herent in any mathematical model before applying the model, and indicates that the recent formulation by Rall may be useful in the analysis of synchronous activation of units that have linearly ordered structural symmetry about the recording electrode.

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7. This corrects an error in the function published by Calvin and Hellerstein. The equation is derived as the time derivative of the step response of the cable equation [see (6)].
8. W. Rall, *Ann. N.Y. Acad. Sci.* **96**, 1971 (1962); *Biophys. J.* **2** (No. 2, part 2), 145 (1962).
9. The $k_1$ now depends on the intracellular and extracellular resistivities and the dendritic tree cross-sectional area relative to the total area.
10. In addition, the cable model EPSP curves show a delay in latency to peak with increasing depth, but no shift in location of spatial peak. Even if one could accept this analysis on other grounds, it does not account for the shift in spatial position of the peak observed in the fast transient or presumed spike recordings of Llinás *et al.* Hence the theory developed by Calvin and Hellerstein does not agree with the data it was meant to explain.
11. R. Lorente de Nó, *Stud. Rockefeller Inst. Med. Res.* **132**, 384 (1947); C. F. Stevens, *Neurophysiology: A Primer* (Wiley, New York, 1966), pp. 161–173. Stevens shows that the field potential expression includes a term for the surface potential as well as the membrane current. Figures 1 and 5 of Clark and Plonsey (*13*) permit comparison of the membrane current and field potential distributions; they are quite similar, so it seems permissible to neglect the potential term and substitute the local membrane current for the weighted integral of all membrane current.
12. J. D. Jackson, *Classical Electrodynamics* (Wiley, New York, 1965), p. 16.
13. This analysis was first provided by Rall and Shepherd (*4*). The effect of boundary conditions on extracellular potentials was studied by J. Clark and R. Plonsey [*Biophys. J.* **8**, 842 (1968)]; they showed that a current-restricting boundary will confine action currents to longitudinal pathways and triphasic spikes become almost monophasic.
14. Contrast this result to that of Calvin and Hellerstein: By assuming proportionality between membrane and field potentials, not gradients, they arbitrarily set the zero point of the field potentials at infinity and assign this as the location of the reference electrode.
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16. All three models fit the data best if a time constant of 15 to 20 msec is used. This is in part due to the use of an impulse to represent an EPSP; the exact EPSP responses of all models would be slower and would fit the recordings with a time constant of about 10 msec. Note that the restricted current potential divider model predicts a space constant of 300 $\mu$; the others fit for $\lambda =$ about 150 $\mu$.
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28 February 1969; revised 14 April 1969

## Temporal Order Judgment and Reaction Time

Abstract. *A model which predicts judgment of the temporal order of stimuli from simple reaction time is proposed. Visual data show covariation of the two measures with luminance changes, and suggest that (i) temporal order judgments reflect a biased response criterion and (ii) the motor component of reaction time has little variability relative to variance in receptor system latency.*

One measure of the relative latency of two receptor systems is obtained from judgments by subjects of the temporal order of occurrence (TO) of stimuli separated by a small time interval. Interstimulus intervals that yield maximum uncertainty about which stimulus was presented first are held to reflect typical receptor system latency differences (*1*). Simple reaction time (RT) is also a measure of receptor system latency and should yield comparable inferences about latency differences between stimuli. A previous study (*2*) compared RT and TO data from heteromodal stimuli and found a considerable discrepancy between typical latency differences inferred from the two measures. Our report demonstrates a direct covariation between RT and TO with ipsimodal stimuli, and proposes a common underlying theoretical framework which allows a prediction of the TO performance from simple RT to the same stimuli.

The essential features of the theoretical proposal are shown in Fig. 1. Densities corresponding to receptor system latencies to the two stimuli are presented one above the other, with the convention that the origin of the time scale is located at presentation of stimulus 1. In this example the interstimulus interval, $\tau$, is negative, meaning that stimulus 2 is presented first, and so the latency density, $f_2$ $(t_2)$, for

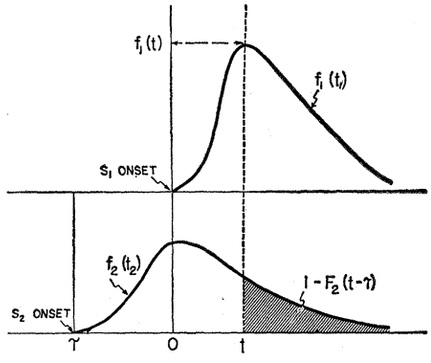


Fig. 1. Densities for detection latencies after stimulation from two sources, offset by the interstimulus interval, $\tau$. Hatched area is the probability that the latency for stimulus 2 exceeds a latency of $t$ for stimulus 1.

