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A VARIABLE SENSITIVITY THEORY OF SIGNAL DETECTION¹

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A 2-process model for signal detection is proposed that is applicable to both yes-no and forced-choice experiments. One process describes systematic changes that may occur in the S's sensitivity level to external stimuli; the other process defines a learning mechanism that determines trial-to-trial changes in the S's decision rule as information accrues to him. From the theory one can derive predictions for gross statistics like receiver-operating-characteristic curves and also for detailed sequential effects such as autocorrelation functions defined over stimulus-response runs. Predictions for sequential effects are particularly important in evaluating the theory and provide a valuable insight into the character of the detection process. Application of the theory to various special cases is considered; some predictions are derived and checked against experimental data.

This paper deals with an analysis of some simple detection experiments in terms of a theory that incorporates two separate but interdependent processes: an activation process and a decision process. The *activation process* specifies the relation between external stimulus events and hypothesized sensory states of the subject. The *decision process* specifies the subject's observable response in terms of his sensory state and information acquired during the course of an experiment. Both processes are dynamic. The activation process defines the subject's level of sensitivity to external stimuli, and we postulate that sensitivity may fluctuate (within certain limits) from trial to trial as a function of past events. The decision process is similarly dynamic, for it may change from trial to trial as information accrues to the subject. The processes interact in that the momentary state of one process

operates in a reciprocal fashion to determine the state of the other. As will be indicated later, most theories of signal detection view the subject's sensitivity level as fixed (or at most fluctuating in a strictly random fashion over time) and account for variations in his performance to a fixed intensity signal by postulating changes in the decision rule. In contrast, for the present theory changes in performance to a fixed intensity signal may arise in several ways: manipulating aspects of the experimental situation that affect the subject's sensitivity level but leave the decision process unchanged, manipulating variables that affect the decision process but leave the sensitivity level unchanged, or manipulating parameters that affect changes in both processes.

The theory that we present generates predictions for all aspects of the subject's response protocol (mean response probabilities, associated variances, sequential statistics such as autocorrelation functions on both responses and stimuli, and so forth) and thereby permits a detailed treat-

¹The ideas presented in this paper have been much influenced by discussions with R. A. Kinchla of Ames Research Center. The research was supported by the National Institute of Health under Contract M-5184.

ment of individual trial-by-trial data. Some predictions are parameter free, but by and large the predictions depend on estimates of parameters that describe the stimulus situation and the hypothesized detection process. Some readers may feel that we have been too liberal in postulating parameters; however, for most applications, restrictions are appropriate that markedly reduce the number of parameters that need to be estimated. For example, predictions regarding receiver operating characteristic curves and certain first order sequential phenomena may require that only two parameters be estimated. In contrast, autocorrelation predictions in complex detection experiments may require that as many as six parameters be estimated.

The type of psychophysical study to be considered is a choice experiment for which the experimenter has established, and explained to the subject a one-to-one correspondence between the *response set* (A_1, A_2, \dots, A_r) and the *stimulus presentation set* (S_1, S_2, \dots, S_r). On each trial a stimulus is presented and the subject attempts to identify the stimulus by making the appropriate response. For excellent reviews of research and theory in this area see Green (1960), Licklider (1959), or Swets (1961).

For purposes of this paper we shall consider only experiments for which $r = 2$. That is, on each trial either S_1 or S_2 is presented and the subject is required to make either response A_1 or A_2 . Also, the theoretical development will be restricted to procedures where the experimenter informs the subject at the end of each trial which response was correct. These two restrictions are not fundamental to the theory, but greatly simplify the presentation. Later it will be apparent that the model can

be extended to multistimulus problems and to procedures in which information feedback is manipulated as an experimental variable.

Two types of experimental procedures are to be distinguished in the analysis. We define these in terms of the following examples:

Yes-No procedure. The S_1 is a tone burst in a background of white noise and S_2 is the white noise alone. On a given trial either S_1 or S_2 is presented and the subject answers yes (A_1) or no (A_2) regarding the presence of the signal.

Forced-choice procedure. Two temporal intervals are defined on each trial, exactly one of which contains a signal: i.e., in one interval a tone burst in a background of white noise is presented, while in the other interval only the white noise is presented. On each trial, the subject is required to identify the interval he believes most likely to have contained the signal. Thus, S_i ($i = 1, 2$) denotes a trial on which the signal occurred in Time Interval i and A_j ($j = 1, 2$) denotes the subject's selection of Interval j as the one containing the signal.

In this paper we shall use the identifications given in these examples. That is, for the yes-no procedure S_1 will always denote signal plus noise, whereas S_2 will denote noise alone; for the forced-choice procedure S_1 will denote signal plus noise in the first interval followed by noise alone in the second interval, and S_2 indicates noise alone in the first interval and signal plus noise in the second interval. In addition, the following notation will be used:

$S_{i,n}$ = The presentation of Stimulus S_i on Trial n of the experiment.

$A_{j,n}$ = The occurrence of Response A_j on Trial n of the experiment.

$E_{i,n}$ = The occurrence of an information event at the end of Trial n that indicates that Stimulus S_i was presented.

