Counting and Timing Mechanisms in Auditory Discrimination and Reaction Time

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1. INTRODUCTION

Models of behavior can be constructed in at least three different ways.

1. One can begin with a few empirical generalizations which, taken as postulates or axioms, lead deductively to a variety of testable predictions.

2. One can postulate some internal mechanism as mediating the behavior and, after estimating the parameters of that mechanism, then predict a variety of other behaviors.

3. One can actually investigate the internal workings of the organism, describe these mechanisms axiomatically, measure the needed parameters, and deduce the behaviors.

If the behavior in question is psychophysical, then the first two approaches call only for psychophysical data, whereas the third requires physiological data of some sort. A pure example of the last approach is rare; usually a physiological model is blended to some degree with a hypothetical model. One reason is that, even with animals, our clearest and most detailed information comes only from the peripheral nervous system, and so we are forced to speculate how that information is processed by higher centers. This chapter presents an example of this compromise approach for auditory psychophysics.

Examples of the other approaches can be found in this volume: (a) Falmagne; (b) Levine, Krantz; and (c) Graham and Ratliff.

Perhaps the single most pervasive characteristic of psychophysical data is the inconsistency of subjects when answering most questions we ask them about simple stimuli. Somewhere, between the stimulus and the response, randomness enters. One hundred years of research, the most careful of methodological practices, and the best signal sources provided by modern technology have not reduced below 6 percent the separation in intensity needed for two 1000 Hz tones to be discriminated 75 percent of the time. For this reason, many theorists believe that a general theory of psychophysics can hardly avoid an explicit formulation of this randomness. Other theorists, most notably Stevens (1957, 1971), have argued that this 'noise,' although pervasive, is completely incidental to the main effects in psychophysics, and so it is best averaged away. The former group, and we are in it, feel that the interlock between the global and local aspects of psychophysics is much more profound, although not as simple as some earlier theorists (Fechner, 1860; Luce, 1959; Thurstone, 1927) have suggested.

Variability can intrude itself at five places: (a) in the physical signal itself; (b) in its transduction from physical energy into the pulsed 'language' of the nervous system; (c) in the various transformations imposed as the peripheral neural information wends its way through the central nervous system; (d) in the decision process which converts the available information into an answer to whatever question has been asked about the nature of the signal; and (e) in the processes that lead to the execution of a response. Different theorists have focused on particular sources of randomness, attempting to show that one of these accounts for most of the overall variability. For example, Hecht, Schlaer, and Pirenne (1942), in a classic study of visual thresholds, held that the quantum variability of a threshold light source coupled with quantal losses prior to the retina were sufficient to account for the observed psychophysical variability. Later (Sec. 10) we argue that in a simple reaction-time experiment to intense signals the variability of observed times is dominated by conduction times and synaptic delays and that essentially no measurable variability is added to it by the sensory or decision aspects of the process. A number of authors (including Thurstone, 1927; Tanner & Swets, 1954; the whole resulting school of signal detectability; Green & Swets, 1966; Luce & Green, 1972) have taken the view that in many situations it is unnecessary to partition the variability due to the first three sources-the signal, its transduction into a neural language, and its transmission in the nervous system up to the point where a decision is made-and for a number of experimental tasks and measures, usually those involving some aspect of discrimination, the total variability associated with these three sources is all that need be considered. For other tasks and measures, however, the variability introduced by the decision process itself or that added by the remainder of the response

process may play a significant role, as we discuss in Sections 4, 5, and 10. From this point of view, a key initial question is how the information is encoded when it reaches the decision center. Sections 2 and 4 of the chapter deal with this topic. Section 2 summarizes some peripheral physiological evidence concerning the coding of auditory information. Section 3 treats what an ideal device, making optimum use of this information, could do. Section 4 advances some hypotheses, admittedly speculative, about the form that the sensory information takes as it is presented to the decision center. These hypotheses are supported to some degree by comparing their predictions with psychophysical data from a detection experiment with response deadlines. Another detailed comparison of these predictions is carried out in Section 5, which discusses the speed-accuracy trade off. Sections 6 through 8 provide an account of the classical data on the discrimination of changes in intensity or frequency of a pure tone signal. Section 9 briefly discusses how still other sources of variability can influence psychophysical data, especially those data in which a number of response categories are employed. Section 10 continues with a discussion of how various parameters of the model can be measured from reaction-time data and an explanation of how another source of variability can be estimated.

2. THE PERIPHERAL NEURAL REPRESENTATION OF AUDITORY SIGNALS

What we say here, and so in the rest of the chapter, pertains only to the representation of auditory signals. No comparable data for other modalities have yet been collected. We suspect that certain features of this auditory representation will be found in other senses, but many of the details will undoubtedly differ in important ways.

An auditory input signal is simply a continuous function of time, for example, it is pressure as a function of time. When one measures electrical activity in individual nerve fibers of the peripheral auditory nervous system, one does not see anything directly analogous to that function. Rather, each fiber conducts a train of electrical pulses of very brief duration and of approximately the same voltage. At first sight, these pulse trains are highly irregular; sometimes they are obviously affected by changes in the signal; at other times, apparently, they are not. A good deal of very careful work over three decades, especially the last one, has led to some understanding of the exact nature of the encoding involved, although one still cannot predict from a limited set of observations on an individual fiber how it will respond to an arbitrary input signal. For our purposes here, however, it will suffice to have a reasonably detailed description of the neural response to a very limited class of signals, namely, pure tones. It is important to realize that the pulses themselves do not directly carry information about the signal. For example, their size does not change systematically with either signal intensity or frequency. Thus information about the signal must be carried either by the occurrence or by the absence of individual pulses, or by some aspect of their temporal pattern, or by the spatial pattern of activity over the whole auditory fiber bundle. Wever (1949, p. 128) summarized it well:

The nerve impulse seems to be everywhere the same, regardless of the type of nerve in which it appears. The modes of variation of nerve transmission therefore are strictly limited. The following dimensions are generally regarded as exhausting the possibilities of representation by nerves of the physical characteristics of the stimulus: (a) the particular fiber or fibers set in operation, (b) the number of fibers excited at any one time, (c) the frequency of impulses in each fiber, (d) the duration of the train of impulses, and (e) the time relations of the separate impulses passing through different fibers. The problem of auditory theory is to show how these variables represent the properties of the stimulus and determine the nuances of auditory experience.

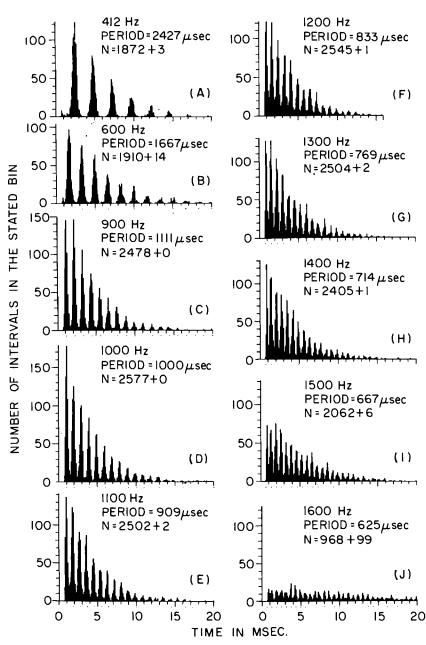
Without going deeply into the details of the arguments, no one today believes that the mere occurrence of a pulse contains any information whatsoever. One reason is that without any signal present, fibers fire spontaneously, sometimes at rather high rates. A second is that there is no sign of synchronization in the peripheral system which would make the absence of a pulse clear. So we turn to questions of temporal and spatial patterns.

For temporal patterns, the first question to resolve is which aspect of a pulse train corresponds to intensity. The well-known fact that reaction time to signal onset decreases with signal intensity (Chocholle, 1940; McGill, 1960) strongly suggests that there must be some deep interplay between intensity and time in the nervous system. One obvious conjecture is that pulse rate increases as signal intensity is increased and everything else is held constant. Peripheral data on the cat (Galambos & Davis, 1943; Kiang, 1965, 1968) and on the squirrel monkey (Rose, Brugge, Anderson, & Hind, 1967) confirm that something of this sort is true, although it is not quite so simple. If we restrict our attention to pure tone signals, the following seems to summarize the situation. Each fiber has a characteristic signal frequency to which it is most, but not exclusively, responsive. At this frequency, there is a lower and upper threshold. Below the lower threshold, it fires at its spontaneous rate; between the two, the rate increases by a factor of from 2 to 10, reaching a maximum rate at the upper threshold; for more intense signals the rate is either maintained or drops somewhat. As the frequency deviates from the characteristic one, both thresholds rise and the maximum firing rate remains about the same. Looked at another way, a pure tone of sufficient intensity activates a particular set of fibers in the sense that it drives their firing rates

above their spontaneous rates. Changing the frequency causes some fibers to drop from the active category and others to enter it; increasing the intensity adds fibers to the active category. Thus, frequency change involves the substitution of fibers (metathetic continuum); intensity change, either the addition or the subtraction of fibers (prothetic continuum). This distinction was discussed by Stevens (1957) and is described fully by Wever (1949).

It is clear, therefore, that both frequency and intensity are represented spatially. In addition, of course, intensity is represented temporally as a firing rate. The question remains what, if any, additional information about frequency may exist in the temporal representation. The only way to answer this is to examine the detailed statistical structure of the pulse trains in the presence of steady, pure tone signals. Galambos and Davis (1943) were the first to do so with the care needed, and improved techniques have been employed by Kiang (1965) and his colleagues and by Rose et al. (1967). Kiang's group mostly used clicks, that is brief pressure pulses, as their stimuli. They concluded that to a first approximation the peripheral neural pulses form a renewal process in which the times between successive pulses are independent of one another and have the same distribution when the signal is constant. The times between neural bursts cluster at the reciprocal of the characteristic frequency of the fiber, indicating that the fibers are most likely to fire at only one phase of the essentially sinusoidal response produced by the brief stimulus. With no stimulus input the process appears to be approximately Poisson; i.e., a renewal process in which the distribution of interarrival times (IATs) between successive neural pulses is exponential. A deviation from the exponential occurs at 0 because very short (less than $\frac{1}{2}$ msec) IATs appear to be lacking, presumably because of absolute refractoriness in the nerve. Increasingly, as various experimental artifacts have been removed, the data appear to be very well approximated by a Poisson process except for very brief times.