stimulus 2 has been shifted $\tau$ units to the left. Three assumptions are sufficient to produce a prediction of the psychometric function relating the probability of a stimulus 1 report to $\tau$. (i) Receptor system latencies are independent of each other; (ii) neither distribution is changed by changes in $\tau$; and (iii) the subject reports "stimulus 1 first" whenever on a particular trial stimulus 1 latency is exceeded by stimulus 2 latency plus $\tau$. That is, the subject reports physiological asynchrony as physical asynchrony and has no difficulty in discriminating which input system was first.

Under these assumptions for an arbitrary input latency, $t$, from receptor system 1 (dotted line in Fig. 1), the probability of a stimulus 1 report is simply $1 - F_2(t - \tau)$. This formulation is appropriate for positive values of $\tau$ as well, as the latency distribution functions are zero for negative arguments. Weighting each probability by the density of $t$, and integrating, we find

$$P_\tau(\text{"S1 first"}) \equiv F(\tau) = \int_0^\infty f_1(t)\,[1 - F_2(t - \tau)]\,dt \quad (1)$$

where $F(\tau)$ is the cumulative form of the latency difference distribution (*3*). The decision rule might have been stated as the following: the subject reports "stimulus 1 first" whenever $t_1 - t_2 < \tau$. Varying $\tau$ produces different criterion values and the decision procedure is the same as that described for the two-alternative forced-choice situation in signal detection theory (*4*). The mean and variance of the difference distribution are the difference

and sum, respectively, of the means and variances of the individual latency distributions.

The data analysis presented uses simple RT distributions as estimates of the shape of the receptor system latency distributions. If the motor component of RT is, to a first approximation, constant, $F(\tau)$ is directly obtainable from RT since the constant vanishes in the difference distribution. If the motor component of RT is not constant, then additional variance should appear in the predicted $F(\tau)$ resulting in a shallower slope.

Predicted and obtained $F(\tau)$'s are reported for visual stimulus pairs which differed in the luminance of one of the two stimuli. The data thus provide an assessment of whether changes in relative latency are reflected in comparable ways in RT and TO performance.

Ten-millisecond flashes generated by Sylvania R1131C glow modulator tubes were presented against a spherical background of uniform luminance (−1.5 log mlam). With central fixation, one flash stimulated the fovea of the left eye and the other the nasal retina of the right eye at 50° on the horizontal meridian. The luminance of the foveal flash was varied between conditions to be higher (condition I, +2.1 log mlam), equal (condition II, −0.1 log mlam), and lower (condition III, −1.2 log mlam) than the peripheral flash. These luminance combinations were selected because previous results suggested they would yield changes in foveal latency from shorter to longer than peripheral latency (*1*).

Sessions consisted of two blocks of 80 TO trials and two blocks of 80 RT trials. The order of blocks was varied between sessions. On TO trials $\tau$ values were varied in equal interval steps by the method of constant stimuli, and the subject was instructed to make a forced-choice judgment of which flash appeared first. On RT trials the same stimuli were presented singly in random sequence, and the subject was instructed to react by lifting his finger as fast as possible (*1*, *2*).

The predicted (open circles) and obtained (filled circles) psychometric functions for each luminance condition are shown in Fig. 2 for both subjects. The data in each panel are averaged over sessions (*5*). Within each condition the predicted and obtained $F(\tau)$ are similar in shape and close together relative to the span in $\tau$ required to bracket the psychometric function. Across conditions both functions move toward less negative $\tau$'s with decreasing luminance of the foveal flash. Since the intensity of the peripheral flash remained constant, this shift reflects the lengthening of latency to the foveal flash. The decrease in slope of the functions with decreasing foveal luminance is consistent with latency mechanisms which increase variances with means (*6*). The subjects differ in the size of the latency difference to a given stimulus pair, and in the amount of the change in foveal latency across conditions. Individual variations of this sort have been observed previously and appear to be typical of TO performance (*1*). For example, with flashes of equal luminance (condition II), one subject (JT) shows a lag in the peripheral system relative to the foveal system for both the RT prediction and the obtained TO data, whereas the other subject (BM) shows little difference in typical latency to these flashes. Both subjects, however, respond in qualitatively similar ways to changes in stimulus conditions. Under our assumptions, the two tasks yield comparable descriptions of changes in relative latency.