A theoretical result of particular interest in analyzing detection data deals with the relation of $Pr(A_{1,n}|S_{1,n})$ to $Pr(A_{1,n}|S_{2,n})$. For simplicity we write

$$\begin{aligned} p_{1,n} &= Pr(A_{1,n}|S_{1,n}) \\ p_{2,n} &= Pr(A_{1,n}|S_{2,n}) \end{aligned} \quad [1]$$

and when the appropriate limit exists

$$\lim_{n \rightarrow \infty} p_{i,n} = p_i$$

For the yes-no procedure p_1 is the asymptotic probability of a yes report when the signal is presented (the likelihood of a "hit") and p_2 is the probability of a yes report when noise alone is presented (the likelihood of a "false alarm"). In the literature, plots of the relation of p_2 to p_1 are commonly called ROC curves, which stands for *receiver operating characteristic* curves. It is important to note that we use the term ROC curve in reference to both the yes-no and forced-choice method. When one deals with n interval forced-choice problems, then the ROC curve is a surface in n space and predictable from the theory.

This paper treats the effects of three classes of variables: the physical parameters of the stimulus presentation set; the trial-to-trial schedule for presenting stimuli; and, the class of variables such as monetary payoffs and instructions that are viewed as influencing the motivation and set of the subject. To simplify the discussion, we shall consider only a simple probabilistic scheme for presenting stimuli; namely,

$$Pr(S_{1,n}) = \gamma \quad [2]$$

where γ is constant over trials. More complex stimulus schedules can be analyzed; e.g., the stimulus presentation on Trial n might depend on the response on Trial $n - k$, or on the stimulus on Trial $n - k'$, or both. However, an analysis of this simpler schedule will be sufficient to illustrate the basic concepts and encompasses most of the experimental literature.

Axioms and Rules of Identification

The hypothesized sensory state of the subject that results from the presentation of an external stimulus is specified in terms of two *sensory patterns* s_1 and s_2 and a set S^* of stimulus patterns associated with background stimulation. These stimulus patterns are theoretical constructs to which we will assign certain properties. They are not the receptor neurons of neurophysiology but a schematic representation of the physical stimulus, having certain simple and uniform properties.

On every trial a single pattern is sampled from the background set S^* and simultaneously one of the sensory patterns may or may not be activated. If the s_1 sensory pattern is activated, an A_1 response will occur; if s_2 is activated, an A_2 will occur. If neither sensory pattern is activated, the subject makes the response to which the background pattern is conditioned. Conditioning of elements in S^* may change from trial to trial via a simple learning process.

The likelihood of activating Sensory Pattern s_i given Stimulus Event S_i on Trial n (and thereby insuring a correct response) is denoted as $m_{i,n}$. The parameter $m_{i,n}$ is a measure of the subject's momentary sensitivity level and may fluctuate from trial to trial. However, the momentary sensitivity level is bounded between zero and M_i ,

and the parameter M_i represents the subject's maximum level of sensitivity to a fixed signal. The parameters M_1 and M_2 are to be interpreted as measures of the physical characteristics of S_1 and S_2 and are monotonic with signal strength. Further, we assume that variables such as stimulus presentation schedule, instructions, monetary payoffs, and experimental design have no effect on M_1 and M_2 .

Changes in sensitivity level occur from trial to trial and depend on previous events. Specifically, if the subject tends to do well (i.e., emit correct responses) by ignoring the sensory patterns when they are activated and responding in terms of the background stimuli alone, then he will tend to lower his level of sensitivity. If, however, he tends to do poorly by basing his response solely on the background cues, then he will tend to raise the value of $m_{i,n}$. Roughly speaking, we assume that there is a certain cost associated with maintaining a high level of sensitivity and view the subject as being predisposed to reduce his sensitivity level whenever possible. However, the subject's tendency to lower his sensitivity level is counteracted if the reduction gives rise to a significant decrement in his ability to perform effectively. Thus the activation process can be described as a negative feedback system in which the cost associated with maintaining a high level of sensitivity interacts with the cost associated with a decrement in performance so as to determine a momentary level of sensitivity. The parameters that specify the increments and decrements in sensitivity are μ and δ , and we assume that their values may change if the subject's motivation or set changes. We return to this point later. The concept of a variable level of sensitivity is not new and there is considerable experimental

evidence at both the behavioral and physiological level to support the idea (e.g., Blackwell, 1953; Guilford, 1927; Howarth & Bulmer, 1956; Oldfield, 1955; Verplank, Collier, & Cotton, 1952; Wertheimer, 1953). In addition notions of this sort have played a role in the speculations of Gestalt psychologists (e.g., Kohler, 1947) and more recently, in theoretical developments regarding the interplay between the reticular system and the association cortex (Lindsley, 1958). The important feature of the present theory is the relation postulated between variations in the sensitivity level and past stimulus-response events.

The axioms will be formulated verbally; it is not difficult to state them in a mathematically exact form, but for our purposes this will not be necessary. The axioms fall into three groups: the first group deals with the activation process; the second, with the decision process; and the last group with variations in sensitivity.

Activation axioms. A1. If S_i occurs on Trial n , then Sensory Pattern s_i will be activated with probability $m_{i,n}$.