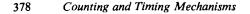
Frequency information about a pure tone signal, at least for frequencies below 2000 Hz (which includes most of the relevant musical range), is also encoded in the pulse train, as has been demonstrated most clearly by Rose and his collaborators. This can be seen by looking at IAT distributions, of which Figures 1 and 2 are typical, provided the measurements are sufficiently precise (at least to 100μ sec). The distributions are startlingly multimodal, with one mode at about 0.5 to 1 msec and the others lying at multiples of the period of the input signal. We refer to the former as the 'sputtering' mode and to the others as 'normal' modes. Thus, for a 1000 Hz signal, the normal modes are 1 msec apart, for a 500 Hz signal they are 2 msec apart, etc. Moreover, the ratio of the heights of the successive normal modes are roughly constant suggesting a geometric distribution having some constant probability p of firing at each successive mode. The probability of the neuron firing exactly i periods after the last firing is $p(1 - p)^{i-1}$. The smearing of these geometric modes may reflect the randomness in the spontaneous Poisson



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FIGURE 1.

Distribution of interspike times on a single cochlear fiber of a squirrel monkey when the acoustic signal is a pure tone of the intensities and frequencies shown. (Rose et al., 1967, Fig. 1.)



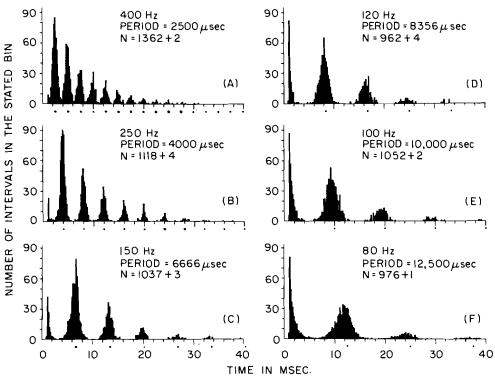


FIGURE 2. Same as Figure 1, but for lower frequencies. (Rose et al., 1967, Fig. 4.)

process which has been modulated by the sinusoidal signal input. Note that, contrary to common belief, there is no sign of refractoriness of more than a fraction of a msec. This is even shorter than the value suggested by Kiang (1965, p. 101). Further, the geometric parameter p is clearly an increasing function of intensity, at least over some limited range.

For some purposes having to do with stimulus intensity, it is reasonable to smear the IAT distribution and to approximate it by an exponential. But one must be careful since the most probable IAT under an exponential density is 0 whereas it is 1/f in Figure 1. If the nervous system ever computes 1/IAT, this error of approximation can loom large (see Sec. 7).

We may conclude that at the periphery, the intensity and frequency of pure tones of low frequency are both represented spatially and temporally in the nervous system. Discrimination mechanisms that draw only on the spatial representation are usually called place mechanisms; those that draw only on the temporal representation, periodicity mechanisms. An obvious question is whether discriminative behavior is based on one or both of these mechanisms. The fact that reaction time is a strong function of intensity suggests that longer time samples are required with weak signals (slow rates) than with intense ones, which in turn suggests that some temporal sampling is involved. Furthermore, as we have verified empirically, the reaction time to either intensity or frequency discrimination of the same quality (say, d' = 1) is the same, suggesting that comparable samples are taken for the two tasks. Other data below will reinforce our belief that much discriminative behavior depends on temporal mechanisms. It is much less clear whether a place mechanism plays any role whatsoever for low-frequency signals. It is entirely possible that the increase in total activity with intensity serves merely to increase the total sample size available. Wever (1949) developed a theory in which periodicity mechanisms dominate at low frequencies and place ones at high frequencies. Our calculations in Sections 6 and 8 can be thought of as elaborating the behavioral predictions of his periodicity mechanisms.

The kind of peripheral encoding of information just described sets firm limits on what the nervous system can possibly do. In whatever way it may ultimately transform this information, it can never decrease the amount of noise that is inherent in the stochastic representation of the signals. In particular, it can never undo the temporal smearing of instantaneous values of intensity. One approach, then, is to formalize the peripheral representation this we do at the end of this section—and then ask what is the best that an ideal machine, using both the temporal and spatial representations, could do with that information. We discuss this approach in the next section, but it appears to be difficult to work it through to a definitive conclusion.

A more adventurous approach, which we undertake in Sections 4 through 8, is to guess what the rest of the (relevant) nervous system does when making decisions in terms of information encoded temporally, ignoring the spatial representation. This approach is both more specific, and so entails more detailed predictions, and far more speculative, and so probably is wrong. The question, of course, is not whether it is wrong in detail, which it is almost bound to be, but whether it is wrong in spirit and so in qualitative character.

We next formulate two models for the spike train on a single neuron. Each of these models describes imperfectly the data of which Figures 1 and 2 are typical. The defects are discussed as the models are presented.

In the first we assume that the pulse train is generated by a nonstationary Poisson process whose hazard function waxes and wanes with the signal amplitude. The particular hazard function postulated by Siebert (1970) is of the form

$$\mu(t; I, f) = \mu_0 \exp \{G[AH(f/f_0)] \cos 2\pi ft\},$$
(1)

where t is time, f_0 is the characteristic frequency of the fiber, $I \propto A^2$ is the intensity of the pressure wave, and f is the frequency; G and H are functions that must be specified, although we will not do so here. Such a process is

obviously phase locked to the sinusoidal signal; it is not a renewal process because successive IATs are not independent; but it is multimodal with modes at 0 and at multiples of the period of the signal. Thus, it accounts for the data almost exactly except, according to the model, the sputtering mode should be at or near 0 instead of $\frac{1}{2}$ to 1 msec. The reason for this discrepancy is simply that the model does not provide for any refractoriness whatsoever.

In the second model we assume that the process is a renewal one in which the IAT random variable is of the form

$$\mathbf{IAT} = \frac{\mathbf{I}}{f} + \mathbf{X},\tag{2}$$

where I is geometrically distributed, i.e.,

$$P(\mathbf{I} = i) = p(1 - p)^{i-1}, \quad i = 1, 2, \dots,$$
(3)

with p some unknown function of intensity I and frequency f. The data suggest that the distribution of X is symmetric and bounded by an interval of the form $\left(-\frac{\epsilon}{f}, \frac{\epsilon}{f}\right)$, $0 < \epsilon < \frac{1}{2}$. In fact, it is reasonable to suppose that there is a symmetric density function g on (-1, 1) with the property that

$$g(1) = g'(1) = 0,$$

and for a signal of frequency f that

$$P(\mathbf{X} = x) = \frac{f}{\epsilon} g\left(\frac{f}{\epsilon} x\right), \quad 0 < \epsilon < \frac{1}{2}.$$
 (4)

Observe that by symmetry

$$E(\mathbf{X}) = 0 \tag{5}$$

and

$$V(\mathbf{X}) = \theta^2 / f^2, \tag{6}$$

where

$$\theta^2 = \epsilon^2 \int_{-1}^1 x^2 g(x) \, dx.$$

Combining Equations 2 and 3, we obtain for the density of IATs,

$$\psi(x) = P(\mathbf{IAT} = x) = \begin{cases} p(1-p)^{i-1} \frac{f}{\epsilon} g\left[\left(x - \frac{i}{f}\right) \frac{f}{\epsilon}\right], & i = 1, 2, \dots, \\ \left|x - \frac{i}{f}\right| \le \frac{\epsilon}{f}, \quad (7) \\ 0, & \text{otherwise.} \end{cases}$$

This renewal model has two major drawbacks. First, it has no 'sputtering' mode at all, that is, no IATs appreciably shorter than 1/f. So when we study discrimination in terms of it we are implicitly assuming that the nervous system is able to filter out the Poisson noise that shows up as the 'sputtering' mode and that it carries out all of its computations on the normal modes. Exactly how this filtering is done we do not attempt to say. One plausible possibility is that some higher order neurons have a refractoriness that is slightly less than the period of the sinusoid. This amounts to a mild form of a place mechanism at some higher order center. Second, by assuming a renewal process (independent IATs) we have no mechanism to maintain the phase locking of the process to the signal. This is certainly wrong, but whether it matters is another question. If the nervous system actually bases its decisions on the IATs, as we shall assume, rather than paying attention to coincidences of pulses over different fibers, then the phase locking is an incidental matter of minor importance.

3. IDEAL DISCRIMINATION OF INTENSITY AND FREQUENCY

Siebert (1968, 1970) has pointed out that the Cramér-Rao inequality (see below) is the important key to determining the possible limits of discriminability. It establishes the limit on the discriminability of a small change of intensity, ΔI , or of frequency, Δf , in terms of the inherent statistical variability of the neural representation of the signal. Of course, we do not know a priori how well the organism actually approaches these theoretical limits. Still, the calculation could be useful in establishing an upper bound on the performance and, depending on how close actual performance is to this bound, it might establish constraints on the possible hypotheses we may entertain concerning the actual detection process.

The form of the Cramér-Rao inequality used by Siebert (1970) is rather more general than the most general statement given in Cramér (1946).¹ Suppose *n* processes are each sampled, the *i*th yielding a vector of κ_i observations (where κ_i may be a random variable), say $x_i = (x_{i1}, \ldots, x_{i\kappa_i})$. Combining these, we may use the abbreviation

$$(\mathbf{x}, \mathbf{\kappa}) = (x_1, \ldots, x_n, \kappa_1, \ldots, \kappa_n).$$

We assume that their joint probability density $\psi(x, \kappa; \xi)$ exists for each value of some parameter ξ (which we will take to be either I or f). Assuming that

¹ Our first draft of this section included a number of misinterpretations of Siebert's arguments, and we are deeply grateful to him for showing us exactly how the argument proceeds. The proofs included in Notes 2 and 3 are taken from his letters to us.

the density function is sufficiently regular (precise conditions are well known) and that $\hat{\xi}(x, \kappa)$ is an unbiased estimator of ξ , then the variance of that estimate must satisfy²

$$\sigma_{\xi}^{2} \geq \frac{1}{\sum_{x} \int_{x} \left(\frac{\partial \ln \psi}{\partial \xi}\right)^{2} \psi \, dx},\tag{8}$$

١

1.

where

$$\sum_{\kappa} = \sum_{\kappa_1=0}^{\infty} \cdots \sum_{\kappa_n=0}^{\infty}$$
 and $\int_{x} = \int_{x_1} \cdots \int_{x_n} \cdots$

Any estimator for which the equality holds in Equation 8 is called *efficient*, and any such discrimination mechanism is called *ideal*.