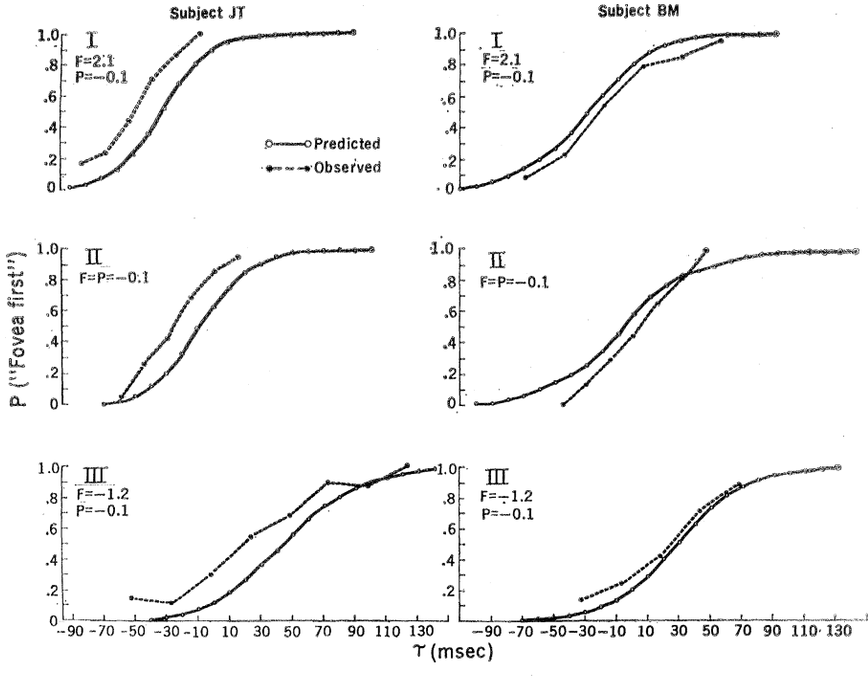


Fig. 2. The probability of a report of "fovea first" as a function of the interstimulus interval, $\tau$. Negative $\tau$'s mean that the peripheral flash was presented first. Three luminance combinations (rows) are shown for each subject (columns). Filled circles are data from TO judgments, and open circles are predictions of the TO performance from RT.

Reaction time and temporal order do not, however, yield identical descriptions of relative latency. The predicted and obtained functions in Fig. 2 differ reliably. Chi-square goodness-of-fit tests (*7*) discriminate the predicted and obtained functions at better than the .05 level for subject BM, condition III, and at better than the .01 level for the other five function pairs. These differences, however, are in location, not form. The obtained TO functions for subject JT may be approximated by the RT predictions with a decision rule to respond "fovea first" when foveal latency is less than peripheral latency plus $\tau$ plus a positive constant. For subject BM, equally good approximations require two different constants, one favoring the periphery for conditions I and II, and another favoring the fovea for condition III (*8*).

The differences between the obtained and predicted psychometric functions appear to result from biased criteria in the latency difference distributions. Such a shift in subjective criterion away from the value of $\tau$ amounts to a handicap of one system over the other. The discrepancy observed previously with heteromodal stimulus pairs (*2*) may result from a bias of this sort. If this view is correct, the size and direction of the handicap, which appears in TO but not in RT, should be manipu-

latable by contemporary psychophysical procedures such as payoff changes.

The striking similarity in the shape of the RT and TO functions was surprising to us. It strongly suggests that the motor component in the RT task adds little variance relative to variability in receptor system latency. Since any variance in the motor component is doubled in the difference distribution, the predicted psychometric function should appear shallower than the obtained. As this is not the case, it is likely that motor component variability is low. An alternative view would require that the contribution of motor variance in the prediction be just offset by an additional source of variability in the TO performance.

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5. A minimum of 5 (condition III) and a maximum of 18 (condition I) sessions were run under each luminance combination, with the first session for each condition not included in the data analysis. The obtained TO functions were adjusted for intersession variability by shifting the functions from individual sessions so that 50 percent points were superimposed upon the average of the 50 percent points for all sessions within a given condition, and the resulting data were pooled. The RT functions were adjusted in a similar manner by shifting all RT distributions from individual sessions so that their means were superimposed upon the grand mean of all sessions within a given condition. The resulting pooled RT distributions for fovea and periphery were then used to calculate the RT difference distribution from the discrete analog of Eq. 1

$$P(k) = \begin{cases} \sum_{i=1}^{N} P_1(i+k) \sum_{j=i}^{N} P_2(j), & k \leqslant 0 \\ \sum_{i=1}^{N} P_1(i) \sum_{j=i+k}^{N} P_2(j), & k > 0 \end{cases}$$

