the synchronization errors decreased. The final factor, related to **W** in the model, appears to undergo the least amount of change. Perhaps these waiting times are actually fixed and do represent true quantal components in the processing of a stimulus-response chain. Alternatively, these delays may also show some change with practice. It is difficult to decide based on the existing data. If they do change somewhat, these changes may be due to improved strategies for gating the flow of information from one stage to another. Whatever the case, these three factors constitute the sources of variance observed in the response latency distributions and, consequently, the sources for improving performance.

To conclude this chapter, it might be worthwhile to restate the strongest pieces of evidence supporting the proposed model. Substantial support comes from the fact that substituting parameter values, estimated from independent areas of temporal information processing research, into the model predicted asymptotic variance levels that were almost identical to the observed values. The model also provided a remarkable mathematical representation of the data which could account for the independence of mean and variance obtained over the range of P1P2 intervals tested. Finally, not only did the model provide a better representation of the data, relative to several alternatives tested, it also was the most theoretically appealing for postulating central mechanisms and processes underlying response-stimulus synchronization behaviour.

VII. Summary and Final Discussion

Before entering into a final discussion of response-stimulus synchronization and making some concluding remarks, it may be useful to briefly summarize the empirical and theoretical information presented in the preceding chapters. This summary will include a review of the several concerns responsible for stimulating the research, the main objectives, and the major empirical and theoretical findings.

A. Main Objectives of the Research

The current series of experiments was initiated and designed in an attempt to fill in some the gaps in our understanding of response-stimulus synchronization behaviour and how it relates to existing theory about human central temporal mechanisms. Of primary importance in this quest was the development of special procedures for minimizing response latency variances to the lowest possible levels in order to facilitate the drawing of inferences about characteristics of the central timing control of a stimulus-response chain. This modified response-stimulus synchronization procedure provided the opportunity to answer several empirical questions. These included questions related to: (1) comparing performance levels at different anticipation intervals, to gain more support for the notion

of deterministic, central delays; (2) isolating the stimuli governing synchronization performance and measuring the effects created by their removal; (3) ascertaining the relative importance of different types of feedback information; (4) examining the effect of prolonged practice at a single synchronization interval; (5) monitoring the ability of subjects to synchronize with different stimulus modalities and transfer between them; (6) analyzing the nature of sequential dependencies between response latencies, both during and after acquisition; and (7) looking at any important individual differences. Thus, the main objectives were to devise a synchronization task with all of these questions in mind, then train the subjects for long periods to obtain stable data, while carefully measuring effects of stimulus manipulations. The final objective was to relate the obtained results to existing theory, involving deterministic delays, and extend the theory by formulating a mathematical model to describe the data.

B. Major Empirical and Theoretical Findings

The experiments and theoretical discussions associated with them produced many interesting results. The following text is simply a synopsis of the major findings. For all subjects, prolonged practice with a constant synchronization interval was required to reach a stable, near asymptotic

level of performance. At this point in practice, response latency variances were typically under 100 $msec^2$ with the lowest variance levels falling in the 30 to 35 $msec^2$ range. This latter level of performance lowered the upper limit on the estimate of minimum response latency variance by a factor of 3, relative to previous estimates in the literature. It was also an important result because this level of performance had been predicted from independent parameter estimates provided by other studies (Kristofferson, 1980; and Wing & Kristofferson, 1973b) concerned with the processing of temporal information in a stimulus-response chain. Performance was relatively constant across the range of P1P2 intervals tested, providing additional support for the notion that deterministic delays participate in the timing of accurate synchronization responses (Kristofferson, 1976). Not only was performance similar across intervals, it was also similar across subjects. Individual differences were notably lacking throughout. All subjects exhibited a similar rate of acquisition, shape of response latency distribution, and a level of performance that was independent of the particular synchronization interval with which they were training.

The rate of acquisition was well described by a power function which predicted performance after more than 150,000 trials, based on only 12,000 trials, with remarkable

accuracy. However, it should be noted that this relationship seemed to break down at the end of practice for subject G.H. He showed no significant improvement past session 260, which suggests that the estimate of minimum response latency variance provided by the model and the estimates of variance components derived from other studies does, in fact, provide an accurate estimate of the limit of performance in this type of task.

Throughout acquisition, all the response latency distributions observed were distinctly symmetrical, with sharp peaks and short tails. The mean of these distributions quickly centred around the point of veridical synchrony while continued practice served to reduce the spread about this point. Block-to-block and session-to-session changes in mean contributed only minimally to the total variance.

In the absence of P1, subjects were able to quickly transfer timing control of their synchronization response from P1 to the tactile stimulation produced by their initiation response. In some situations this involved an effective lengthening of the synchronization interval of over 90 msec, yet these changes were accommodated quite easily. It should also be recalled that subjects were not aware of the foreperiod and, thus, were not aware of the synchronization interval changing in the absence of P1. These changes were carried out without conscious awareness.

Removal of P2 had devastating effects on performance because it removed a crucial source of feedback information. Under these conditions, individual differences began to emerge, indicating differing degrees of reliance on P2 as a feedback source and suggesting differences in variability of central processing across subjects. The additional removal of the delayed feedback information further degraded performance until response latency variances were higher than those typically obtained in simple reaction time. Not only were variance levels affected by the absence of feedback but errors of synchrony increased dramatically also.

A careful analysis of the effects of feedback indicated that presence of the delayed feedback pulse, which was very salient and easily interpretable, resulted in lower levels of response latency variance throughout acquisition but became redundant information after prolonged periods of practice. Removal of the delayed feedback had no effect on performance after sufficient practice. It was argued that this was because the subjects had learned to interpret the immediate, perceptual feedback information provided by the temporal relationship between P2 onset and the subject's synchronization response. Remember, however, that these results were only obtained with auditory synchronization and not when the interval markers were visually presented.

Sequential dependencies between response latencies were looked at throughout the series of experiments. In general, these analyses indicated the existence of rather strong serial autocorrelations during the early stages of acquisition which totally disappeared after moderate levels of practice. They only reappeared in conditions where the amount of feedback information was insufficient. Consideration of the nature of sequential dependencies and its relation to amount of feedback indicated that modifications to the timing of the stimulus-response chains observed probably involves an averaging process which integrates more and more information with practice, making the subject more proficient.

Response-stimulus synchronization with visual interval markers was significantly inferior to that obtained with auditory markers. In fact, the variability associated with the visual markers was so great that two subjects ended up using a strategy that completely ignored the visual markers and relied instead on their initiation response for timing their synchronization response. This strategy entailed using a time-point that was variable with respect to P2 onset, yet the performance improved. A direct measure of the difference in variance between the processing of an auditory versus a visual stimulus was obtained using a simple reaction time task. The results showed the processing of the visual stimulus to be more variable to

such an extent as to render the strategy change, adopted by two of the subjects, eminently reasonable. It should be recalled, however, that the subjects were unaware of this strategy change. They thought that they were attending to the visual stimuli and synchronizing with them, as instructed.