If, further, the *n* processes are independent in the sense that for each ξ ,

$$\psi(\boldsymbol{x},\boldsymbol{\kappa};\boldsymbol{\xi}) = \prod_{i=1}^{n} \psi_i(x_i,\boldsymbol{\kappa}_i;\boldsymbol{\xi}),$$

then one can show³ that Equation 8 can be replaced by

² By definition of an unbiased estimator,

$$\sum \int \psi(x, \kappa; \xi)[\xi(x, \kappa) - \xi] dx = 0.$$

Differentiate this with respect to ξ which, when ψ is sufficiently smooth, can be carried out to the right of $\sum \int_{\gamma}$,

$$0 = \sum \int \left[\frac{\partial \psi}{\partial \xi} (\hat{\xi} - \xi) - \psi \right] d\mathbf{x}$$
$$= \sum \int \frac{\partial \ln \psi}{\partial \xi} \psi(\hat{\xi} - \xi) d\mathbf{x} - 1$$
$$= \sum \int \left[\frac{\partial \ln \psi}{\partial \xi} \psi^{1/2} \right] [\psi^{1/2} (\hat{\xi} - \xi)] d\mathbf{x} - 1$$

Assuming that ψ is sufficiently well behaved so that Schwarz' inequality holds,

$$1 \leq \left[\sum \int \left(\frac{\partial \ln \psi}{\partial \xi} \right)^2 \psi \, dx \right]^{1/2} \left[\sum \int \psi(\hat{\xi} - \xi)^2 \, dx \right]^{1/2},$$
$$= \left[\sum \int \left(\frac{\partial \ln \psi}{\partial \xi} \right)^2 \psi \, dx \right]^{1/2} \sigma_{\xi}^2. \quad \text{QED}.$$

³ By independence,

$$\frac{\partial \ln \psi}{\partial \xi} = \sum_{i=1}^{n} \frac{\partial \ln \psi_i}{\partial \xi}$$

When substituted in Equation 8 we get squared terms, which constitute the right side of Equation 9, and cross-product terms which we now show are 0:

$$\sigma_{\xi}^{2} \geq \frac{1}{\sum_{i=1}^{n} \sum_{\kappa_{i}=0}^{\infty} \int_{x_{i}} \left(\frac{\partial \psi_{i}}{\partial \xi}\right)^{2} \psi_{i} \, dx_{i}}$$
(9)

Siebert based both of his analyses on Equation 8, but he proceeded somewhat differently in the two papers. In 1968, he assumed that for all fibers *i* $\kappa_i = 1$ and x_{i1} is the number of pulses observed on fiber *i* during some fixed listening interval when signal (I, f) is presented. Assuming that the fibers are independent, a plausible assumption which at present is difficult to justify or reject, and assuming that the process on each fiber is Poisson, so that the distribution of IATs is exponential, it is easy to write an explicit formula for ψ which can then be substituted into Equation 9. This does not really tell us anything until we assume how the Poisson parameters depend on *I* and *f* and how they vary over fibers. Siebert made a number of assumptions which were motivated by his attempt to fit some physiological data, and from these he concluded $\Delta I = \sigma^T$ satisfies Weber's Law,

$$\frac{\Delta I}{I}=\frac{A}{I}+B,$$

and that $\Delta f = \sigma_f$ also satisfies Weber's Law,

$$\frac{\Delta f}{f} = \frac{1}{14} \left(\frac{A}{I} + B \right),$$

where the constants A and B are independent of I and f and are the same in the two equations. We do not pursue this further because, although it may be a reasonably satisfactory way to analyze intensity discrimination, it is surely inappropriate for frequency since the Poisson assumption completely ignores the frequency information available on single channels. Fundamentally, this model denies any role to temporal mechanisms.

To overcome this limitation, Siebert (1970) undertook a more complete analysis in which temporal mechanisms were incorporated. Obviously, a count of pulses on each channel will not reveal the periodicity, so the basic sample of information on each fiber *i* must be the times $x_{i1}, \ldots, x_{i_{k_i}}$ of the

$$\begin{split} \sum_{\kappa} \int_{\mathbf{x}} \frac{\partial \ln \psi_i}{\partial \xi} \frac{\partial \ln \psi_j}{\partial \xi} \psi \, d\mathbf{x} &= \left(\sum_{\kappa_i} \int_{x_i} \frac{\partial \ln \psi_i}{\partial \xi} \psi_i \, dx_i \right) \left(\sum_{\kappa_j} \int_{x_j} \frac{\partial \ln \psi_j}{\partial \xi} \psi_j \, dx_j \right) \\ &= \left(\sum_{\kappa_i} \int_{x_i} \frac{\partial \psi_i}{\partial \xi} \, dx_i \right) \left(\sum_{\kappa_i} \int_{x_i} \frac{\partial \psi_j}{\partial \xi} \, dx_j \right) \\ &= \frac{\partial}{\partial \xi} \left(\sum_{\kappa_i} \int_{x_i} \psi_i \, dx_i \right) \frac{\partial}{\partial \xi} \left(\sum_{\kappa_j} \int_{x_i} \psi_j \, dx_j \right) \\ &= \left[\frac{\partial(1)}{\partial \xi} \right]^2 \\ &= 0. \quad \text{QED}. \end{split}$$

pulses observed during a listening interval δ . Observe that the sample size κ_i is not fixed, but rather is an RV that depends both on the underlying probability mechanism and on δ . Assuming that these processes are Poisson with a hazard function $\mu_i(x; I, f)$, then it is easy to show that the probability density of κ spikes occurring at the unordered times x_{ij} , $j = 1, \ldots, \kappa_i$, is given by

$$\psi_i(x_i,\kappa_i) = \prod_{j=1}^{\kappa_i} \mu_i(x_{ij};I,f) \exp\left[-\int_0^\delta \mu_i(x;I,f)\,dx\right] / \kappa_i!.$$

Assuming independent fibers and an efficient estimator, we obtain from Equation 9

$$(\Delta f)^2 = \frac{1}{\sum_{i=1}^n \int_0^s \frac{1}{\mu_i} \left(\frac{\partial \mu_i}{\partial f}\right)^2 dx}.$$
 (10)

To evaluate Equation 10, Siebert substituted in Equation 1 for μ_i and he assumed forms for the functions G and H which accord well with current physiological knowledge. From this, he deduced

$$\frac{1}{(\Delta f)^2} \cong 3 \times 10^6 \frac{\delta}{f^2} + 1.5 \times 10^6 \delta^3 \ln A.$$
 (11)

He then concluded that the first of these terms corresponds to the contribution of place information and the second to periodicity information. The argument is that if one assumes a spike train in which there is no periodicity information (achieved by deleting the cosine term from Eq. 1), then only the first term of Equation 11 arises; whereas, if one assumes that all of the spike trains are identical and so there is no place information (make G a constant function in Eq. 1), then only the second term arises. Choosing typical values of $\delta = 0.1 \sec$, f = 1000 Hz, and A = 300, the terms of $1/\Delta f^2$ have values of about 0.3 sec² and 10⁴ sec². Clearly, the periodicity information is far superior to place; however, the observed data of Δf approximately equal to 1 Hz are of about the same equality as the place information. And so Siebert (1970, p. 727) concludes:

1) the brain does *not* make full, efficient use of the periodicity information coded in the auditory nerve firing patterns, and

2) there *is* adequate information in the place pattern alone to account for behavior, if it is used efficiently.

If one conjectures some inefficiency in the brain, as seems plausible, the periodicity mechanisms are favored. In the following sections we show that a reasonable, but surely inefficient, periodicity decision scheme seems to yield a satisfactory prediction of both ΔI and Δf as a function of I and f.

4. HYPOTHETICAL CENTRAL REPRESENTATION AND DECISION MECHANISM

For the remainder of the chapter we use the renewal process model, with the simple probability laws given in Equations 2 through 7, rather than the Poisson model. What is complicated about this model is how intensity and frequency determine which fibers are active and how they affect the geometric probability p in an active fiber. Although complicated at the periphery, perhaps by the time the information is consolidated at a later decision stage it is simplified. We shall postulate that when it reaches the decision center the information continues to be in the form of Equations 2 through 7, but that it is simpler in two major respects.

First, we suppose that all active channels—we do not use the word 'fibers' because our assumptions are now hypothetical and functional, not necessarily anatomical—are statistically identical. This seems a relatively minor idealization in which we replace a set of different channels by the same number of identical average ones. There is no basic difficulty in dropping this assumption, but to do so would add a lot of extra baggage which, at this point, does not seem really useful. We shall continue to suppose, as is true at the periphery, that the number of active channels is a function both of intensity and frequency. In Section 7 we will be led to assumptions about that dependency.

Our second idealization is much more serious. At the periphery, pulse rates change by only a factor of from 2 to 10 over a relatively narrow intensity range for any given fiber. We have no very clear idea how this information is combined in order to give information about the full dynamic range. We shall, in any event, suppose that the combination is such that at the central mechanism p is a strictly increasing function of I. Put another way, by Equations 2, 3, and 5,

$$E(\mathbf{IAT}) = \frac{1}{pf} \tag{12}$$

is a decreasing function of I. In Section 7 we discuss more fully the form of this function which obviously must compress the physical range of 10^{10} into something manageable as firing rates. This assumption means that estimates of p, which by Equation 12 is determined by pulse rate for f fixed, provide estimates of I.

If one tentatively accepts Equations 2 through 7 together with these two idealizations as an adequate description of the form in which the information exists when decisions are made, our next task is to consider the nature of the decision rules employed. Our first assumption will be that the decision rule is sensitive only to the pulse trains and completely ignores all place informa-

tion. Of course, it takes into account the dependence of the number of active channels on intensity and frequency, for that will alter the sample sizes of IATs on which to base decisions, but the decision procedure will completely ignore *which* channels are active. This is a radical assumption, and if we are unable to account for the psychophysical data, it is surely one to be reconsidered.

Now consider possible decision rules for intensity. According to what we have assumed, intensity is reflected solely by the pulse rate, and so any rule must involve some estimate of that rate. For example, if the subject is to decide whether a more or less intense signal has been presented, the simplest rule—so simple that some feel it should not be called a decision rule—consists of just comparing the estimate of the rate with a criterion, much as in the theory of signal detectability. More complex rules involve some form of increased sampling whenever the estimate is near the criterion, but we do not explore these here (except for a sequential rule that arises naturally in our analysis of simple reaction time in Sec. 10). Obviously, a sequential rule introduces more variance into the decision time than does a criterion one, and so it will be important to decide which rule is correct if we are to give a correct account of response times. With the simple criterion rule, the only remaining theoretical question is how the pulse rate is estimated. There are two extreme ways to do this. One is to fix a time interval and to count the total number of pulses that arrive during that time on all of the active channels; the estimated rate is the mean count per channel. Another is to fix a count per channel and to time how long it takes for that number of pulses to arrive on each channel; the estimated rate is the reciprocal of the mean time per IAT per channel. Using the former rule, one counts pulses; using the latter, one times IATs. For this reason, models based on the former are called *counting* ones (McGill, 1967), and those based on the latter are called either *timing* (Luce & Green, 1972) or clocking models (Uttal & Krissoff, 1965).