A2. Exactly one pattern is sampled from set S^* on every trial. Given the set S^* of N patterns, the probability of sampling a particular element is $1/N$, independent of trial number and preceding events.

Response axioms. R1. If Sensory Pattern s_i is activated, then the A_i response will occur. If neither sensory pattern is activated, then the response to which the sampled pattern from S^* is conditioned will occur.

R2. On every trial each pattern in S^* is conditioned to either A_1 or A_2 . If a pattern from S^* is sampled on a trial, it becomes conditioned with probability θ_i to the A_i response if E_i occurs on that trial; if it is already

conditioned to that response, it remains so.

Sensitivity level axioms. L1. The parameter M_i specifies the maximum value of $m_{i,n}$. Further

$$m_{i,n} = w_n M_i$$

L2. The weighting function w_n changes from trial to trial as follows:

$$w_{n+1} = A_n^{(\xi)} [(1 - \delta)w_n] + [1 - A_n^{(\xi)}] [(1 - \mu)w_n + \mu]$$

The function $A_n^{(\xi)}$ denotes the proportion of trials from Trial $n - \xi + 1$ to Trial n on which the information event E_i agreed with the response conditioned to the pattern sampled from S^* .

The distinction between yes-no and forced-choice methods is specified in terms of the parameters M_1 and M_2 . To explicate the distinction between these two experimental procedures we redefine M_1 and M_2 in terms of the more molecular parameters σ and η . Consider a limiting condition in which the subject is performing at his highest level of sensitivity (i.e., $w = 1$). Under these conditions, if a signal is presented in noise we assume that the subject either detects the signal (with probability σ) or is uncertain whether the signal occurred. Similarly, when noise alone is presented we assume that the subject either detects the absence of a signal (with probability η) or is uncertain whether or not the signal occurred. The three events will be denoted as follows: s = detected signal; \bar{s} = detected omission of signal; and u = uncertain. For the yes-no method the occurrence of s is identified with the activation of Sensory Pattern s_1 and therefore a "yes" response; \bar{s} with the activation of s_2 and the occurrence of a "no" response; and the event u with the activation of neither s_1 nor s_2 and

consequently the occurrence of the response conditioned to the element sampled from S^* . Hence for the yes-no procedure

$$\begin{aligned} M_1 &= \sigma \\ M_2 &= \eta \end{aligned} \quad [3]$$

For the forced-choice procedure the analysis is similar. Consider an S_1 trial—signal plus noise in the first interval followed by noise alone in the second interval. One of the following event sequences can occur:

1. Event s occurs in the first interval and is followed by Event \bar{s} in the second interval—with probability $\sigma\eta$
2. s followed by u —with probability $\sigma(1 - \eta)$
3. u followed by \bar{s} —with probability $(1 - \sigma)\eta$
4. u followed by u —with probability $(1 - \sigma)(1 - \eta)$.

Information transmitted by either Outcome 1, 2, or 3 suffices to identify the trial, and therefore the occurrence of any one of these outcomes is associated with the activation of Sensory Pattern s_1 and the occurrence of the A_1 response. If the fourth outcome occurs, we assume that neither sensory pattern is sampled.² Therefore, $M_1 = \sigma\eta + \sigma(1 - \eta) + (1 - \sigma)\eta$ and by a similar argument it can be shown that $M_1 = M_2$. Hence for the forced-

² In formulating a model that also treated choice time and confidence ratings it would be natural to distinguish among Outcomes 1 to 3. However, for an analysis of response selection, such a distinction is not necessary. Also, note that the assignment of probabilities to the four outcomes assumes no time-order effect; i.e., no interaction between events in one temporal interval and the next. For a given experimental situation, the precision of the comparison between the forced-choice and the yes-no method will depend on the accuracy of this assumption.

choice method

$$M_1 = M_2 = \sigma + \eta - \sigma\eta \quad [4]$$

In theory, once σ and η have been estimated, say, for the yes-no method, they can be used to predict in the forced-choice procedure. In this regard note that (for fixed values of σ and η) the parameter $M_1 = M_2$ for the forced-choice method is always greater than or equal to M_1 and M_2 for the yes-no method.

In the present formalization of the theory only Events s and u can occur given signal plus noise and only Events \bar{s} and u , given noise alone. When the model was first developed, we permitted s , \bar{s} , and u to occur (with different probability distributions) given either signal plus noise or noise alone. However, in the analysis of several sets of data (Atkinson & Carterette, in preparation³; Carterette & Wyman, 1962; Kinchla, 1962) estimates of the probability of Event s given signal plus noise and the probability of \bar{s} given signal plus noise were consistently equal to zero. Hence for the present discussion we have chosen to let $Pr(s|\text{noise alone}) = Pr(\bar{s}|\text{signal plus noise}) = 0$ and thereby simplify the presentation. It also is interesting that in the analysis of the above data the estimate of η was very close to zero. In fact, by setting $\eta = 0$ the correspondence between theoretical and observed values was not much different than when a separate estimate of the parameter was made. However, even for small values of η the \bar{s} event plays an important role in accounting for second choice data in multiinterval forced-choice experiments and for this reason the sim-

³ Atkinson, R. C., & Carterette, E. C. Signal detection as a function of the stimulus presentation schedule: A comparison of forced-choice and yes-no procedures (in preparation).

plifying assumption of $\eta = 0$ was not made.