One could argue that the differences in performance observed between visual and auditory synchronization might simply be due to the fact that visual afferent latencies are longer, but not necessarily more variable. If the integrity of the immediate feedback in auditory synchronization were based on simultaneity of occurrence of the central representation of P2 onset and the tactile stimulation produced by Rs, then the lack of such simultaneity in the visual case, due to delayed registration of P2 onset in this instance, could render this information useless in terms of feedback. However, such an argument ignores the data from Experiment 2 which shows that subjects perform better when they ignore P1 (visual), and instead, rely on the tactile stimulation provided by their initiation response for timing their synchronization response. At this end of the S-R chain, lateness of the central representation of P1 onset should be inconsequential. Moreover, subjects' performances are better, when P1 is ignored, even when *Ri* is not perfectly time-locked to the onset of P1 (time-point where objective measurement of the response latency is initiated),

such that an additional, experimentally induced component of variance is added into the response latency measurements. Thus, the most reasonable interpretation for the strategy of ignoring P1 onset for synchronization timing is to conclude that the variability of the central registration of P1 onset is greater than that induced by having a variable foreperiod and relying on R*i* for timing control.

Deletion of the delayed feedback during visual synchronization had disasterous results because it removed the only accurate source of feedback. Processing of the onset of the visual stimulus P2 was too variable to provide the same reliable source of immediate, perceptual feedback as that provided by P2 during auditory synchronization.

The various stimulus manipulations indicated that most of the improvement in performance with practice is due to fine tuning of the response system to minimize efferent delay variance, learning how to interpret the available feedback information accurately, and acquiring the ability to integrate feedback over several trials in order to provide reliable information for updating the central information processor. It appeared that changes in the accuracy of generating central delays and in the processing of the interval markers contributed little to improving response-stimulus synchronization performance. Input modality of the interval markers, however, was an important consideration because it determined, to some extent, the

consistency of the feedback information the subject was trying to learn how to interpret.

With all the empirical data in mind, a mathematical model of response-stimulus synchronization behaviour was formulated which provided an excellent representation of the data and prompted considerable theorizing about the central and peripheral mechanisms underlying the behaviour. The model consisted of a deterministic delay timer, constant afferent delay, two stochastic waiting times (uniformly distributed), and an efferent delay variance which was assumed to be normally distributed. Combining the probabilistic components produced a theoretical distribution of response latencies that provided a better representation of the data then several alternative models tested. The model's psychological relevance was the most appealing also. It provided parameter estimates of central and efferent delay components that were quite consistent with the literature.

This concludes the summary of the current research. The next section seeks to extend the integration of theory generated by this series of experiments with current thinking about response-stimulus synchronization and other aspects of temporal information processing.

C. Final Theoretical Discussion

The proposed model of response-stimulus synchronization incorporated deterministic delays to account for the independence observed between mean and variance over the range of synchronization intervals tested. Such a notion is not new, but it is contrary to the common view that the nervous system is probabilistic in nature. Previous evidence in support of the idea of deterministic delays has mainly come from duration discrimination studies (e.g., Allan & Kristofferson, 1974; Kristofferson, 1977, 1980) and an earlier response-stimulus synchronization experiment (Kristofferson, 1976).

Although there is strong evidence for the existence of these delays, which are adjustable and can be inserted into a stimulus-response chain without adding any variability, the mechanism by which the nervous system is capable of generating such delays is unknown. Moreover, it is not clear how such precise delays enter into the processing of more complex, stimulus-response control of behaviour. What is clear, is that in certain situations the processing of temporal information is fundamentally different from the processing of other sensory information. For example, Allan (1979) noted that manipulation of the interstimulus interval between the standard and comparison, in a duration discrimination task, did not affect performance in any way, whereas manipulation over the same

range of values with other psychophysical discrimination tasks produces rather substantial effects. In addition, single-stimulus and forced-choice procedures produced comparable duration discrimination performance whereas, in general, these procedures produce quite different levels of discriminability. Findings such as these only serve to emphasize the importance that should be placed on understanding these differences more fully.

Another difference that deserves more attention in the literature is that between auditory and visual information processing. Several studies conducted by Goldstone and his colleagues (e.g., Goldstone & Goldfarb, 1963) have tried to elucidate this intersensory difference and have proposed several suggestions as to why the difference exists but more information is still needed. Their proposals centre around the idea that the processing of visual information is carried out at a higher, more complex and abstract level of processing than is that for auditory stimulation. This difference in level of processing is thought to produce several consequences. Those suggested include: (1) more time is required for processing, causing longer reaction times; (2) a higher level of complexity makes detection of the stimulus more uncertain; and (3) increased noise, caused by transfer of information through additional steps, reduces the amount of transmitted information making the processing sequence more

variable.

In the discussion of Experiments 2 and 3, some attempt was made at furthering our understanding of the auditory-visual difference in information processing. The data provided a quantitative measure of the added delay involved with processing of a visual stimulus as well as a measure of the additional variability incurred. Estimates of the added variability that were derived from the simple reaction time data were in good agreement with independent estimates provided by Zacks (1973). In synchronization, however, the intersensory differences seemed to be exaggerated. This is probably because the use of visual markers not only increases the temporal uncertainty of the central time-point used for timing triggering of the synchronization response but it also increases the uncertainty of the feedback information. This greatly hampers the subject's ability to learn how to interpret the feedback information accurately.

The fact that such intersensory differences exist should not be totally unexpected. After all, the visual system does seem to be geared primarily to the processing of spatial rather than temporal information while the opposite is true for the auditory system. In vision, information seems to be blurred over time in order to maintain persistance and continuity of visual functioning through eye blinks and saccadic eye movements. On the other hand,

information in the auditory system is maintained in discrete form, with respect to time, allowing us to maintain the intricate patterning of notes heard in a musical score. Due to these differences, it is very important that input modality be taken into careful consideration when theorizing about the central nervous system's ability to process temporal information.

Feedback appears to be another crucial factor to be taken into consideration because it is required to maintain the integrity of the timekeeping process and to effect improvements in performance. It seems clear that many of the discrepancies and inconsistencies in the literature are merely a function of the different amounts of useable feedback information provided within the tasks. Such differences not only account for different levels of asymptotic performance being reported but also to conflicting reports on the nature of individual differences associated with various tasks. Obviously, amount of practice must be equated in any comparisons, but with sufficient feedback there is every indication that individual differences in processing strategies are minimal. All subjects seem to exhibit comparable rates of acquisition and similar shaped response latency distributions.

Several other studies of temporal information processing have manipulated feedback and found that idiosyncratic or subjective aspects of performance were

minimized as the amount of feedback increased. For example, Jamieson and Petrusic (1976) observed that time-order errors associated with duration discrimination disappeared such that the subjects' responses corresponded more closely with objective, "real" time. Penner (1976) found that randomly varying the amplitude and duration of the boundary markers, used to delimit silent intervals in a duration discrimination task, produced a major deterioration of performance. This is because there was no fixed referent for evaluating previous performance. The stimulus context was constantly changing. When these stimulus manipulations were kept constant across a block of trials, however, performance improved because the subject's past behaviour could then be evaluated relative to a consistent stimulus sequence, allowing adaptation to the different stimulus contexts to occur. In general, although nontemporal characteristics of a task can influence perceived duration and introduce systematic relations between subjective and objective time, these effects can be adapted to and eliminated if the proper feedback is provided (Allan, 1979).