One might guess that these two estimation schemes would make little difference in behavior but, as we show below, that guess is wrong. Moreover, it appears that both rules are available to subjects. To establish these points, consider an absolute identification experiment in which one of two tones of different intensities is presented on each trial and the subject is to identify which was presented. Suppose further that each signal remains on until he responds (so that, from a theoretical point of view, we do not have to worry about running out of signal), but let us put the subject under pressure to respond rapidly as well as accurately. The time pressure can be effected by a deadline with fines for late responses, and accuracy can be affected by a payoff matrix. We analyze this experiment using both the counting and timing rules.

First consider the mean response time. It is composed of two parts, the mean decision delay and the mean of all other delays. Let the latter be denoted

by \bar{r} . It is evident that, according to the counting rule based on a fixed listening interval δ , the decision time is independent both of the signal and the response. Since \bar{r} is also, we predict that the MRT = $\bar{r} + \delta$ is the same in all four cells of the signal-response matrix. It is equally evident that according to the timing rule, the more intense signal should have faster response times than the less intense one because of the difference in pulse rates. In fact, let us suppose that κ IATs are collected per channel and that J channels are active. Then the distribution of decision times is the slowest of J samples from the distribution of the sum of κ independent IATs. If for signal i = 1 or 2 we write $M_i = E(IAT_i)$ and $V_i = V(IAT_i)$, it is easy to see that

$$MRT_{i} = \bar{r} + h(J, \kappa, V_{i}^{1/2}/M_{i})M_{i}, \quad i = 1, 2,$$
(13)

where $h(J, \kappa, \sigma)$ is the mean decision time when E(IAT) = 1 and $V(IAT) = \sigma^2$. We do not need to derive the form of $h(J, \kappa, \sigma)$ here, although we will do so in the next section. Suppose that $M_1 > M_2$, then eliminating $h(J, \kappa, \sigma)$ from the equation (we assume that the differences in intensity are so small that we can neglect the differences in J and σ),

$$MRT_{1} = \left(\frac{M_{1}}{M_{2}}\right) MRT_{2} - \bar{r}\left(\frac{M_{1}}{M_{2}} - 1\right)$$
(14)

This we can test by varying the deadline and plotting the two signal MRTs against one another, checking for linearity and the value of the slope.

A second prediction is obtained from the two models by considering the ROC curves [plot of $P(1 | s_1)$ against $P(1 | s_2)$] obtained with a fixed deadline. To carry out this analysis we assume that the expected value M and variance V of the IAT distribution are related so that both V and M^3/V are strictly increasing functions of M. This is true, for example, in the exponential case where $V = M^2$ and for Equations 2 through 7 provided⁴ $p \leq \frac{1}{3\theta} [1 - (3\theta)^{1/2}]$.

Consider the timing rule first. Assuming that J_{κ} is large, the central limit theorem implies that the sum of the IATs is approximately normally distributed with mean $J_{\kappa}M$ and variance $J_{\kappa}V$. Using the probability cutoff of c, the corresponding z score is

$$z=\frac{c-J\kappa M}{(J\kappa V)^{1/2}}.$$

⁴ Observe, $M = E(IAT) = \frac{1}{pf}$ and $V = \frac{q + p^2\theta}{p^2f^2} = M^2(q + p^2\theta)$ and so $\frac{M^3}{V} = \frac{1}{p(q + p^2\theta)f}$ For f fixed, M increases as p decreases. As is easily shown by computing its maximum, $p(q + p^2\theta)$ decreases with p for $p \le \frac{1}{3\theta} [1 - (1 - 3\theta)^{1/2}]$ and V increases everywhere. For $\theta = 0.2$, this bound is 0.61, which covers the known data.

Considering two signals with mean IATs of $M_1 > M_2$ (i.e., the second signal is more intense), the ROC curve (in z scores) is obtained by eliminating c:

$$z_2 = \left(\frac{V_1}{V_2}\right)^{1/2} z_1 + \frac{(M_1 - M_2)}{V_2^{1/2}} (J\kappa)^{1/2}.$$
 (15)

Since V is a strictly increasing function of M, the slope of this ROC curve is > 1. In the exponential case, it is M_1/M_2 .

To work out the prediction for the counting model, we note that in a renewal process, the number N of pulses counted in a time δ per channel is, asymptotically as $J\delta \longrightarrow \infty$, given by

$$E(\mathbf{N}) = J\delta/M, \qquad V(\mathbf{N}) = J\delta V/M^3$$

(Parzen, 1962, p. 180). So the z score is of the form

$$z = \frac{c + E(\mathbf{N})}{V(\mathbf{N})^{1/2}} = \frac{(c + J\delta/M)M^{3/2}}{(J\delta V)^{1/2}}$$

whence the ROC curve is

$$z_{2} = \left[\frac{V_{1}}{V_{2}}\left(\frac{M_{2}}{M_{1}}\right)^{3}\right]^{1/2} z_{1} + (J\delta)^{1/2} \left(1 - \frac{M_{2}}{M_{1}}\right) \left(\frac{M_{2}}{V_{2}}\right)^{1/2}$$
(16)

Since M^3/V is an increasing function of M, the slope is < 1. In the exponential case it is $(M_2/M_1)^{1/2}$.

Green and Luce (1973; also see Luce, 1972) ran the above experiment using a 1000 Hz signal in noise and noise alone. When the deadline was varied, the MRTs were virtually identical except for the very long deadlines (see the next section), and with the deadline fixed and the payoffs varied, the ROC curve was linear with slopes of 0.92, 0.90, and 0.69 for three subjects. Thus, the counting rule is more appropriate for this experiment than the timing one. When, however, the experiment was slightly altered so that the deadline applied only to those trials on which the more intense signal was presented, then both the MRT and ROC curves were approximately linear and the pairs of MRT and ROC slopes for three subjects were: (1.34, 1.30), (1.48, 1.47), and (1.38, 1.37). Obviously, the timing rule is now more appropriate than the counting one. Moreover, the close agreement of the values suggests $V = M^2$, in which case the exponential distribution is approximately correct.

Obviously, considerable care is going to have to be taken both experimentally and theoretically to make sure whether a timing or counting model is appropriate in any given context. Our experience to date with these models suggests that the timing ones are generally more plausible except in situations when it is distinctly to the subject's advantage to employ counting behavior. Such an advantage obtains when we impose a uniform deadline on all responses, as just discussed, and when we use very brief signals whose presence is well marked, as is typical of many psychophysical designs.

5. SPEED-ACCURACY TRADE OFF

The contrast between the counting and timing decision rules is nicely illustrated by the different trade off they predict between speed and accuracy. Speed is, of course, measured by MRT. Accuracy can be measured in several ways. Provided that the data are fairly linear on an ROC plot in z scores, one of these is d', i.e., the z_2 coordinate of the ROC curve corresponding to $z_1 = 0$. We see from Equation 16 that for the counting model

$$d' = A\delta^{1/2},\tag{17}$$

where

$$A = J^{1/2} \left(1 - \frac{M_2}{M_1} \right) \left(\frac{M_2}{V_2} \right)^{1/2}$$
 (18)

As the listening interval δ is increased by manipulating the deadlines, both d'and MRT = $\bar{r} + \delta$ should increase with the trade off being of the form

$$d' = \begin{cases} A(MRT - \bar{r})^{1/2}, & MRT \ge \bar{r}, \\ 0, & MRT < \bar{r}. \end{cases}$$
(19)

In like manner, Equation 15 yields for the timing model

$$d' = \frac{M_1}{M_2^{1/2}} A \kappa^{1/2}, \qquad (20)$$

where A is given in Equation 18, and the expression for MRT was derived in Equation 13. Again, increasing the deadline should increase κ and, hence, both d' and MRT. To derive the exact form of the trade off, we must see how $h(J, \kappa, \sigma)$ depends on κ . Assuming a train of pulses with mean 1 and standard deviation σ for the IATs, and letting ψ_{κ} denote the distribution of the (κ + 1)st pulse, we have by definition

$$h(J, \kappa, \sigma) = J \int_0^\infty x \psi_{\kappa}(x) \left[\int_0^x \psi_{\kappa}(y) \, dy \right]^{J-1} dx.$$

Assuming that ψ_{κ} is approximately normal, which by the central limit theorem it certainly is when κ is large, it is easy to see that

$$h(J, \kappa, \sigma) \cong \kappa + 1 + (\kappa + 1)^{1/2} \sigma H(J),$$

where H(J) is the mean of the largest of J normally distributed RVs with mean 0 and variance 1. Selected values are H(2) = 0.56, H(10) = 1.54, H(100) = 2.51, and H(1000) = 3.24. Substituting in Equation 13,

$$MRT_{i} = [\bar{r} + M_{i} + V_{i}^{1/2}H(J)] + M_{i}\{\kappa + [(\kappa + 1)^{1/2} - 1](V_{i}^{1/2}/M_{i})H(J)\},$$
(21)

where we have written the right side so that the first term on the right corresponds to the irreducible minimum MRT when $\kappa = 0$. Since $\kappa = 0$ implies no information (d' = 0), the first term also describes the intercept in an equation relating speed and accuracy. This suggests introducing the variable

$$T_i = \mathrm{MRT}_i - \bar{r} - M_i - V_i^{1/2} H(J),$$

solving for κ in Equation 21, and substituting that in Equation 20, which yields

$$(d')^{2} = \begin{cases} \frac{M_{1}^{2}}{M_{2}M_{i}} A^{2} \left\{ T_{i} + \frac{M_{i}B_{i}}{2} \left((B_{i} + 2) - \left[(B_{i} + 2)^{2} + \frac{4T_{i}}{M_{i}} \right]^{1/2} \right) \right\}, \\ T_{i} \ge 0, \quad (22) \\ 0, \qquad \qquad T_{i} < 0 \end{cases}$$

where

$$B_i = V_i^{1/2} H(J) / M_i.$$

Comparison of Equations 19 and 22 reveals three differences in the two models. First, in the counting model the function starts at \bar{r} , whereas in the timing model it begins later, at $\bar{r} + M_i + V_i^{1/2}H(J)$. Second, in the counting model there is only a single function since MRT₁ = MRT₂, whereas in the timing model there are two distinct functions corresponding to i = 1, 2. And third, in the timing model the initial growth of the function corresponding to the weaker signal (i = 1) is $(M_1/M_2)^{1/2}$ times that of the counting model and that of the stronger signal is that much again, or M_1/M_2 , times that of the counting for the timing model should begin later than that of the counting model, but it should grow considerably more rapidly.