Asymptotic Response Probabilities and ROC Curves

If we let ψ_n denote the proportion of elements in S^* conditioned to an A_1 response at the start of Trial n , then (by Axioms A2 and R2) we may write the following difference equation:

$$\psi_{n+1} = \psi_n \left[1 - (1 - \gamma) \frac{\theta_2}{N} - \gamma \frac{\theta_1}{N} \right] + \gamma \frac{\theta_1}{N}$$

This recursion can be solved by standard methods (see Atkinson & Estes, 1963) to yield the explicit formula

$$\psi_n = \psi - [\psi - \psi_1] \times \left[1 - \frac{1}{N} \{ \theta_2(1 - \gamma) + \theta_1\gamma \} \right]^{n-1}$$

where

$$\psi = \frac{\gamma}{\gamma + (1 - \gamma)\beta} \quad [5]$$

and the *response bias* parameter $\beta = \frac{\theta_2}{\theta_1}$.

The quantity ψ denotes the $\lim_{n \rightarrow \infty} \psi_n$ and is the asymptotic probability of an A_1 response given that an element from S^* determines the subject's response. For most analyses we shall be concerned with response protocols that may be viewed as asymptotic data. Hence, in general, theoretical results are presented only for the case in which n is large.

Using techniques similar to those employed in Equation 5 and applying Axiom T2 yields an expression for $\lim_{n \rightarrow \infty} w_n = w$; namely,

$$w = \frac{1 - A}{1 - A + A\alpha} \quad [6]$$

where the *activation parameter* $\alpha = \frac{\delta}{\mu}$

and $A = \gamma\psi + (1 - \gamma)(1 - \psi)$. In the statement of Axiom T2 we assume that the amount w_n increases or decreases on a trial depends on $A_n^{(\xi)}$; the value of this function being the proportion of times on the last ξ trials on which the subject would have been correct by ignoring the sensory pattern and responding solely in terms of the background cue. It is interesting that the asymptotic expression for w_n in Equation 6 is not a function of ξ ; i.e., independent of the number of trials the subject scans over, the value of w depends only on α , β , and γ . To be more exact, at asymptote the random variable associated with the weighting function has an expectation of w independent of ξ ; however, the variance of the distribution does depend on ξ , being maximum when $\xi = 1$ and approaching zero as ξ becomes large. Analyses of data reported by Carterette and Wyman (1963), and Atkinson and Carterette³ yielded estimates of ξ that were quite large. In view of these empirical results and for reasons of mathematical simplicity we will, in general, assume that $\xi \rightarrow \infty$. Later the effect of ξ on sequential predictions will be discussed but, otherwise, the mathematical results presented in this paper will be for the case where the scan range is large.

Employing our previous results, and using Axioms A1 and A2 we obtain:

$$p_1 = m_1 + (1 - m_1)\psi \quad [7a]$$

$$p_2 = (1 - m_2)\psi \quad [7b]$$

where $m_i = \lim_{n \rightarrow \infty} m_{i,n}$, and

$$m_i = wM_i \quad [8]$$

An inspection of Equations 7 and 8 indicates that p_1 and p_2 are functions of M_1 , M_2 , α , β , and γ . Of course γ is

specified by the experimenter and therefore, to fit any ROC curve, four parameters need to be estimated. However, for most applications restrictions are appropriate that reduce this number. For example, in a forced-choice experiment the symmetry between S_1 and S_2 stimuli is such to require that $\theta_1 = \theta_2$ (unless the subject has a bias extraneous to the experiment that favors one response over the other) and hence $\beta = 1$. Further, by an earlier argument (see Equation 4) we require that $M_1 = M_2$. Therefore, in a forced-choice procedure the ROC curve depends only on M and α .

ROC curves. We now examine two methods for experimentally generating ROC curves. One procedure is to vary the schedule for presenting S_1 and S_2 ; for purposes of the present paper this involves varying γ from session to session while holding all other factors constant (Tanner, Swets, & Green, 1956). Another method for generating ROC curves is to manipulate instructional variables and/or payoffs from one experimental session to another while using the same stimuli and holding γ fixed (Swets, Tanner, & Birdsall, 1955). The predictions for each of these cases will be examined separately.