One of the most troublesome inconsistencies in the literature concerns the nature of the relationship between mean and variance of responses in various tasks involving temporal information processing. As an example, Getty (1975) reports duration discrimination data which provides rather convincing evidence that the relationship between the

standard deviation of the psychometric function and stimulus duration is a monotonically increasing function that is well fit by a model based on a generalization of Weber's law. On the other hand, Kristofferson (1980) provides equally convincing evidence that the relationship is one of independence between mean and standard deviation over relatively large ranges of duration discrimination intervals, with performance changing in a step-like fashion as the intervals to be discriminated become longer. Is it possible that such discrepant findings could be reconciled by examining the feedback characteristics associated with the respective methodologies? It appears that, to a large extent, the answer is probably yes.

In Getty's two-alternative forced choice procedure, feedback was notably lacking. Subjects received only one session of practice at a particular value of the standard duration and the order of the standards used was randomized across sessions. Moreover, no information concerning the correctness of their responses was provided. In contrast, Kristofferson's procedure did provide feedback and trained the subject sufficiently long at each discrimination interval for the information to be utilized quite effectively. He used a single-stimulus "many-to-few" method (see Allan & Kristofferson, 1974; Kristofferson, 1977) in which the stimulus set consisted of four temporal intervals which differed slightly in duration and were delimited by

two brief auditory stimuli. The subject's task was to decide on a particular trial whether the stimulus presented was one of the two shorter durations or one of the longer ones. After the response, a visual feedback signal was presented to inform the subject about the correctness of his response.

Kristofferson (1980) pointed out that the results obtained during the first few sessions at each base duration were totally consistent with those of Getty (1975). It was only after several sessions of practice that the independent relationship between mean and standard deviation began to emerge. Kristofferson argued that this change occurred because of the extra practice provided at each base duration, however, it seems more likely that the major contributing factor was the amount of feedback provided. Simply extending the amount of practice with each of the standard durations used in Getty's procedure probably would not have made much difference in the results because no feedback was available for the subjects to evaluate and improve their performance. Thus, it is the presence of feedback, in conjunction with sufficient practice for the evaluative information to be utilized, that is important for reducing variance in the psychophysical function.

In fact, on closer examination of Kristofferson's procedure, it may even be possible to account for some of the abrupt changes in variance he observed simply by

analyzing differences in the amount of feedback available as a function of base duration. Feedback simply consisted of a signal indicating whether the stimulus had been long or short but the amount of information conveyed by this signal depended, to some extent, on the distribution of the four durations comprising a stimulus set. The smaller the range of durations encompassed by the two middle members of the stimulus set, the greater the informational value potentially provided by the feedback. For example, assume that a subject generates some criterial interval of time, which determines his discrimination response, and that this criterion is subject to a slow wandering of the mean over time. In such a situation, a 10 msec range between the two central members of a stimulus set will allow detection of any change in the mean criterial interval much sooner than if the range were 100 msec. This means that updates to the timing mechanism governing placement of the criterion can be made more quickly and accurately the smaller the range of stimulus durations. This would also serve to reduce the variance in the psychophysical function. Consequently, one would expect performance to be inversely related to the spread between the two central members of the stimulus sets used. Interestingly, this is exactly what Kristofferson found. The abrupt changes in variance observed occurred at exactly the same points where the spread between the central stimulus durations was increased. It would be very

interesting to see whether these "steps" would disappear if the amount of feedback was held constant across base durations. Under these conditions, it seems reasonable to suggest that at least the steps within the range of intervals employed in the synchronization experiments would disappear. In any event, it is clear that feedback plays a powerful role in determining performance and should be considered carefully in any theorizing about mechanisms underlying temporal information processing.

D. Concluding Remarks

In concluding, it should be remembered that anticipatory timing is not an isolated phenomenon relegated to manifestation only in a laboratory environment. Instead, it must be considered as an essential part of human behaviour because we are constantly confronted with temporal relations to which we must respond appropriately. Whether it be involved with the playing of a musical instrument or a skilled sport, response timing in the context of our environment must be maintained accurate and be dynamically anticipatory in nature.

In studying this somewhat ubiquitous aspect of human behaviour, a number of major contributions to our understanding of this phenomenon have been made. Due to the use of a specialized response-stimulus synchronization procedure, the results revealed several new and interesting

empirical properties of delayed, stimulus-response latencies and, although this information has great value by itself, analyses of these new empirical relations, combined with attempts to explain them, also contributed greatly to the development of a general theory of timing. Quantitative modelling of the synchronization data was also important because it provided insights into the extent and nature of the temporal relations existing between elements of a stimulus-response chain. If one considers the human organism to be an active information processing system, then it is important to understand the characteristics and dynamics of the central processes involved in synchronization behaviour. In this regard, few theories provide a more comprehensive, quantitative description of the empirical observations than the model entertained in this research. Finally, analysis of the role played by feedback in this task provides the promise that many of the inconsistencies in the literature, created as a function of using different procedures, can be reconciled.

The theoretical framework and experimental techniques used in this research should not be considered unique to the area of temporal information processing. Other areas of information processing and the study of skilled behaviour might also benefit from similar methodological considerations; for whenever the processes of the human mind are not directly observable, and one is

forced to rely on inferences, it is important that the methodology maximizes the amount and reliability of the information extracted.

As a final point, it might be worthwhile to reexamine the traditional distinction made between subjective and objective time. Subjective time has often been thought of as a dimension of experience only in which the nature of the activities occurring during a period is the major determinant of the phenomenal duration, rather than the movements of the hands of a clock (Ornstein, 1969). In fact, many investigators, back to the time of William James (1908, Ch. 15), have been intrigued by these alterations in perceived duration produced by varying the physical events generating the subjective experiences. Obviously these are important aspects of cognitive functioning to understand, but it is also intriguing to find out that under some circumstances there is no transformation made between physical time and psychological time. The two are the same. The characteristics of these circumstances, however, have just recently begun to emerge from the synchronization and duration discrimination studies described in this research. Consequently, it is important that this mode of information processing receive further study because presumably it provides the crucial link between our minds and our environment in "real" time.

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Appendix A - Summary of Subjects Employed in Experiments

Experiment	Subject	Age	Sex	P1P2 Interval
1	G.H.	24	M	460
	J.B.	26	F	460
	M.W.	29	M	360
	C.H.	23	F	310
	G.T.	32	M	310
2	G.H.	26	M	460
	E.A.	25	M	360
	V.A.	24	F	360
3	G.H.	27	M	RT
4	G.H.	27	M	460
	J.V.	27	M	310

Note. Subjects for each experiment were all graduate students, except for C.H. (research assistant), who volunteered to participate for the extended periods required. Subject G.H. is the author. All subjects were right handed. They were paid $3.00 per session.

Subject V.A. was dropped because she became very frustrated with her relatively poor performance, lost motivation, and preferred to discontinue her acquisition training.