Because we collected data for a complete ROC curve at only one deadline, the values of d' at other deadlines were inferred by passing a line with the one estimated ROC slope through the single observed point at each deadline. According to both the counting and the timing models (Eqs. 15 and 16) the ROC slope should be independent of the deadline (δ or κ), so this method of estimation does not favor either model.

The data are shown in Figure 3. The first panel includes the data of three observers run with the deadline applied on all trials. The trade off appears to be substantially the same for all three, and the value of \bar{r} is about 150 msec. The growth of the function is more nearly linear than the $\frac{1}{2}$ power of MRT $-\bar{r}$, as was predicted; we return to this discrepancy shortly. The other three panels show another three observers run with the deadline applied only to the signal, i.e., to i = 2. Again, they are similar to each other: the inter-

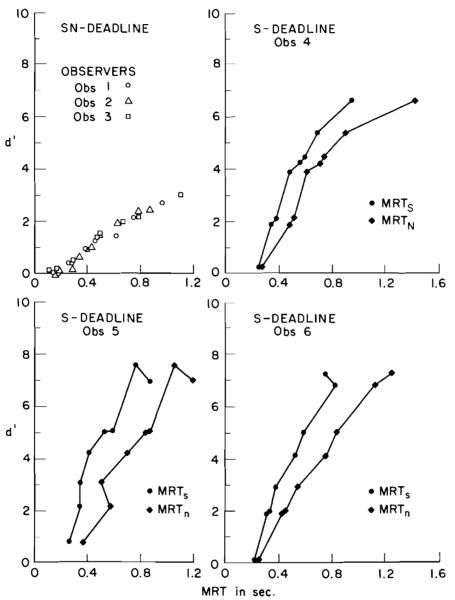


FIGURE 3. Plots of d' versus MRT. The upper left panel combines the data for three observers when the deadline applies to all trials. The other three panels, one per observer, plot d' versus both MRTs and MRT_n when the deadline applies only to signal trials. (Green & Luce, 1973, Fig. 11.)

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cepts of the functions are well over 200 msec and all of the functions rise distinctly more rapidly than in the first panel. Qualitatively, at least, these results are in excellent accord with the predictions of, respectively, the counting and timing models.

A direct numerical comparison is not possible for several reasons. First, different observers were used in the two experiments. Second, the predicted speed-accuracy trade off for the timing model requires estimates of M_i , V_i , and J, which we do not have. And third, the predicted square-root form for the speed-accuracy trade of the counting model was not substantiated by the data. There are at least two possible reasons for this discrepancy. First, estimates of d' near 0 are very unstable, and it is conceivable that the small d's in the region of 150 to 200 msec are in fact all really zero, in which case the square-root growth is not a bad approximation. Such an explanation is, however, inconsistent with our conclusion that the intercepts for the two experiments are different. Second, for long durations it is clearly to the subject's advantage to switch from a counting to a timing mode: he will thereby increase his accuracy. There is some suggestion of such a change in that, at the longest deadlines, the difference between MRT_1 and MRT_2 is not zero. For example, at 2000 msec it is 56, 65, and 9 msec for the three subjects. This suggests that at least two of the subjects were mixing the counting and the timing modes at the long deadlines. The degree of departure is, however, less than one would expect if they were trying to optimize their accuracy payoffs.

A number of authors, using various techniques to manipulate or classify response times, have examined the speed-accuracy trade off. Taylor, Lindsay, and Forbes (1967) predicted that d'^2 should be approximately linear with MRT and they confirmed it in data of Schouten and Bekker (1967). Other closely related studies are those by Fitts (1966), Lappin and Disch (1972), Pachella and Fisher (1972), Pachella, Fisher, and Karsh (1968), Pachella and Pew (1968), and Pew (1969).

In the case of readily detectable signals, Ollman (1966) and Yellott (1967) proposed a quite different, two-state model to account for the speed-accuracy trade off. They assumed that the subject chooses on each trial either to respond to the signal onset, in which case he is fast and inaccurate (chance level), or to wait until the signal is positively identified, in which case he is slow and accurate. By varying the probability of waiting, a trade off is effected. This is known as the *fast-guess* model. Without formalizing the model and working through the algebra, the following relation can be derived. Letting c (for correct) denote the 11 and 22 cells and e (for error) the 12 and 21 cells in the stimulus-response matrix, then

$$P_c MRT_c - P_e MRT_e = A(P_c - P_e),$$

where A is a constant and

$$P_{e} = (\frac{1}{2})[P(1 \mid 1) + P(2 \mid 2)],$$

$$P_{e} = (\frac{1}{2})[P(2 \mid 1) + P(1 \mid 2)].$$

We evaluate the same quantity for our two models, again omitting the algebra. For the counting model,

$$P_c MRT_c - P_e MRT_e = (\ddot{r} + \delta)(P_c - P_e).$$

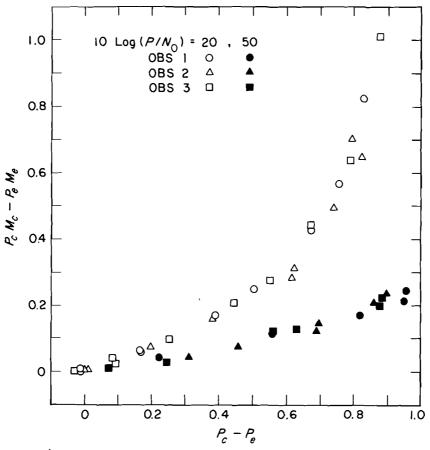
Since $P_c - P_e$ increases with δ , we see that an accelerated function is predicted rather than a linear one. For the timing model,

$$P_{c}MRT_{c} - P_{e}MRT_{e} = \bar{r}(P_{c} - P_{e}) + h(J, \kappa, \sigma) \{M_{1}[P(1 \mid 1) - \frac{1}{2}] + M_{2}[P(2 \mid 2) - \frac{1}{2}]\}$$

The first term is simply the linear prediction on the fast-guess model, and the second one increases with κ and so with $P_c - P_e$, again yielding an accelerated function. Observe, however, that unlike the counting model, the degree of acceleration is a function of signal strength, M_i . For intense signals (small M_i), it is negligible and so we should obtain the linear relation; for weak ones, however, it should be convex.

The data for both weak and intense signals in the experiment with a deadline imposed on all trials (both i = 1 and 2) are shown in Figure 4, and the data for weak signals in the experiment with a deadline only on the more intense signal (deadline for i = 2 only) are shown in Figure 5. Obviously, the fast-guess model is wrong for weak signals since, for both types of deadline, the curves are decidedly convex. Qualitatively, both sets of data for weak signals agree with both the counting and timing models. The only data we have for intense signals come from the experiment where, for weak signals, we concluded earlier that the counting model held. The strong signal data appear linear and hence are consistent with only the timing model. It appears, therefore, that with increasing signal strength there is a tendency to switch into the timing mode. Presumably this is the more natural mode of behavior, and there is little advantage to using counting when the maximum number of IATs that can be collected per channel has a high probability of being within the deadline for either signal because M_i is so small.

Our tentative conclusion is that subjects use the timing rule except when it is decidedly disadvantageous to do so, in which case they shift to the counting one. Examples of experiments where the timing rule is disadvantageous are those involving weak signals that are either of short duration or to which fast responses (and so a short sample of the signal) are encouraged. As we shall see below, several other types of experiments appear to be accounted for by timing rules.





Fast-guess analysis of speed-accuracy data when the deadline applies to all trials. The open points are for weak signals; the solid ones, for strong signals. (Green & Luce, 1973, Fig. 2.)

6. DISCRIMINATION FUNCTION FOR INTENSITY

One ultimate test of a theory of neural coding is its ability to account for psychophysical data on the limits of discriminability for small changes in both the frequency and the intensity of a sinusoidal signal. As we show here and in Section 8, the theory does indeed make such predictions. Although one does not anticipate any difficulty in comparing these predictions with the empirical data—since surely after a century of study these basic relations are

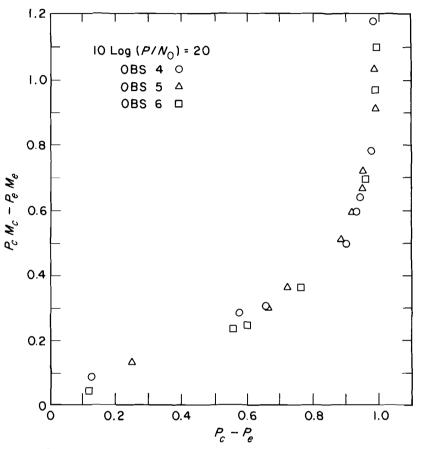


FIGURE 5.

Fast-guess analysis of speed-accuracy data (weak signals) when the deadline applies to signal trials. (Green & Luce, 1973, Fig. 5.)

well established and generally accepted—nothing could be further from the truth.

It has long been known that a variety of intensive stimuli obey Weber's in the form

$$\frac{\Delta I}{I} = \frac{A}{I} + B, \tag{23}$$

where I denotes a reference intensity, ΔI the deviation from I that is necessary to achieve a fixed level of discriminability, and A and B are constants. Hawkins and Stevens (1950) showed that the signal energy to noise power density needed for a sinusoid to be just detectable in wideband noise is re-

markably constant over a range of about 90 dB. Miller (1947) showed that the just detectable increment in a wideband noise is some constant fraction of the background noise once the background is slightly above the absolute threshold. On the other hand, the detection of an intensity increment in a sine wave has never exhibited this relation over any reasonably large range of intensity. Rather, if one plots $\Delta I/I$ versus *I*, the function begins at some large value and generally decreases as the intensity of the background *I* is increased (Campbell & Lasky, 1967; Dimmick & Olsen, 1941; Green, 1967; McGill & Goldberg, 1968; Reisz, 1928; Viemeister, 1972).

McGill and Goldberg (1968) were the first to discuss carefully this deviation from Weber's law—what they call the near miss to Weber's law—and they pointed out that it is well approximated for large I by an equation of the form

$$\frac{\Delta I}{I} = BI^{r-1}, \qquad (24)$$

where B and c are constants independent of I. The data are fit with c about 0.90. The explanation they offer is that the small deviation from Weber's law arises because of the nonlinear dependence of the number of neural spikes in a counting model as a function of intensity.