where $k$ is the $\tau$ category, $N$ is the $\tau$ category for the largest $\tau$, and $P_s(i)$, $s = 1, 2$, is the probability that a latency from receptor $s$ falls in category $i$.
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7. The goodness-of-fit test was adapted from D. J. Finney, *Probit Analysis* (Cambridge Univ. Press, London, ed. 2, 1952). The criterion statistic is the squared normalized binomial variate with expected values calculated by interpolation from the predicted functions. The reliability levels reported in the text are for $\chi^2$ computed with the extreme upper and lower points of the obtained functions omitted. The number of trials run at these $\tau$ values was considerably lower than for intermediate points (an average of 25.6 per point for the extremes versus 132.1 per point for intermediate points). Consequently the middle ranges of the obtained functions are more reliable than the extremes. When the extremes are included in the $\chi^2$ computations, all function pairs differ at well below the .001 level.
8. The predicted functions were shifted by an amount equal to a weighted sum of the difference between interpolated medians for the two functions. The weights used were the proportion of the total $N$ in each condition. The resulting values were −19.07 msec for subject JT, conditions I, II, and III; +8.40 msec for subject BM, conditions I and II; and −5.25 msec for subject BM, condition III. Adding these constants to $\tau$ in the predicted $F(\tau)$'s, resulted in $\chi^2$ values with associated probabilities above .25 with the extremes of the obtained functions omitted. When the extreme values were included, these probabilities fell to between .1 and .05 for subject JT, and between .05 and .025 for subject BM. Iterative procedures would undoubtedly improve these estimates, however the difference between medians appeared to provide a reasonable first approximation to the data. Moreover, it is difficult to decide on an appropriate test size when a discrimination is attempted between a model and no model. Goodness-of-fit levels carry more weight against sharp alternatives.
9. Supported in part by NINDB grant NB 05221, PHS grant MHO 3616, and NIH grants MH 07279 and GRS 5-SO-1-FRO 5650. Dr. J. Kerr wrote the computer programs used for data analysis.

23 December 1968; revised 16 April 1969

## Coevolution

I challenge Muller's interpretation (*1*) of the coevolution of plant and animal interactions in that: (i) He has cited no direct evidence (and neither is any evidence available for the vast majority of defensive compounds) that the secondary substances are "primarily metabolic wastes." It is hard to understand how many compounds (for example, alkaloids, free amino acids, saponins, glycosides, and so forth) virtually unique to plant metabolic systems can be considered waste products, when animal metabolic systems do quite well with very few kinds of waste products (except for sessile marine animals which are known to contain many of the same compounds Muller regards as plant waste products). (ii) The need to void, sequester, or otherwise render a toxic compound unavailable to the producing organism is not evidence that the toxic compound is a waste product. By Muller's line of reasoning, the defensive compounds of animals must also be waste products. (iii) Natural selection serves as a mechanism by which a population of herbivores may "call forth *de novo*" the evolution of a biosynthetic pathway producing compounds toxic to the herbivore. Obviously, initial stages in such a pathway may arise through mutation, or other genetic changes, just as do initial stages of any other biosynthetic pathway. For selection for the production of a toxic compound, all that is required is that the new form of compound in the mutant plant strain be slightly toxic, deterrent, hallucinogenic, distasteful, sleep inducing, and so forth, to the herbivore. (iv) The failure "to regard such [secondary compounds] as primarily animal [and other plant] toxins renders impossible the explanation of how these products came to be." What other selective force in the environment besides herbivores (*sensu latu*) and competing plants has the diversity of quality, yet specific persistence, of environmental challenge to lead to essentially a unique combination and array of secondary compounds for each species of plant? (v) Acetylcholine, bile, trypsin, and vitamin A are toxic to animals in large doses. Muller's reasoning would lead to the conclusion that these are metabolic waste products, because they would intoxicate the system if not eliminated or used. That a complex compound is a potential intoxicant of the system producing it can hardly be taken as the definition of a waste product.

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2 May 1969

The debate initiated by Ehrlich (*1*), and now joined by Janzen, began with objection to my interpretation of the nature of toxic compounds released by plants and effective against other plants. The implication of both critics that there exists no difference between those toxins effective in plant-plant and those effective in plant-animal interactions is too simplistic to fit the facts and is unduly emphatic in its unswerving zoocentricism. This stance is, of course, necessary to their thesis that animals somehow cause plants to initiate novel metabolic pathways (where I have ascribed to animals the role of selecting between existing pathways by means