Appendix B - Tabled Values for Figures Described in Experiments

Number of Responses in Each 5-msec Bin for the Latency Distributions in Figures 7 and 8.

Deviation from Modal Bin (msec)	Subject			
	G.H.	G.T.	J.B.	C.H.
-35			1	
-30			4	
-25	1	6	11	3
-20	9	11	36	29
-15	62	64	98	60
-10	176	192	179	163
-5	293	319	290	294
0	411	373	316	343
5	345	320	290	297
10	142	150	156	180
15	50	55	84	100
20	11	9	23	23
25			8	3
30			2	
35			0	
40			2	
Total	1500	1499	1500	1495

Note. Deviations from modal bins are in terms of time between lower bounds of bins. Lower bounds of modal bins tabled are: G.H.(459), G.T.(306), J.B.(459), C.H.(306). The data were gathered from sessions 121 to 125 for subjects J.B. and C.H. and sessions 176 to 180 for G.H. and G.T.

Appendix B - Cont'd

Number of Responses in Each 3-msec Bin for the Latency Distributions in Figures 9, 28, and 33.

Deviation from Modal Bin (msec)	Subject(Sessions)		
	G.H.(266-270)	G.H.(391-395)	G.H.(5-Best)
-21		1	
-18	2	5	3
-15	9	17	13
-12	37	27	38
-9	93	109	98
-6	198	173	184
-3	261	269	267
0	301	317	329
3	258	260	280
6	188	189	165
9	97	78	80
12	37	36	31
15	13	11	7
18	5	4	4
21	1	1	
24		1	
Total	1500	1498	1499

Note. Deviations from modal bins are in terms of time between lower bounds of bins. Lower bounds of modal bins tabled are 460, 461, and 460 msec respectively. The 5-best distribution combines the data from sessions 392, 394, 400, 401, and 408, obtained under constant foreperiod-P1P2 conditions (no delayed feedback).

Appendix B - Cont'd

Number of Responses in Each 5-msec Bin for the Latency Distributions in Figure 10.

Bin Range (msec)	Subject(Sessions)	
	G.H.(196-200)	G.H.(206-210)
440-444	9	10
445-449	43	62
450-454	151	178
455-459	348	341
460-464	424	437
465-469	348	299
470-474	134	125
475-479	36	41
480-484	4	3
485-489	1	
Total	1498	1496

Note. Sessions 196 to 200 were obtained under P1P2 conditions with a constant foreperiod, while sessions 206-210 were obtained under baseline (P1P2) conditions with a variable foreperiod.

Appendix B - Cont'd

Number of Responses in Each 5-msec Bin for the Latency Distributions in Figure 12.

Deviation from Modal Bin (msec)	Subject(Sessions)		
	C.H.(121-125)	C.H.(131-135)	C.H.(141-145)
-35			1
-30		6	0
-25	3	24	2
-20	29	40	22
-15	60	92	71
-10	163	197	166
-5	294	224	301
0	343	258	363
5	297	225	286
10	180	206	166
15	100	114	80
20	23	78	24
25	3	22	8
30		9	0
35		3	1
40			1
Total	1495	1498	1491

Note. Deviations from modal bins are in terms of distance of the lower bound of a bin from the lower bound of the modal bin. Lower bounds of modal bins tabled are 306, 303, and 307 msec respectively.

Appendix B - Cont'd

Number of Responses in Each 5-msec Bin for the Latency Distributions in Figure 13.

Deviation from Modal Bin (msec)	Subject(Sessions)		
	G.H.(151-155)	G.H.(161-165)	G.H.(176-180)
-35		1	
-30	2	2	
-25	19	12	1
-20	31	32	9
-15	100	78	62
-10	160	173	176
-5	268	276	293
0	305	290	411
5	273	284	345
10	186	188	142
15	91	95	50
20	35	43	11
25	19	18	
30	6	8	
35	1		
Total	1496	1500	1500

Note. Deviations from modal bins are in terms of distance of the lower bound of a bin from the lower bound of the modal bin. Lower bounds of modal bins tabled are 458, 456, and 459 msec respectively.

Appendix B - Cont'd

Lag Means in Each 5-msec Bin for the Sequential Dependency Plots of Figure 14.

Bin Range (msec)	Lag Mean Minus Block Mean			Number of Responses
	Lag 1	Lag 2	Lag 3	
285-289	-0.50	0.77	-1.25	30
290-294	-1.12	-1.14	0.04	78
295-299	-0.75	0.79	1.71	143
300-304	-0.55	-0.31	-0.98	212
305-309	-1.33	-1.33	-1.15	249
310-314	0.50	-0.18	1.00	262
315-319	1.63	-0.25	-0.48	213
320-324	1.42	0.45	0.85	139
325-329	-1.13	1.21	-1.86	64
330-334	0.32	1.89	-0.23	44
335-339	0.48	-0.65	-0.93	22
Chi-Square	7.17	3.44	1.73	

Note. Lag mean refers to the mean on trials $(n+i)$, for lags $i=$ 1, 2, or 3, given the response latency on trial n falls within a particular bin range. The chi-square statistic has 1 degree of freedom. The critical value for significance (p=.05) is 3.84.

Appendix B - Cont'd

Lag Means in Each 5-msec Bin for the Sequential Dependency Plots of Figure 15.

Deviation from Modal Bin (msec)	Subject			
	C.H.		J.B.	
	Lag 1 Mean	# Resps.	Lag 1 Mean	# Resps.
-20	-0.97	29	1.80	36
-15	-0.40	60	-0.05	98
-10	0.30	163	-0.68	179
-5	-0.05	294	-0.64	290
0	0.48	343	-0.12	316
5	-0.64	297	0.90	290
10	0.32	180	0.00	156
15	-0.69	100	0.34	84
20	0.50	23	0.78	23
Chi-Square	0.13		3.69	

Note. Lag mean refers to the mean on trials $(n+i)$, for lags $i=$ 1, 2, or 3, given the response latency on trial n falls within a particular bin range. Deviations from modal bin are in terms of distance of the lower bound of a bin from the lower bound of the modal bin. Lower bounds of modal bins tabled are 306 and 459 msec respectively. The chi-square statistic has 1 degree of freedom. The critical value for significance (p=.05) is 3.84.

Appendix B - Cont'd

Lag Means in Each 5-msec Bin for the Sequential Dependency Plots of Figure 16.

Deviation from Modal Bin (msec)	Subject G.T. Lag 1 Mean	G.T. # Resps.	G.H. Lag 1 Mean	G.H. # Resps.
-18	0.42	11	-0.14	10
-15	1.35	33	-1.83	28
-12	-0.06	82	-0.72	53
-9	-1.19	133	0.41	101
-6	-0.09	177	-0.69	172
-3	0.10	219	0.10	227
0	-0.04	230	0.23	257
3	-0.02	215	0.34	245
6	0.11	171	-0.24	193
9	-0.39	113	-0.39	103
12	0.63	59	0.58	63
15	1.18	26	0.36	28
18	0.64	11	1.01	14
Chi-Square	0.06		0.92	

Note. Lag mean refers to the mean on trials $(n+i)$, for lags i= 1, 2, or 3, given the response latency on trial n falls within a particular bin range. Deviations from modal bin are in terms of distance of the lower bound of a bin from the lower bound of the modal bin. Lower bounds of modal bins tabled are 307 and 460 msec respectively. The chi-square statistic has 1 degree of freedom. The critical value for significance (p=.05) is 3.84.