Another interpretation of these data has recently been suggested by Viemeister (1972), who seems to have shown empirically that the reason for the deviation from Weber's law is wholly different from a neural explanation. He noted that as intensity is increased, the inherent nonlinearity of the ear introduces amplitude distortion contributions at the various harmonics of the signal. At low intensities, the energy at each harmonic is considerably smaller than at the next lower harmonic. In fact, at sufficiently low intensities, all harmonics higher than the fundamental are inaudible. However, as the intensity of the fundamental is increased, the rate of growth is faster the higher the harmonic. For example, if the distortion follows a square law, then the amplitude of the second harmonic grows as the square of the amplitude of the primary. And so a 1 dB change in the primary produces a 2 dB change in the second harmonic. Thus, at the point at which the second harmonic becomes audible, it is better to use it in making discriminations because the change in dB is larger there than at the fundamental. Similar arguments apply to the other harmonics—an n dB change occurs at the nth harmonic. Thus, as the stimulus is increased in intensity, if the subject moves from harmonic to harmonic as each becomes audible, he will clearly do better than Weber's law even if that law applies to discrimination of the primary.

Viemeister analyzed existing data on amplitude distortion to see if this argument is viable, and it is. So he performed the following direct test of it. If this is what the subjects are doing and if one can make it impossible for them to do so, then they should exhibit Weber's law. He used high-pass masking noise (with its boundary above the fundamental but below the first harmonic) to obscure the harmonics, and found Weber's law to be almost exactly satisfied. Despite the fact that his experiment has not been replicated, we find the basic logic compelling. Nonetheless, we work out the predictions of the present theory. A more complete discussion, a counting alternative emphasizing and using data other than Reisz' is given by Luce and Green (1974).

We now compute how ΔI depends on I and f in the timing model. Usually ΔI is defined to be that stimulus increment producing 75 percent correct detections in a two-alternative forced-choice design, and this is very close to $d'_I = 1$. From Equation 15 we know that d'_I is given by

$$d'_{I} = (J\kappa)^{1/2} \left(\frac{M_{I} - M_{I+\Delta I}}{V_{I}^{1/2}} \right)$$

From Equations 2 through 7,

$$M = E(IAT) = 1/pf$$

and

$$V = V(IAT) = (q + p^2\theta^2)/p^2f^2$$
$$\cong q/p^2f^2$$
$$= qM^2,$$

where q = 1 - p and $\theta \simeq 0.2$ is a measure of the smearing of the modes (see Eq. 6). For $p \le \frac{1}{2}$, the approximation is obviously good. Substituting and setting d' = 1 yields

$$\frac{\Delta p}{p} = \frac{1}{(J\kappa/q)^{1/2} - 1}.$$
 (25)

To calculate ΔI , we must first determine how *p* depends on *I*. If we approximate the IAT distribution by an exponential (parameter μ) displaced by the amount 1/f from the origin, then equating means it is easy to see that

$$\frac{1}{1-p} = \frac{\mu}{f}$$

As we show empirically in Section 10, reaction-time data strongly suggest that μ is a power function of intensity—at least for low intensities. Letting p_0 be the probability corresponding to the threshold intensity I_0 at frequency f, we may therefore rewrite the above equation and the empirical hypothesis as

$$\frac{p}{1-p} = \frac{p_0}{1-p_0} \left(\frac{I}{I_0}\right)^{\gamma}.$$
 (26)

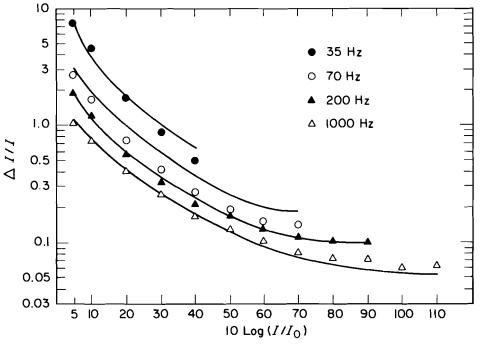


FIGURE 6.

 $\Delta I/I$ versus I with f as a parameter. The data are taken from Reisz (1928). The theoretical curves are Equations 27 through 30 with the following parameter values: $\gamma = 0.20$, $\eta = 0.10$, $A_0 = 0.60$ sec, $\kappa_0 = 1$, $\kappa_m = 25$, $J_0 = 17.10$, B = 1.246.

It then follows readily that

$$\frac{\Delta I}{I} = \left[\frac{1}{1 - 1/(qJ_{\kappa})^{1/2}}\right]^{1/\gamma} - 1.$$
(27)

Thus, we now need to see how qJ_{κ} depends on *I* and *f*. For the present we simply write down the three equations that we use in our calculations; their partial justifications are provided in the next section.

$$p = \frac{(I/I_0)^{\gamma}}{A_0 f - 1 + (I/I_0)^{\gamma}}, \quad q = 1 - p = \frac{A_0 f - 1}{A_0 f - 1 + (I/I_0)^{\gamma}}.$$
 (28)

$$J = J_0(\log_{10} f - B)(I/I_0)^{\eta}.$$
 (29)

$$\kappa = \begin{cases} (\kappa_0 + 1)A_0 pf - 1, & \kappa \le \kappa_m, \\ \kappa_m, & \text{otherwise.} \end{cases}$$
(30)

Using the parameter values shown in the caption of Figure 6 and substituting Equations 28 through 30 into Equation 27 yields the family of curves shown in Figure 6. The data points are those of Reisz. We discuss some of the choices of the parameter values as we 'derive' Equations 28 through 30.

7. THE DEPENDENCE OF p, J, AND κ ON I AND f

To arrive at the preceding three expressions, we make three rather speculative hypotheses which have very incomplete experimental support.

1. The neural criterion of activity in a channel, and hence the criterion for a sensory threshold, is characterized by $E(IAT) = A_0$, where A_0 is a constant that has to be estimated.

In whatever way the nervous system decides whether or not a channel is active, it seems plausible that the decision is based solely on sample of activity from that channel. Since the only information available from a renewal process is contained in the IATs, the criterion must be based on them and certainly one of the simplest is merely to use the mean IAT. If this were true, then except for a possible small effect due to changes in J with I and f (Eq. 29), the MRT at threshold should be independent of f. The small amount of data that we have collected seem to support this; however, a more extensive study is needed.

Using the fact (Eq. 12) that E(IAT) = 1/pf and the hypothesis that at threshold $E(IAT) = A_0$, Equation 28 follows immediately from Equation 26.

The only real difficulty we have had in choosing acceptable parameter values is with A_0 . According to Equation 21,

$$MRT \geq \bar{r} + A_0(1 + \kappa_0).$$

Using $\kappa_0 = 1$ and $A_0 = 600$ msec, as in Figure 6, and (from Sec. 5 or from simple RT data with intense signals) $\bar{r} = 150$ msec, we see that MRT is not smaller than 1350 msec—in fact, it is considerably larger since the inequality is crude. Our RT data at threshold suggest a value of about 1000 msec. Of course, our data were collected some 45 years after Reisz' and used a very different discrimination technique, so we cannot be certain whether or not there is a real difficulty.

Our second hypothesis is:5

⁶ Note added in proof. Since writing this paper, our views on modeling discrimination data have changed in important ways. First, Reisz' data resulted from an unusual experimental procedure, one not particularly well modeled by the present considerations, and unlike all later intensity discrimination data they exhibit a strong dependency on frequency. Second, the later data all used relatively brief duration signals, suggesting that a counting model is appropriate. Luce and Green (1974b) fit the obvious one, using hypothesis 1 and constant values for δ and J; they had no need, therefore, for hypotheses 2 and 3. Third, and perhaps most important, a detailed study of magnitude estimates reported by Green and Luce (1974) suggest that the situation is actually somewhat different from, and much more interesting than, the conjectures that follow. See footnote 6 and also Luce and Green (1974a).

2. When a subject is asked to magnitude estimate a signal, he collects κ IATs on each channel, forms the reciprocal of the total time on each channel, adds these numbers over all active channels, and then multiplies that by a constant and emits the result as his magnitude estimate.

This hypothesis is a special case of the more basic hypothesis that the subject interprets magnitude-estimation instructions as a request to estimate and report the neural pulse rate. If so, there are a number of ways in which it might be estimated. For example, we assumed earlier (Luce & Green, 1972) an exponential IAT distribution and that the times were summed over channels prior to forming the reciprocal. In that model, magnitude estimates are reciprocals of a RV with a gamma distribution of order J_{κ} . Because J_{κ} is necessarily large, the resulting distribution should have a stable mean and variance. Data we have collected make us doubt whether the variance really is very stable. This could arise if κ were small, 2 or 3, and if the reciprocal were formed on each channel individually before summing. For example, with $\kappa = 2$ the theoretical variance of the reciprocal of a second-order, gammadistributed variable does not exist. The trouble arises, of course, because the exponential places so much density at 0 which the reciprocal maps into ∞ . These observations have led us to think, first, that maybe only a very small number of IATs are collected on each channel; second, that the reciprocal is computed for each channel separately; and third, that for theoretical ME calculations the exponential distribution is a very poor approximation to Equation 7. Some indirect support to the idea that only a few IATs are collected per channel is provided by the studies of Stevens (1966) and Stevens and Hall (1966), in which it was shown that auditory magnitude estimates reach their maximum value when the signal duration is about 150 msec.

In working out the magnitude-estimation distribution using the IAT model of Equations 2 through 7 and this hypothesis, we make the simplifying assumption that X can be neglected. This introduces some error in E(ME) and, were we to compute it, a rather sizable error in V(ME), especially when p is large. With this approximation, we compute the expectation of f/I where I is an integer-valued random variable whose distribution is the convolution of κ geometric distributions, each with its origin at 1 rather than 0. As the calculations are routine, we exhibit only the mean (over J channels) for the first three values of κ :

$$E(ME) = Jf\left(\frac{p}{1-p}\right) \begin{cases} \ln\frac{1}{p}, & \kappa = 1, \\ 1 - \left(\frac{p}{1-p}\right)\ln\frac{1}{p}, & \kappa = 2, \\ \frac{1}{2} - \left(\frac{p}{1-p}\right)\left[1 - \left(\frac{p}{1-p}\right)\ln\frac{1}{p}\right], & \kappa = 3. \end{cases}$$

Since empirically E(ME) is approximately a power function of I, with an exponent of about 0.3 in the case of loudness, since the dominant term appears to be Jfp/(1-p), and since p/(1-p) is a power function (Eq. 26) we are led to conjecture that

$$J \propto (I/I_0)^{\eta},$$

where $\gamma + \eta = 0.3$, a constraint we maintain in our choice of constants.

The further assumption that the effect of intensity and frequency contribute multiplicatively is pure speculation, and the choice of a logarithmic dependence on f in Equation 29 is entirely empirical.

Our final hypothesis is:

3. The number of IATs collected on each channel is adjusted so that the total time consumed is approximately a constant up to some limit, κ_m , determined by the size of a buffer store for IATs.

It seems plausible that the nervous system either holds κ constant independent of frequency or makes some sort of adjustment to maintain approximately constant times. The model does not fit the data if κ is kept constant, so we were led to hypothesis 3. Of course, if the intensity levels were randomized, one suspects that a constant κ would result.