Consider first the case in which γ is permitted to vary while all other factors remain unchanged. Under these conditions it is assumed that the instructions and payoffs specify fixed values of the response bias parameter β and the activation parameter α . Also M_1 and M_2 are not affected by the value of γ for, in theory, they depend only on the physical characteristics of the stimulus presentation set. Therefore, for a given experimental situation M_1 , M_2 , α , and β are fixed, and variations in p_1 and p_2 induced by manipulating the schedule for pre-

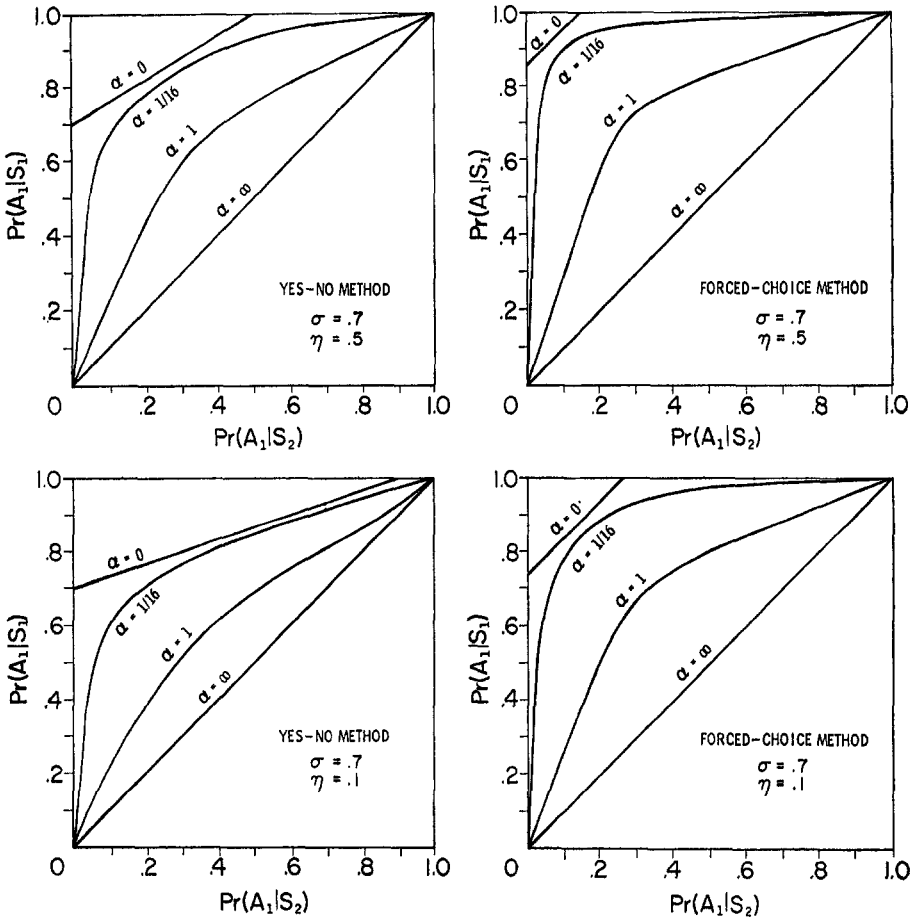


FIG. 1. ROC curves generated by manipulating the presentation schedule of stimulus events.

senting S_1 and S_2 must be accounted for strictly by variations in γ .

If we hold M_1 , M_2 , α , and β constant and vary γ between 0 and 1 (the permissible range), then the ROC curve defined by Equation 7 is in general, a monotone increasing function that originates at point (0, 0) and terminates at point (1, 1). However, it is necessary to be more precise and distinguish three cases:

1. If $\delta = 0$ and $\mu > 0$, then asymptotically the subject performs at his maximum level of sensitivity independent of other factors, and the ROC

curve is given by the linear function

$$p_1 = \frac{1 - M_1}{1 - M_2} p_2 + M_1 \quad [9]$$

2. If $\delta > 0$ and $\mu = 0$, then asymptotically the subject performs at his minimum level of sensitivity, and the ROC curve is simply

$$p_1 = p_2 \quad [10]$$

3. For the general case where μ and δ are both greater than zero, the ROC curve is a nonlinear monotone increasing function bounded between

Equation 9 and Equation 10 that originates at (0, 0) and terminates at (1, 1).

Figure 1 gives several ROC curves for both yes-no and forced-choice procedures when $\beta = 1$, $\sigma = .7$, and $\eta = .5$ or $.1$. The parameter on each set of curves is the value of α . Successive points on an individual curve were swept out by letting γ vary from 0 to 1. For the general case, α is a ratio of two nonzero probabilities and hence takes any value greater than zero. For α close to zero (low sensitivity level) the ROC curve tends toward the line $p_1 = p_2$; as α becomes large the curve approaches the line given by Equation 9. Further, as indicated in Figure 1, when α and β are the same in both the yes-no and the forced-choice procedure, then (by the conditions of Equations 3 and 4) the theory predicts that the ROC curve generated by the forced-choice group will be above the ROC curve for the yes-no group.

It also can be shown that the ROC curve defined by varying γ is either symmetric about the main diagonal from point (0, 1) to (1, 0), skewed right, or skewed left. For symmetry we require $M_1 = M_2$ and $\beta = 1$; otherwise the curve may be skewed right or left. Note that the conditions that specify a symmetric ROC curve hold in the forced-choice experiment; they may or may not hold for different yes-no experiments.

Another method for generating ROC curves is to fix both γ and the signal intensity, and manipulate instructions and/or payoffs from one experimental session to another. Under these conditions M_1 and M_2 would be constant over sessions but we might assume that the response parameter and the activation parameter vary. Thus the ROC curve produced by changing instructions or

payoffs would theoretically be explained by variations in α and/or β given fixed values of M_1 , M_2 , and γ . In the discussion of this method we let $\gamma = 1/2$; this condition simplifies the mathematics and includes most of the experimental work. We examine first the cases in which only α or β is permitted to vary and then the case in which they vary concomitantly.