Appendix B - Cont'd

Number of Responses in Each 5-msec Bin for the Latency Distributions in Figure 21.

Deviation from Modal Bin (msec)	Subject(Sessions)		
	G.H.(286-290)	G.H.(291-295)	G.H.(301-305)
-30	5	1	
-25	13	9	3
-20	37	42	9
-15	94	97	58
-10	177	203	183
-5	262	267	319
0	314	314	406
5	279	259	308
10	191	198	142
15	86	68	61
20	29	34	8
25	10	3	2
30	3	5	
Total	1500	1500	1499

Note. Deviations from modal bins are in terms of distance of the lower bound of a bin from the lower bound of the modal bin. Lower bounds of modal bins tabled are 459, 460, and 460 msec respectively.

Appendix B - Cont'd

Lag Means in Each 5-msec Bin for the Sequential Dependency Plots of Figure 22.

Bin Range (msec)	Lag Mean Minus Block Mean			Number of Responses
	Lag 1	Lag 2	Lag 3	
434-438	-4.48	-0.14	1.16	13
439-443	-4.45	0.15	1.04	37
444-448	0.38	-2.07	-1.23	94
449-453	-0.41	0.10	0.09	177
454-458	-0.60	0.63	-0.06	262
459-463	0.83	-0.52	0.07	314
464-468	0.71	0.49	-0.26	279
469-473	0.74	0.30	0.54	191
474-478	-0.88	0.73	-0.46	86
479-483	-0.10	-1.54	1.21	29
Chi-Square	5.08	1.29	0.53	

Note. Lag mean refers to the mean on trials $(n+i)$, for lags i= 1, 2, or 3, given the response latency on trial n falls within a particular bin range. The chi-square statistic has 1 degree of freedom. The critical value for significance (p=.05) is 3.84.

Appendix B - Cont'd

Number of Responses in Each 5-msec Bin for the Latency Distributions in Figure 23.

Deviation from Modal Bin (msec)	Subject(Sessions)	
	E.A.(131-135)	E.A.(141-145)
-35	1	
-30	10	1
-25	17	7
-20	62	19
-15	113	71
-10	193	155
-5	254	288
0	271	360
5	251	291
10	164	203
15	88	77
20	48	21
25	20	5
30	5	2
35	5	
Total	1500	1500

Note. Deviations from modal bin are in terms of distance of the lower bound of a bin from the lower bound of the modal bin. Lower bounds of modal bins tabled are 361 and 358 msec respectively.

Appendix B - Cont'd

Number of Responses in Each 5-msec Bin for the Latency Distributions in Figure 24.

Deviation from Modal Bin (msec)	Subject(Sessions)		
	E.A.(131-135)	E.A.(161-165)	E.A.(171-175)
-35	1		1
-30	10		2
-25	17	5	17
-20	62	24	38
-15	113	73	76
-10	193	146	158
-5	254	298	246
0	271	347	296
5	251	326	252
10	164	160	196
15	88	86	120
20	48	24	63
25	20	8	26
30	5	1	6
35	5	2	1
40			1
Total	1500	1500	1499

Note. Deviations from modal bins are in terms of distance of the lower bound of a bin from the lower bound of the modal bin. Lower bounds of modal bins tabled are 361, 359, and 358 msec respectively.

Appendix B - Cont'd

Lag Means in Each 5-msec Bin for the Sequential Dependency Plots of Figure 29.

Bin Range (msec)	Lag Mean Minus Block Mean			Number of Responses
	Lag 1	Lag 2	Lag 3	
446-448	-1.39	-1.75	0.67	17
449-451	-0.75	-0.15	0.22	27
452-454	-0.52	-1.01	-0.19	109
455-457	0.00	-0.66	-0.81	171
458-460	0.11	-0.51	0.12	265
461-463	0.25	0.07	0.35	312
464-466	-0.04	0.60	0.54	254
467-469	0.30	0.55	-0.37	189
470-472	0.12	1.18	-0.36	77
473-475	-1.31	1.01	0.53	36
Chi-Square	1.99	5.58	0.38	

Note. Lag mean refers to the mean on trials $(n+i)$, for lags i= 1, 2, or 3, given the response latency on trial n falls within a particular bin range. The chi-square statistic has 1 degree of freedom. The critical value for significance (p=.05) is 3.84.

Appendix B - Cont'd

Lag Means in Each 5-msec Bin for the Sequential Dependency Plots of Figure 30.

Bin Range (msec)	Lag Mean Minus Block Mean			Number of Responses
	Lag 1	Lag 2	Lag 3	
342-351	-6.02	-2.02	-1.35	15
352-361	-2.29	-1.98	-0.34	88
362-371	-0.70	-0.51	-0.70	232
372-381	-0.65	-1.07	-1.03	354
382-391	0.08	0.37	0.37	332
392-401	1.16	1.29	1.74	250
402-411	2.17	1.59	0.05	130
412-421	1.63	1.44	1.41	61
Chi-Square	22.15	21.17	10.12	

Note. Lag mean refers to the mean on trials $(n+i)$, for lags i= 1, 2, or 3, given the response latency on trial n falls within a particular bin range. The chi-square statistic has 1 degree of freedom. The critical value for significance (p=.05) is 3.84.

Appendix B - Cont'd

Lag Means in Each 5-msec Bin for the Sequential Dependency Plots of Figure 31.

Bin Range (msec)	Lag Mean Minus Block Mean			Number of Responses
	Lag 1	Lag 2	Lag 3	
430-439	-30.29	-20.21	-12.14	13
440-449	-23.75	-15.23	-13.23	21
450-459	-22.70	-21.07	-12.73	46
460-469	-16.29	-10.06	-5.72	108
470-479	-6.63	-5.11	-4.25	183
480-489	-3.20	-2.34	-1.84	275
490-499	3.62	2.27	0.34	342
500-509	7.79	6.11	5.21	267
510-519	11.50	9.53	9.93	149
520-529	12.93	10.74	7.59	48
Chi-Square	217.72	137.52	54.03	

Note. Lag mean refers to the mean on trials $(n+i)$, for lags i= 1, 2, or 3, given the response latency on trial n falls within a particular bin range. The chi-square statistic has 1 degree of freedom. The critical value for significance (p=.05) is 3.84.

Appendix C - Tabled Values for Figures in Modelling Section

Parameter Estimation and Goodness-of-Fit of Model to the Data Obtained from Subject G.T. on Sessions 26-30.

x	p(R≤x)	F(x)
282	.0080	.0100
289	.0314	.0370
295	.0975	.1009
300	.2031	.2025
304	.3227	.3185
307	.4229	.4208
309	.4937	.4927
310	.5271	.5290
311	.5645	.5650
313	.6426	.6350
316	.7355	.7312
320	.8370	.8353
325	.9178	.9214
331	.9673	.9721
338	.9933	.9926

Note. The symbol **R** refers to the time of response and **x** is the upper limit of integration for the distribution function. The best estimate of the maximum waiting time for information transfer, **W**, is 16.6 msec with the estimate for the standard deviation of the logistic, **b**, being 9.12 msec. Minimum Chi-square for this fit was 6.83 on 13 degrees of freedom.