Letting κ_0 denote the threshold value of κ , it follows that, since the number of pulse is one more than the number of IATs, for $\kappa \leq \kappa_m$,

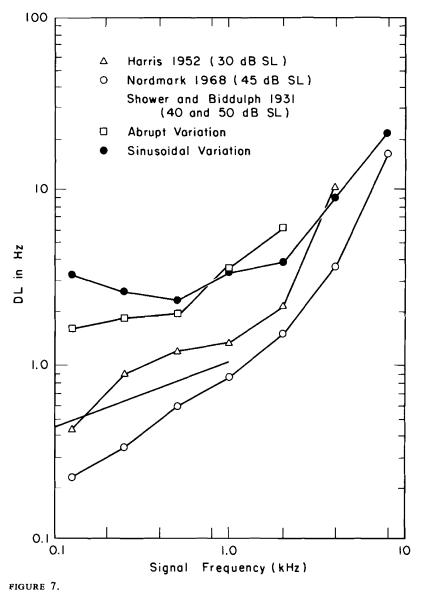
$$(\kappa_0 + 1)A_0 = (\kappa + 1)E(IAT) = (\kappa + 1)/pf,$$

from which Equation 30 is an immediate consequence.

8. DISCRIMINATION FUNCTION FOR FREQUENCY

The data on the discrimination of small changes in frequency are, if anything, less satisfactory than those for intensity. Three major papers (Harris, 1952; Nordmark, 1968; Shower & Biddulph, 1931) summarize how Δf varies with f and I (but only for 30 dB SPL in Harris and 45 dB SPL in Nordmark). The results at comparable SPL and low frequencies differ by almost an order of magnitude. It is still not known what differences in method are crucial to these differences in performance. There is one optimistic note in that, although the values of Δf differ considerably, their ratios at different frequencies are nearly the same. Figure 7 presents these data.

At high frequencies the agreement is better, but Henning (1966) has challenged all of these high-frequency measurements as being seriously influenced by artifacts. He demonstrated that above 4000 Hz, the discrimination of a



 Δf versus f. The data are from the sources shown. The theoretical curve is Equation 37 with the parameters given in the caption of Figure 5, $\theta = 0.20$, and $I/I_0 = 45$ dB.

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frequency change is probably based largely on a change in loudness. The evidence is that when the intensity of the two tones is randomized, the accuracy of frequency discrimination deteriorates markedly. For example, at 8000 Hz the jnd is 16 Hz when the intensities are fixed and 100 Hz when they are randomized. No comparable change was observed at low frequencies, but one would not expect any since a change in frequency at low frequencies does not alter the amplitude of the signal appreciably.

The only study of Δf over the whole (I, f) plane that we know of is Shower and Biddulph's. Since this study is the earliest, it exhibited the poorest discrimination, and the technique of signal variation employed is very difficult to model, we remain very unsure how Δf in its usual sense depends on I.

Turning to theory, we first recall that Wever (1949) argued from various sorts of data that place mechanisms probably account for the discrimination of frequencies above approximately 2000 Hz and periodicity mechanisms probably are involved for lower frequencies. Such a break is almost certainly enforced by the inability of a nerve to fire faster than about every $\frac{1}{2}$ msec. Turning to our low-frequency model given in Equation 1, we see that if I were known, then

$$\mathbf{T} = \frac{\mathbf{IAT}}{\mathbf{I}} = \frac{1}{f} + \frac{\mathbf{X}}{\mathbf{I}}$$
(32)

estimates the period, 1/f, of the input signal. It is equally clear that given a reasonably large sample of IATs—one from each active channel should do—the variability due to X is so small that I can be determined correctly for each IAT. (This would not be true if the modes of the IAT distribution overlapped.) If we let \overline{T} be the average value of T over J_{κ} IATs, then it is easy to show (Luce & Green, 1974) that

$$E(\overline{\mathbf{T}}) = 1/f,$$

$$V(\overline{\mathbf{T}}) = \left(\frac{\theta}{f}\right)^2 \frac{p}{q} \frac{\phi(q)}{J_{\kappa}},$$

where

$$\phi(q) = \sum_{i=1}^{\infty} q^i/i^2 = -\int_0^q \frac{\ln(1-x)}{x} dx.$$

Defining d'_{t} as the difference in means divided by their (approximately common) standard deviation,

$$d_f' = \left(1 - \frac{f}{f + \Delta f}\right) \frac{1}{\theta} \left(\frac{J\kappa}{p\phi(q)/q}\right)^{1/2}$$

Solving for Δf with $d'_f = 1$ yields

$$\Delta f = \frac{f\theta[\phi(q)/q]^{1/2}}{(J\kappa/p)^{1/2} - \theta[\phi(q)/q]^{1/2}}$$

$$\cong \frac{f\theta[\phi(q)/q]^{1/2}}{(J\kappa/p)^{1/2}},$$
(33)

where the approximation is justified because $\theta \cong 0.2$ and $[\phi(q)/q]^{1/2}$ has $\pi/6^{1/2}$ as its maximum value. Substituting p from Equation 28, J from Equation 29, and κ from Equation 30 in Equation 33, and using the constants listed in the caption of Figure 6 together with $\theta = 0.20$ yields the theoretical curve shown in Figure 7.

It is worth noting that the portion of the theoretical curve for low intensities decreases with increasing intensity; however, after κ reaches κ_m frequency discrimination for large f is predicted to deteriorate slowly with intensity. There are recurrent indications of this in Henning's (1967, Fig. 3) work, but it has never been carefully studied.

We conclude that the same set of constants is adequate to account for frequency as well as intensity discrimination.

9. OTHER SOURCES OF VARIABILITY

Up to this section, we have attempted to account for behavior in terms of the sum of three sources of variability: that due to the stimulus itself, that arising from its transduction into neural impulses, and that occurring during the transmission of these neural impulses up to the point at which the decision is made. Specific assumptions about the nature of these kinds of variability allowed us to predict a variety of classical psychophysical data. In particular, most of the data for simple discrimination and their corresponding response latencies can be analyzed from this point of view. Whether or not *all* simple discriminative behavior can be dealt with so simply is problematical, but many theorists, including us, are optimistic.

We would be remiss, however, to leave the impression that this is the only variability that ever arises or, indeed, that this approach can be extended to more complicated psychophysical tasks without introducing some fundamentally new assumptions. Although a full discussion of this topic is not appropriate here, we will review briefly some work both because it is timely and relevant and because it suggests that variability in applying the decision rule itself is necessary to explain some psychophysical behavior.

Consider an absolute identification, or categorization, experiment in which one of N signals is presented on each trial and the subject attempts to identify which it is. Assuming that the process is stationary and independent from trial to trial, then the basic data can be presented in an $N \times N$ matrix of probabilities where the *ij* entry is the relative frequency of the *j*th response when the *i*th signal is presented. In most such studies the signals are chosen from some ordered continuum, such as intensity level of a pure tone, and the responses are the first N integers assigned to the signals so that their natural order corresponds to the order of the sensory continuum.

One question is how to measure accuracy so we can study how accuracy depends on the number, spacing, and range of the signals. The most obvious measure, percentage of correct responses, has the drawback that as N increases it decreases without in any way taking into account how many responses were almost correct—it is insensitive to the magnitude of the errors. More satisfactory measures use more than just the main diagonal of the data matrix. Perhaps the most famous of these is the information measure; below we will describe a d'-type of measure.

A remarkable, but ubiquitous, finding of this area is that the information transmitted grows with N up to about N = 7, at which point it reaches an asymptote or, perhaps, a maximum (Miller, 1956). Note that we have stated this entirely in terms of the number of signals without qualification as to their spacing or range. To a good first approximation, such qualification is immaterial: various signal spacings and ranges have been studied and the results are surprisingly insensitive to those choices, except when the range is small, say, less than 20 dB in auditory intensity.

Recently, Durlach and his collaborators (Braida & Durlach, 1972; Durlach & Braida, 1969; Pynn, Braida, & Durlach, 1972) have embarked on a series of experimental and theoretical investigations of this and related paradigms. Although their data-analysis procedure is somewhat unconventional—a modified version of Thurstone's successive intervals rather than, for example, information theory—their main findings are consistent with earlier studies. Their basic measure is obtained by computing over all responses (with estimated probabilities not too near 0 or 1) the values of d' for each pair of adjacent signals. The sum of these values over all adjacent pairs of signals is their overall measure of accuracy. Among other things, they find that if N is held fixed and the signal range is increased, this measure reaches an asymptote.

To account for this at a theoretical level they assume that the variance which enters into the computation of d' for each pair of adjacent stimuli has two independent sources. The one is the usual variability in the representation of the signal, which is what we have discussed in the bulk of the chapter. The other source they interpret as due to fallible memory; e.g., it might result from variability in the location of category boundaries from trial to trial. Their crucial new assumption is not that imperfect memory introduces variability, but that this variance is proportional to the entire square of the range, measured in dB, of the signals being used. Why this should be is not obvious.

To make their theory quite explicit, we list its three principle features. First, signals are represented as normally distributed random variables with con-

stant variance (the latter is not true in a pulse model). Second, the mean of the signal random variable is assumed to be a logarithmic function of signal intensity (this also is not true in our pulse model, where a power function growth was postulated). Third, in computing d' the effective variance consists of the sum of the constant variance of the signal representation and that of the category boundaries, which is assumed to be proportional to $(\log I_N/I_1)^2$, where I_1 and I_N are the intensities of the least and most intense signals, respectively.

According to this theory, for fixed N the total accuracy asymptotes with increasing range because the increase in variance in the category boundaries more than offsets the increase in discriminability in the signal representation due to increased signal separation. Indeed, these postulates account for the bulk of their data, but some notable discrepancies do exist. Without going into them in detail, they seem to result from, first, difficulties in the logarithmic assumption and, second, from what they interpret as 'edge' effects that arise from the end signals. The regularity and, in some experiments, the size of the discrepancies invite alternative models, but so far none has been suggested. We have attempted to construct a pulse model of the type discussed in this chapter, but we are not yet satisfied with it. Our concern has been to find some way to account for the enlarged variance without being forced to assume that it grows directly with range.⁶

The simple reaction-time situation, in which the subject attempts to detect the onset of a signal as rapidly as possible, is a second case when the natural decision rule introduces considerable additional variability. As this topic is important in its own right, we devote the final section to it.