If we hold the bias parameter β constant and let α vary from 0 to ∞ then the ROC curve is a straight line segment between the point

$$p_1 = M_1 + \frac{1 - M_1}{1 + \beta} \quad , \quad p_2 = \frac{1 - M_2}{1 + \beta}$$

and the point

$$p_1 = \frac{1}{1 + \beta} \quad , \quad p_2 = \frac{1}{1 + \beta}$$

That is, as the activation parameter varies (and all other parameters are fixed) we move along the function

$$p_1 = -\frac{M_1}{M_2} \beta p_2 + \frac{1}{1 + \beta} \times \left[1 + \frac{M_1}{M_2} \beta \right] \quad [11]$$

Such a prediction readily can be realized experimentally. For the forced-choice method β is fixed and we could manipulate α by varying the amount of payoff for a correct response from one experimental session to another. Then, the ROC curve generated over experimental sessions would be specified by Equation 11. Such an experiment has been conducted by Blackwell (1953) and this is precisely the type of effect observed.

To be sure, the ROC function given by Equation 11 is rather different from the typical curve that one thinks of with regard to signal detection. However, there is no doubt that such functions can be generated experi-

mentally by symmetrically manipulating motivation variables in the forced-choice problem. In this regard, it should be noted that the ROC curve has been referred to in the literature as an equisensitivity curve (Luce, 1961). For theories of signal detection that have static concepts of the activation process, such a term is appropriate because all points on the function represent equally sensitive activation levels. However, from our viewpoint the term equisensitive does not connote the correct meaning, for we admit the possibility of generating an ROC curve via variations in sensitivity. Specifically, in terms of the present theory, ROC curves may arise in the following ways: experimentally manipulating parameters that affect the activation process but leave the decision process unchanged (e.g., Equation 11); manipulating parameters that affect the decision process but leave the activation process unchanged (e.g., Equation 12); or manipulating parameters that affect changes in both the activation and decision processes (e.g., the case in which γ varies while all other parameters are fixed).

If we hold α fixed and let β vary (for M_i fixed and $\gamma = 1/2$), then the ROC curve is given by the function

$$p_1 = \frac{1 + \alpha - M_1}{1 + \alpha - M_2} p_2 + \frac{M_1}{1 + \alpha} \quad [12]$$

We know of no experimental results that relate to this prediction.

Finally, in a yes-no experiment it seems reasonable to assume that both α and β may vary simultaneously as instructions and/or payoff change. To illustrate the type of effect that can be obtained consider the case in which $\alpha = f(\beta)$ such that the function f is strictly monotone increasing and $f(0) = 0$. Under these conditions if β

varies between 0 and ∞ , then a convex ROC curve is traced out from point ($p_1 = 1, p_2 = 1 - M_2$) to point ($p_1 = 0, p_2 = 0$) that is bounded between Equations 9 and 10. The degree of convexity and the symmetry of the ROC curve will depend on the function f . In this regard, it is interesting to view the estimate of f for a given set of data as a device for scaling the effects of instructions and payoffs.

In terms of the above discussion, it should be obvious that virtually any ROC curve can be fitted by selecting appropriate parameter values. Thus, within the framework of the present theory, the ability of the model to fit ROC data is a rather trivial test. It is for this reason that we now turn to more detailed predictions regarding the fine structure of signal detection data.

Sequential Predictions

It has long been recognized that rather complex trial-to-trial dependencies are involved in most psychophysical data. Some particularly striking effects have been reported by Carterette and Wyman (1963), Howarth and Bulmer (1956), and Verplank, Collier, and Cotton (1952); these experimenters have demonstrated that detection rates (even for sophisticated subjects) may increase or decrease depending on the immediately prior sequence of stimulus-response events. In this section we present some sequential predictions for signal detection studies, having selected those quantities that are particularly useful in making estimates of parameters. The reader is referred to Suppes and Atkinson (1960, Ch. 2) for a discussion of appropriate estimation procedures.

We shall examine predictions regarding the influence of stimulus and

response events on Trial n as They affect the response on Trial $n + 1$. Specifically

$$Pr(A_{1,n+1} | S_{i,n+1} A_{j,n} S_{k,n})$$

where $i, j, k = 1, 2$. Explicit expressions for these quantities can be derived from the axioms. The actual derivations are quite lengthy and will not be presented here; the reader interested in the mathematical tech-

niques involved should consult Atkinson and Estes (1963). Also, for purposes of this paper, the analysis of sequential effects will be confined to asymptotic statistics. To simplify notation the quantity

$$\lim_{n \rightarrow \infty} Pr(A_{1,n+1} | S_{i,n+1} A_{j,n} S_{k,n})$$

will be written as $Pr(A_1 | S_i A_j S_k)$. The expressions for these probabilities are as follows:

$$Pr(A_1 | S_1 A_1 S_1) = \frac{(N - 1)p_1}{N} + \frac{\psi + (1 - \psi)m_1[\theta_1 + (1 - \theta_1)m_1]}{Np_1} \quad [13a]$$