Appendix C - Cont'd

Parameter Estimation and Goodness-of-Fit of Model to the Data Obtained from Subject G.T. on Sessions 176-180.

x	p(R≤x)	F(x)
279	.0000	.0001
286	.0047	.0020
292	.0207	.0198
297	.0934	.0901
301	.2134	.2120
304	.3449	.3425
306	.4463	.4427
307	.5023	.4947
308	.5544	.5469
310	.6438	.6478
313	.7799	.7802
317	.9093	.9052
322	.9800	.9787
328	1.000	.9979
335	1.000	.9999

Note. The symbol **R** refers to the time of response and **x** is the upper limit of integration for the distribution function. The best estimate of the maximum waiting time for information transfer, **W**, is 14.8 msec with the estimate for the standard deviation of the logistic, **b**, being 4.4 msec. Minimum Chi-square for this fit was 3.34 on 11 degrees of freedom.

Appendix C - Cont'd

Parameter Estimation and Goodness-of-Fit of Model to the Data Obtained from Subject G.H. on Sessions 266-270.

x	p(R≤x)	F(x)
433	.0000	.0000
440	.0000	.0003
446	.0027	.0063
451	.0500	.0545
455	.1760	.1829
458	.3460	.3443
460	.4747	.4733
461	.5440	.5399
462	.6007	.6053
464	.7260	.7261
467	.8587	.8663
471	.9627	.9656
476	.9933	.9965
482	1.000	.9999
489	1.000	1.000

Note. The symbol **R** refers to the time of response and **x** is the upper limit of integration for the distribution function. The best estimate of the maximum waiting time for information transfer, **W**, is 11.4 msec with the estimate for the standard deviation of the logistic, **b**, being 3.6 msec. Minimum Chi-square for this fit was 4.81 on 9 degrees of freedom.

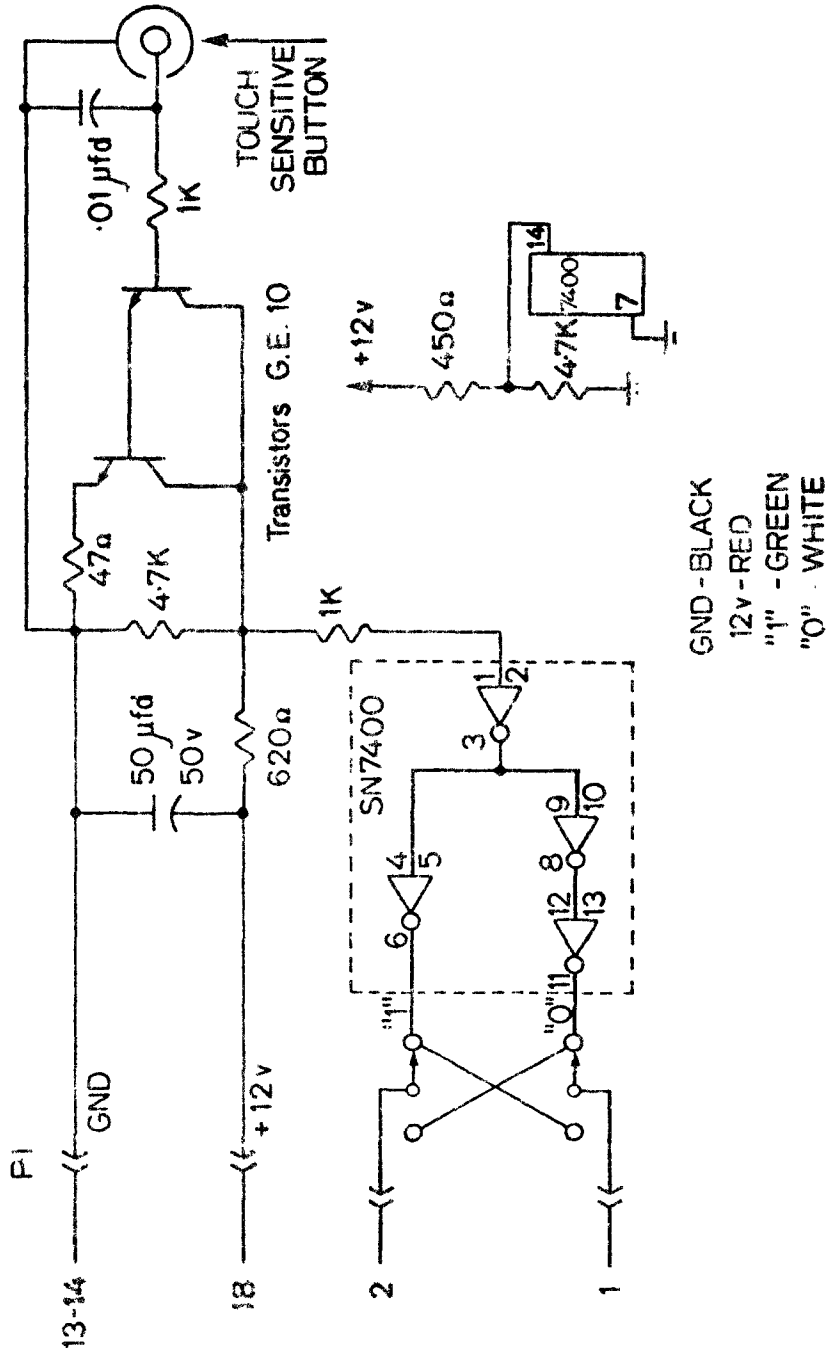
Appendix C - Cont'd

Parameter Estimation and Goodness-of-Fit of Model to the Data Obtained from Subject G.H. by Combining the Five Best Sessions from Sessions 376-395.

x	p(R≤x)	F(x)
432	.0000	.0000
439	.0000	.0003
445	.0033	.0041
450	.0360	.0322
454	.1281	.1270
457	.2742	.2749
459	.4023	.4068
460	.4750	.4783
461	.5550	.5505
463	.6871	.6877
466	.8479	.8489
470	.9646	.9595
475	.9960	.9947
481	1.000	.9997
488	1.000	1.000

Note. The symbol **R** refers to the time of response and **x** is the upper limit of integration for the distribution function. The best estimate of the maximum waiting time for information transfer, **W**, is 9.1 msec with the estimate for the standard deviation of the logistic, **b**, being 4.2 msec. Minimum Chi-square for this fit was 4.11 on 9 degrees of freedom.