10. SIMPLE REACTION TIMES TO WEAK SIGNALS

It has long been believed that the distribution of reaction times contains information about the decisions being carried out when the subject senses the

⁶ Note added in proof. Our work on the variability of magnitude estimates, reported in Green and Luce (1974), has led to a hypothesis that seems to be consistent with these results. We postulate a band of attention, about 20 dB wide for auditory intensity, which can be located anywhere. Signals falling in the band give rise to samples of IATs that are about an order of magnitude larger than the samples for signals outside the band. Thus, as the range of signals increases, the probability of a small sample and hence increased variability also increases, possibly accounting for the apparent effect of range in degrading absolute identification below that predicted from two-signal data. Of course, detailed work will be needed to verify that this is in fact the explanation. Luce and Green (1974a) also pointed out that such an attention mechanism may underlie the sequential effects observed in both magnitude estimation and absolute identification experiments.

reaction signal, but until comparatively recently little has come of attempts to extract that information. One difficulty is that the temporal reflections of the decision process are almost certainly badly obscured by the ripples resulting from other delays in the overall system unless, of course, the decision process is slow relative to these other delays. Almost paradoxically, reducing the signal intensity serves to amplify the decision process. For a clear and audible signal, the pulse rates are high, decisions rapid, and what we observe-a fairly peaked density whose mean is perhaps 150-200 msec and whose standard deviation is about 10 percent of that—is mostly the result of delays other than those introduced by the decision process. By contrast, for a weak or, because of masking noise, barely audible signal, the pulse rates are low, decisions slow, and what we observe-a broad distribution whose mean is as large as 1000 msec and standard deviation is of the same magnitude-is some mix of the decision latency and other, fixed and variable, residual latencies. Observe that such an increase in the standard deviation is to be expected under the hypothesis that the basic intensity information is encoded in a near-Poisson pulse train since the mean and standard deviation are equal in an exponential distribution.

This general view is probably not very controversial; however, the exact realization of these assumptions differs considerably from one theory to another. All of our own models have explicitly assumed that the decision process is statistically independent of the residual one. In particular, we assume that changes in the signal intensity affect the IATs and so the decision process, but they do not affect the residual process. This is controversial. By independence, the distribution of reaction times is the convolution of the distributions of the decision and residual processes. Other closely related views are the classical one of an irreducible reaction time and Donders' (1868) method of subtraction, which in recent times has been refurbished and exploited effectively by Sternberg (1969). These are weaker models because they assume only additivity, not independence, of the two (or more) stages.

In all such models, no matter what the detailed assumptions are, the effect of the residual latency is simply to obscure the decision process. One way or another, one attempts to evade this noise. A key idea, first pointed out in this context by Christie and Luce (1956) and recently exploited by us, is that the classical transforms—e.g., Laplace and Fourier—convert a convolution into a product. Let capital letters stand for the Fourier transform of the lower case density, i.e.,

$$F(\omega) = \int_{-\infty}^{\infty} e^{-i\omega t} f(t) dt, \quad i = (-1)^{1/2}.$$

$$f(t) = \begin{cases} \int_{0}^{\infty} \ell(t-x) r(x) dx, & t \ge 0, \\ 0, & t < 0, \end{cases}$$
(34)

If

then

$$F(\omega) = L(\omega)R(\omega), \qquad (35)$$

where ℓ is the density of the decision process and r that of the residual one. Given a theory of the decision process, we can derive explicit expressions for ℓ or L which, however, will depend on several parameters that must be estimated from the data. One such parameter is the pulse rate. Two approaches suggest themselves, and there may be others.

Assume for the moment that we have estimates of the parameters. In the one approach we form the histogram that approximates f, smooth it, and then obtain its transform \hat{F} . Substituting the estimated parameters into the decision theory yields an estimate of L, \hat{L} . And so by Equation 35 we estimate R by $\hat{R} = \hat{F}/\hat{L}$, and then by taking the inverse transform, we obtain \hat{r} . In other words, given the data and estimates of the parameters of the theory, we determine what the residual density must be in order for the theory to yield the observed data. Our interest is not in r per se, but rather in whether what we compute is a possible density function. In Green and Luce (1971) we actually carried this out. The theory studied there, which postulated that the occurrence of pulses is treated as evidence that the signal is present, led to a distribution function with two free parameters. They were estimated by a method described below. When we solved for \hat{r} we found a function that had a mean of about 300 msec and that was appreciably negative in the region from about 400 to 500 msec. From data on intense signals we know that this mean is from 100 to 150 msec too large and, of course, the negative region is inconsistent with it being a density function. So we concluded that that theory of the decision process is wrong, and we were led to the IAT theories discussed earlier.

It should be noted that this method of analysis, and any other involving transforms of empirical data, is beset with an inherent difficulty. Considerable effort must be expended to avoid the so-called Gibbs phenomenon—high-frequency oscillations resulting from the discrete jumps that are inherent in histograms—but without losing all resolution. We used a running average on the histogram to suppress some of the discontinuities, but obviously that introduces some temporal smear. Further theoretical work is needed on how best to effect the compromise before this can become a practical technique of analysis.

A second approach is to use very intense signals to estimate \hat{R} (assuming that the decision time is negligible) and then to compare the fit between $\hat{L} = \hat{F}/\hat{R}$ and L. This raises the question, to which we do not know the answer, of how one best evaluates the quality of fit between transformed data and the transform of a theoretical distribution, i.e., between \hat{L} and L. So far as we know, mathematical statisticians have not formulated an answer to this.

Aside from this (apparently) unresolved question of goodness of fit in the transformed domain, two related problems remain. First, we wish to formulate a plausible decision rule for detecting a change in the intensity parameter of a Poisson process and from that to derive the form of the theoretical distribution ℓ (or its transform L). Second, with that in hand, we need to arrive at plausible ways to estimate the parameters of the model, especially the noise and signal Poisson parameters. In practice, we only have incomplete information about ℓ , but just enough to permit crude estimates of the parameters.

As before, we shall assume the simplest decision rule, namely, that an estimate of pulse rate is compared with a criterion, and the subject responds whenever he has evidence that the pulse rate has increased, which suggests that the reaction signal has been presented. Since the subject is under time pressure, we assume that he uses the smallest sample available, namely, one IAT. But unlike the other models we have examined, the number of IATs actually observed before he initiates a response is a random variable, and this fact makes the analysis considerably more difficult. The key result is that (under certain conditions) the tails of the decision distributions (i.e., for $t > \tau'$) are approximately exponential, with time constants that are simple functions of the Poisson parameters of the noise and signal-plus-noise pulse trains.

To fit such a model to data, we must estimate these Poisson parameters. In practice, we have tried only one way and it is not completely satisfactory. Suppose, as seems plausible, that the residual latencies are bounded in the sense that for some $\tau > 0$, r(t) = 0 for $t \ge \tau$. If we substitute this together with the exponential character of the decision process into Equation 34, we see that $f(t) \sim e^{-\mu t} \int_0^{\tau} e^{\mu x} r(x) dx$, $t \ge \tau + \tau'$. So the tails of the observed distributions should exhibit the same exponential character as the decision distribution when the residual times are bounded. Obviously, the same argument is approximately valid if the residual times are not actually bounded, provided that large times are exceedingly rare.

The data in Figure 8 show that the tails of the response-time distributions in a simple reaction-time design (with response terminated signals and exponentially distributed foreperiods) are very nearly exponential. So we may use these data to estimate the Poisson parameters for noise, ν , and for signal plus noise, μ . The ratio of these estimated parameters as a function of signal intensity in dB is shown in Figure 9, and we see that to a good approximation it is a power function of intensity. We used this fact in deriving Equation 26. Luce and Green (1972) provide an argument, based on the assumption of processing on multiple channels, to show that the exponent estimated from Figure 9 is consistent with that estimated from ME data; we do not reproduce that argument here.

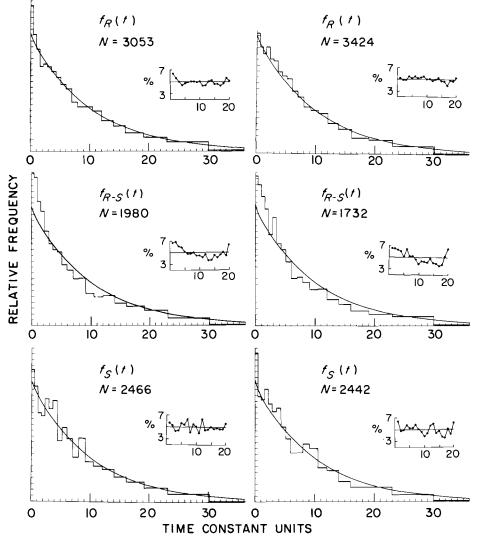


FIGURE 8. Comparison of tails of distributions of time and false alarm to weak signal with bestfitting exponentials (Luce & Green, 1970, Figs. 6, 7).

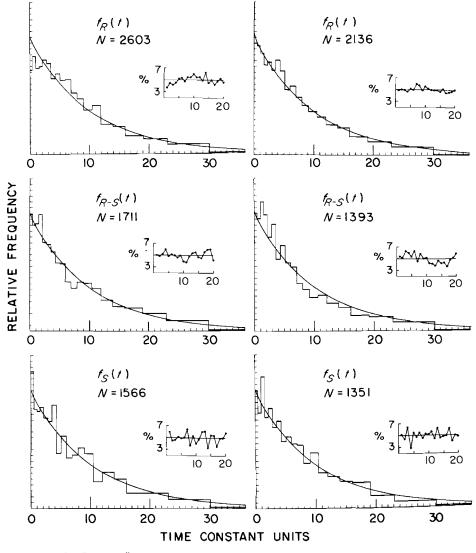
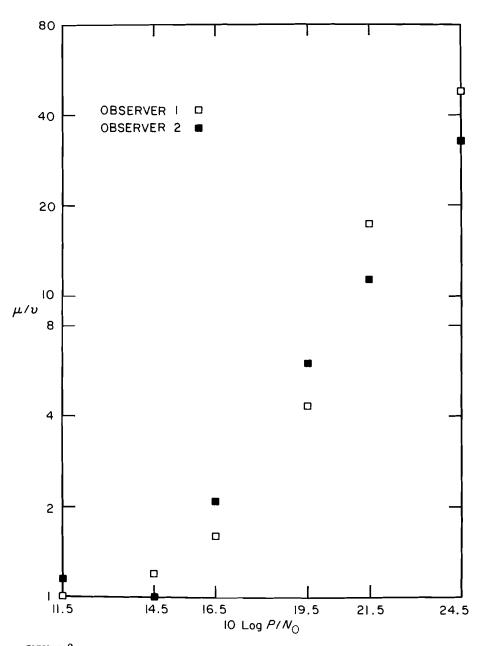


FIGURE 8. (Continued)



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FIGURE 9. Ratio of Poisson parameters, estimated from tails of simple RT distributions, versus signal power to noise density in decibels (Luce & Green, 1970, Fig. 9).

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