$$Pr(A_1 | S_1 A_2 S_1) = \frac{(N - 1)p_1}{N} + \frac{(1 - \psi)(1 - m_1)[\theta_1 + (1 - \theta_1)m_1]}{N(1 - p_1)} \quad [13b]$$

$$Pr(A_1 | S_1 A_1 S_2) = \frac{(N - 1)p_1}{N} + \frac{\psi(1 - m_2)[\theta_2 m_1 + 1 - \theta_2]}{Np_2} \quad [13c]$$

$$Pr(A_1 | S_1 A_2 S_2) = \frac{(N - 1)p_1}{N} + \frac{\psi m_2[\theta_2 m_1 + 1 - \theta_2] + (1 - \psi)m_1}{N(1 - p_2)} \quad [13d]$$

$$Pr(A_1 | S_2 A_1 S_1) = \frac{(N - 1)p_2}{N} + \frac{\psi(1 - m_2) + (1 - \psi)m_1 \theta_1 (1 - m_2)}{Np_1} \quad [13e]$$

$$Pr(A_1 | S_2 A_2 S_1) = \frac{(N - 1)p_2}{N} + \frac{(1 - \psi)(1 - m_1)\theta_1(1 - m_2)}{N(1 - p_1)} \quad [13f]$$

$$Pr(A_1 | S_2 A_1 S_2) = \frac{(N - 1)p_2}{N} + \frac{\psi(1 - m_2)(1 - \theta_2)(1 - m_2)}{Np_2} \quad [13g]$$

$$Pr(A_1 | S_2 A_2 S_2) = \frac{(N - 1)p_2}{N} + \frac{\psi m_2(1 - \theta_2)(1 - m_2)}{N(1 - p_2)} \quad [13h]$$

To obtain $Pr(A_2 | S_i A_j S_k)$ one need only note that

$$Pr(A_1 | S_i A_j S_k) + Pr(A_2 | S_i A_j S_k) = 1.$$

The expressions in Equation 13 are rather formidable looking, but numerical predictions can be easily calculated once values for the parameters have been obtained. Furthermore, independently of the parameter values, certain relations among the sequential probabilities can

be specified. For example, it can be easily shown that

$$Pr(A_1 | S_i A_1 S_i) \geq Pr(A_1 | S_i A_2 S_i)$$

or that

$$Pr(A_1 | S_i A_i S_1) \geq Pr(A_1 | S_i A_i S_2)$$

for $i = 1, 2$ and for any values of γ, M_1 , and M_2 .

To indicate the nature of these predictions we shall examine some data from two subjects run in a

forced-choice auditory experiment. Two temporal intervals were defined on each trial by the onset and offset of two lights. A band-limited Gaussian noise (the masking stimulus) was present continuously throughout the experimental session and on every trial one of the two temporal intervals contained a fixed intensity, 1,000 cps tone. The subject pressed one button if he believed the signal was in the first interval or pressed a second button if he believed the signal was in the second interval. The experimental procedure is described in detail in Atkinson and Carterette³; that paper deals with an analysis of forced-choice and yes-no data from 12 subjects, each run for 350 trials per day for 30 days.

The data we present here is not to be regarded as a test of the theory, but only to illustrate some of the predictions. Table 1 presents the observed values for p_1 , p_2 , and $Pr(A_1|S_iA_jS_k)$. The value of γ was set at 1/2 in the experiment and, since a forced-choice method was used, we assume that $\beta = 1$ (i.e., $\theta_1 = \theta_2 = \theta$). Given that $\beta = 1$ and $\gamma = 1/2$ we have, via Equation 5, that $\psi = 1/2$. Knowing ψ and the observed value of p_1 , Equation 7a may be used to obtain an estimate of m_1 ; namely, $m_1 + (1 - m_1)/2 = .73$ or $m_1 = .46$. Further, for the forced-choice procedure $M_1 = M_2$ and therefore, by Equation 8, it follows that $m_1 = m_2 = m$. Using the above estimate of m we predict by Equation 7b that $p_2 = .27$ which is quite close to the observed value of .28.

In order to compute predictions for the sequential statistics in Table 1 values for θ and N are required in addition to the estimate of m . Several methods may be used to estimate θ and N but, for simplicity, we apply a least squares technique. Specifically,

TABLE 1
PREDICTED AND OBSERVED RESPONSE
PROBABILITIES AT ASYMPTOTE

	Observed	Predicted
$Pr(A_1 S_1)$.73	.73
$Pr(A_1 S_2)$.28	.27
$Pr(A_1 S_1A_1S_1)$.80	.78
$Pr(A_1 S_1A_2S_1)$.76	.75
$Pr(A_1 S_1A_1S_2)$.73	.71
$Pr(A_1 S_1A_2S_2)$.67	.68
$Pr(A_1 S_2A_1S_1)$.30	.32
$Pr(A_1 S_2A_2S_1)$.32	.29
$Pr(A_1 S_2A_1S_2)$.26	.25
$Pr(A_1 S_2A_2S_2)$.22	.22

for $m = .46$, the following function is defined:

$$S(\theta, N) = \sum_{i,j,k} \{Pr(A_1|S_iA_jS_k) - \hat{Pr}(A_1|S_iA_jS_k)\}^2$$

where $\hat{Pr}(\cdot)$ denotes the observed values given in Table 1. Applying the method of least squares, estimates of θ and N are obtained by selecting values for these parameters that minimize the function $S(\theta, N)$.