Appendix D - Schematic of Electronic Response Button



Appendix E - Mathematical Derivation of Model Logistic Distribution

Probability Density Function

$$f_X(x) = \frac{e^{\frac{x-a}{k}}}{k\left[1 + e^{\frac{x-a}{k}}\right]^2} = \frac{1}{4k}\,\mathrm{sech}^2\left[\frac{x-a}{k}\right]$$

a = Location Parameter (mean)
b = Scale Parameter (s.d.)
$k = \frac{\sqrt{3}\,b}{\pi}$ = Alternate Scale Parameter

Cumulative Distribution Function

$$F_X(x) = 1 - \left[1 + e^{\frac{x-a}{k}}\right]^{-1} = \frac{1}{\left[1 + e^{\frac{-(x-a)}{k}}\right]}$$

$$= \frac{1}{2}\left[1 + \tanh\left[\frac{\frac{1}{2}(x-a)}{k}\right]\right]$$

Moment Generating Function

$$E(e^{tx}) = e^{at}\,\Gamma(1-kt)\,\Gamma(1+kt)$$

$$= \pi kt\,\mathrm{cosech}(\pi kt)$$

Mean = a
Median = a
Mode = a

Variance = $b^2 = \frac{k^2\pi^2}{3}$
Standard Deviation = $b = \frac{k\pi}{3}$
Coeff. of Skew = 0

Range = $-\infty \leq x \leq \infty$

Random Number Generation

$R \sim$ rectangularly

$X: a,k \sim a + k \ln[R/(1-R)]$

Appendix E - Cont'd
Distribution to Density Function of Logistic

$$\frac{d}{dx}\left[1 + e^{\frac{-(x-a)}{k}}\right]^{-1}$$

Let: $u = \frac{-(x-a)}{k}$ $\qquad \frac{du}{dx} = \frac{-1}{k}$

$z = 1 + e^{u}$ $\qquad \frac{dz}{du} = e^{u}$

$v = z^{-1}$ $\qquad \frac{dv}{dz} = -z^{-2}$

$$= \frac{dv}{dz} \cdot \frac{dz}{du} \cdot \frac{du}{dx}$$

$$= -z^{-2} \cdot e^{u} \cdot \frac{-1}{k}$$

$$= \left[1 + e^{\frac{-(x-a)}{k}}\right]^{-2} \cdot e^{\frac{-(x-a)}{k}} \cdot \frac{1}{k}$$

$$= \frac{1}{k} \frac{e^{\frac{-(x-a)}{k}}}{\left[1 + e^{\frac{-(x-a)}{k}}\right]^{2}}$$

$$= \frac{1}{k}\left[\frac{\frac{1}{e^{\frac{x-a}{k}}}}{1 + \frac{2}{e^{\frac{x-a}{k}}} + \frac{1}{e^{\frac{2(x-a)}{k}}}}\right] \times \frac{e^{\frac{2(x-a)}{k}}}{e^{\frac{2(x-a)}{k}}}$$

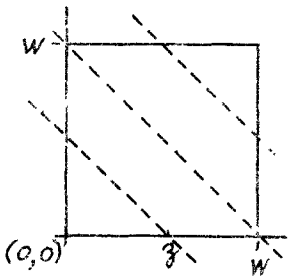
$$= \frac{1}{k}\left[\frac{e^{\frac{x-a}{k}}}{e^{\frac{2(x-a)}{k}} + 2e^{\frac{(x-a)}{k}} + 1}\right]$$

$$f_X(x) = \frac{1}{k}\left[\frac{e^{\frac{x-a}{k}}}{\left(1 + e^{\frac{x-a}{k}}\right)^{2}}\right]$$

Appendix E - Cont'd
Convolution of Two Rectangular Distributions

$$f_X(x) = \frac{1}{W} \qquad f_Y(y) = \frac{1}{W}$$

$$f_{X,Y}(x,y) = \int \frac{1}{W^2}\, dx = f_Z(z)$$



$$f_Z(z) = \int_0^{z} \frac{1}{W^2}\, dx \qquad 0 < z < W$$

$$\int_z^{2W} \frac{1}{W^2}\, dx \qquad W < z < 2W$$

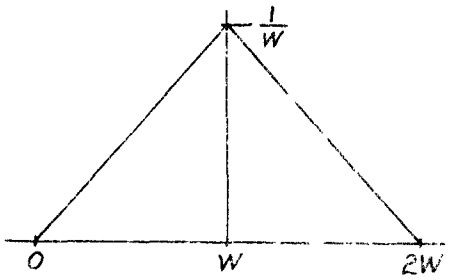
$$f_Z(z) = \frac{1}{W^2}\,[z - 0] \qquad 0 < z < W$$

$$\frac{1}{W^2}\,[2W - z] \qquad W < z < 2W$$

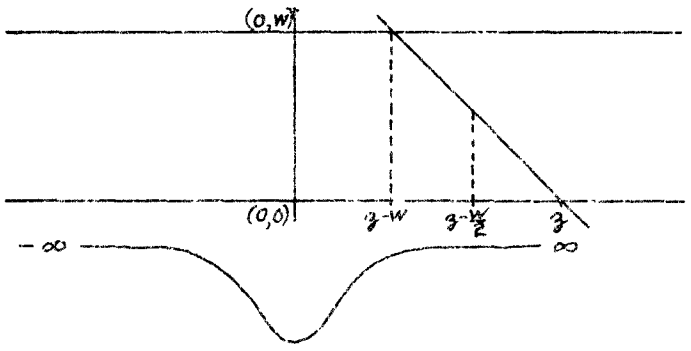
$$f_Z(z) = \frac{z}{W^2} \qquad \text{for } 0 < z < W$$

$$\frac{2W - z}{W^2} \qquad \text{for } W < z < 2W$$

= TRIANGLE



Appendix E - Cont'd
Convolution of Rectangular and Logistic Distributions



$$f_Z(z) = \int_{z-w}^{z} \frac{1}{w} \cdot \frac{e^{\frac{x-a}{k}}}{k\left(1+e^{\frac{x-a}{k}}\right)^2}\, dx$$

Let $y = \frac{x-a}{k}$ $\quad \frac{dy}{dx} = \frac{1}{k}$ $\quad dx = k\,dy$

$$= \frac{1}{w}\int \frac{e^y}{(1+e^y)^2}\, dy$$

Let $u = e^y$ $\quad \frac{du}{dy} = e^y$ $\quad dy = \frac{du}{e^y} = \frac{du}{u}$

$$= \frac{1}{w}\int \frac{1}{(1+u)^2}\, du$$

$$= \frac{1}{w}\left[\frac{-1}{(1+u)}\right]\Bigg|_{z-w}^{z}$$

$$f_Z(z) = \frac{1}{w}\left[\frac{-1}{1+e^{\frac{z-a}{k}}}\right] - \frac{1}{w}\left[\frac{-1}{1+e^{\frac{z-w-a}{k}}}\right]$$

Appendix E - Cont'd
Convolution of Rectangular with Convolution of Rectangular and Logistic Distributions

$$f_X(x) = \frac{1}{W}\left[\frac{-1}{\left(1 + e^{\frac{x-a}{k}}\right)}\right] - \frac{1}{W}\left[\frac{-1}{\left(1 + e^{\frac{x-W-a}{k}}\right)}\right]$$

$$f_Y(y) = \frac{1}{N}$$

$$f_Z(z) = \int_{z-w}^{z} \frac{1}{W} \cdot \left[\frac{1}{W}\left[\frac{-1}{\left(1+e^{\frac{x-a}{k}}\right)}\right] - \frac{1}{W}\left[\frac{-1}{\left(1 + e^{\frac{x-W-a}{k}}\right)}\right]\right] dx$$

$$= \frac{-1}{W^2}\int \frac{1}{\left(1 + e^{\frac{x-a}{k}}\right)}\, dx + \frac{1}{W^2}\int \frac{1}{\left(1+e^{\frac{x-W-a}{k}}\right)}\, dx$$

$$= -A + B$$

Let $w = \frac{x-a}{k}$ $\quad \frac{dw}{dx} = \frac{1}{k}$ $\quad dx = k\,dw$

$$A = \frac{1}{W^2}\int \frac{1}{1+e^{w}} \cdot k\, dw$$

Let $u = e^{w}$ $\quad \frac{du}{dw} = e^{w}$ $\quad dw = \frac{du}{e^{w}} = \frac{du}{u}$

$$= \frac{k}{W^2}\int \frac{1}{1+u} \cdot \frac{du}{u}$$

$$= \frac{k}{W^2}\int \frac{1}{u(1+u)}\, du$$

$$= \frac{k}{W^2}\left[-\ln\left(\frac{1+u}{u}\right)\right]$$

$$= \frac{-k}{W^2}\left[\ln\left(\frac{1+e^{\frac{x-a}{k}}}{e^{\frac{x-a}{k}}}\right)\right]\Bigg|_{z-W}^{z}$$

Appendix E - Cont'd

By Similar Substitutions: $w = \frac{x-W-a}{k}$ $u = e^{w}$

$$B = \frac{1}{W^2}\int \frac{1}{1+e^{w}}\, k\, dw$$

$$= \frac{k}{W^2}\int \frac{1}{u(1+u)}\, du$$

$$= \frac{k}{W^2}\left[-\ln\left(\frac{1+u}{u}\right)\right]$$

$$= \frac{-k}{W^2}\left[\ln\left(\frac{1+e^{\frac{x-W-a}{k}}}{e^{\frac{x-W-a}{k}}}\right)\right]\Bigg|_{z-W}^{z}$$

$$f_Z(z) = -A + B$$

$$= \frac{k}{W^2}\left[\ln\left(\frac{1+e^{\frac{z-a}{k}}}{e^{\frac{z-a}{k}}}\right) - \ln\left(\frac{1+e^{\frac{z-W-a}{k}}}{e^{\frac{z-W-a}{k}}}\right)\right]$$

$$- \frac{k}{W^2}\left[\ln\left(\frac{1+e^{\frac{z-W-a}{k}}}{e^{\frac{z-W-a}{k}}}\right) - \ln\left(\frac{1+e^{\frac{z-2W-a}{k}}}{e^{\frac{z-2W-a}{k}}}\right)\right]$$

$$f_Z(z) = \frac{k}{W^2}\ln\left(\frac{1+e^{\frac{z-a}{k}}}{e^{\frac{z-a}{k}}}\right) + \frac{k}{W^2}\ln\left(\frac{1+e^{\frac{z-2W-a}{k}}}{e^{\frac{z-2W-a}{k}}}\right) - \frac{2k}{W^2}\ln\left(\frac{1+e^{\frac{z-W-a}{k}}}{e^{\frac{z-W-a}{k}}}\right)$$

$$= \frac{k}{W^2}\ln\left(e^{\frac{-(z-a)}{k}} + 1\right) + \frac{k}{W^2}\ln\left(e^{\frac{-(z-2W-a)}{k}} + 1\right) - \frac{2k}{W^2}\ln\left(e^{\frac{-(z-W-a)}{k}} + 1\right)$$

$$= \frac{k}{W^2}\left[\ln\left[\left(e^{\frac{a-z}{k}} + 1\right)\cdot\left(e^{\frac{2W+a-z}{k}} + 1\right)\right]\right] - \frac{2k}{W^2}\cdot\ln\left(e^{\frac{W+a-z}{k}} + 1\right)$$

= DENSITY FUNCTION

Appendix E - Cont'd

$$F_Z(z) = \int \frac{k}{W^2}\left[\ln\left(e^{\frac{a-z}{k}}+1\right)+\ln\left(e^{\frac{2W+a-z}{k}}+1\right)\right] - \frac{2k}{W^2}\cdot\ln\left(e^{\frac{W+a-z}{k}},1\right)\ dz$$

$$= \frac{k}{W^2}\int \ln\left(e^{\frac{a-z}{k}}+1\right) dz + \frac{k}{W^2}\int \ln\left(e^{\frac{2W+a-z}{k}}+1\right) dz - \frac{2k}{W^2}\int \ln\left(e^{\frac{W+a-z}{k}}+1\right) dz$$

$$= A + B - C$$

$$A = \frac{k}{W^2}\int \ln\left(e^{\frac{a-x}{k}}+1\right) dz$$

Let $u = \frac{a-z}{k}$ $\quad \frac{du}{dz} = \frac{-1}{k}$ $\quad dz = -k\,du$

$$= \frac{-k^2}{W^2}\int \ln\left(e^{u}+1\right) du$$

Let $x = e^u+1$ $\quad \frac{dx}{du} = e^u$ $\quad du = \frac{dx}{e^u} = \frac{dx}{x-1} = \frac{dx}{(-1+x)}$

$$= \frac{-k^2}{W^2}\int \frac{\ln(x)}{(-1+x)}\,dx$$

$$A = \frac{-k^2}{W^2}\left[\ln\left(e^{\frac{a-z}{k}}+1\right)\cdot\ln\left(e^{\frac{a-x}{k}}\right) - \frac{1}{2}\left(\ln\left(e^{\frac{a-z}{k}}+1\right)\right)^2 - \sum_{n=1}^{\infty}\left(\frac{1}{\left(e^{\frac{a-z}{k}}+1\right)^n (n)^2}\right)\right]$$

Similarly for B and C:

$$B = \frac{-k^2}{W^2}\left[\ln\left(e^{\frac{2W+a-z}{k}}+1\right)\ln\left(e^{\frac{2W+a-z}{k}}\right) - \frac{1}{2}\left(\ln\left(e^{\frac{2W+a-z}{k}}+1\right)\right)^2 - \sum_{n=1}^{\infty}\left(\frac{1}{\left(e^{\frac{2W+a-z}{k}}+1\right)^n (n)^2}\right)\right]$$

$$C = \frac{2k^2}{W^2}\left[\ln\left(e^{\frac{W+a-z}{k}}+1\right)\ln\left(e^{\frac{W+a-z}{k}}\right) - \frac{1}{2}\left(\ln\left(e^{\frac{W+a-z}{k}}+1\right)\right)^2 - \sum_{n=1}^{\infty}\left(\frac{1}{\left(e^{\frac{W+a}{k}\,z}+1\right)^n (n)^2}\right)\right]$$

$F_Z(z) = A + B - C$ = DISTRIBUTION FUNCTION