Using appropriate numerical techniques, the following estimates were obtained: $\theta = .62$, $N = 3.83$. The predictions corresponding to these parameter values are presented in Table 1. When one considers that only three of the possible eight degrees of freedom represented in the table have been utilized in estimating parameters, the correspondence between theoretical and observed quantities is quite good. The fact that our estimation procedure yields a non-integral value of N may suggest that N varies somewhat from time to time, or it may reflect some contamination of the data by sources of experimental error not represented in the model. The reader interested in other applica-

tions of this model to sequential data should see Atkinson (1963).

DISCUSSION

In some respects the theory proposed in this paper is similar to various applications of statistical decision theory to psychophysical phenomena (Swets, Tanner, & Birdsall, 1961; Tanner & Swets, 1954). The decision theory approach rejects the conventional notion of a threshold and argues for the concept of a criterion range of acceptance. They assume that on each trial the reaction of the sensory system to an external stimulus can be characterized by a number (a likelihood ratio) and the subject's response depends on whether or not the number falls in the criterion range. The process is not deterministic, for repeated presentations of a stimulus do not generate the same number but rather a distribution of numbers (i.e., to a single presentation of the stimulus a number is *randomly* drawn from the distribution). The position of the criterion (the operating level) is assumed to be under the control of the observer and to vary as a function of psychological variables that influence motivation and set. Specifically, the subject fixes the operating level in terms of a priori probabilities of stimuli and the costs associated with the various choices in such a way as to maximize his expected utility. Translated into the language used in this paper, the activation process is represented by the random sampling of a number from a distribution associated with the stimulus; and the decision process refers to the selection, by the subject, of an operating level or criterion.

A principal distinction between our approach and signal detection theory is with regard to the activation

process. In our theory the sensitivity level of the activation process may vary (within a given range) from trial to trial as a function of the preceding events. In contrast, signal detection theory conceptualizes the activation process as static, for the parameters that describe the response of the sensory system to an external stimulus are constant and do not depend on instructions, stimulus schedules, payoffs, or other variables that might influence set or motivation.

Another distinction between our approach and signal detectability theory is with regard to the decision process. Both theories permit variations in the decision rule as a function of various independent variables but in quite different ways. For signal detection theory the subject selects a criterion in terms of certain game-theoretic considerations that take into account a priori probabilities of stimuli and the costs associated with the various choices. Once the criterion has been selected for a given experimental condition it is assumed to be relatively fixed, and consequently there is no possibility for predicting trial-by-trial sequential effects. In contrast, for the present theory, the decision process changes from trial to trial as a function of the type of information that accrues to the subject.

In discussing the decision rule it is important to realize that we have placed a heavy emphasis on a learning process associated with stimuli extraneous to the signal source (i.e., background cues). This learning process plays a central role in determining the values of p_1 and p_2 as a function of various independent variables and provides one means of accounting for sequential effects in psychophysical data. It should be emphasized that the sequential results predicted by Equation 13 are due

entirely to trial-to-trial changes in the conditioning of stimuli in the background set S^* . Another source of sequential variability can arise from trial-to-trial fluctuations in $m_{i,n}$. When the scan range, ξ , is large these effects are negligible at asymptote; however, for small values of ξ they can be quite important. As indicated earlier, we have obtained good accounts of sequential effects for several sets of data by assuming that the scan range is large. Further, when $\xi \rightarrow \infty$ the mathematical analysis is simplified. It is for these reasons that we have been willing to begin by making this assumption.

Without actually estimating the value of ξ one can obtain various crude, but easily calculated, measures of trial-to-trial fluctuations in sensitivity (as opposed to the long term changes in sensitivity level described by Equation 6). As an example, let C_n and \bar{C}_n denote correct ($S_1 - A_1$ or $S_2 - A_2$) and incorrect ($S_1 - A_2$ or $S_2 - A_1$) responses on Trial n , respectively. Then in a forced-choice experiment in which $\gamma = 1/2$, the theory in general predicts that

$$Pr(C_{n+1}|C_n) \neq Pr(C_{n+1}|\bar{C}_n) \quad [14]$$

except when $\xi \rightarrow \infty$.⁴ If over an extended series of trials estimates of these two probabilities are significantly different, then it will be necessary to take into account not only long-term changes in sensitivity level but also the more local effects. In this regard, it should be pointed out that any theory of signal detection that postulates a static activation process has as a consequence the prediction that

$$Pr(C_{n+1}|C_n) = Pr(C_{n+1}|\bar{C}_n)$$

⁴ It should be emphasized that the prediction in Equation 14 does not depend on the value of β but only on the fact that $M_1 = M_2$ and $\gamma = 1/2$.

in a forced-choice experiment with $\gamma = 1/2$; this result holds for both a correct information procedure and a no information procedure.

Our presentation of the theory has dealt with experimental situations in which the subject is given correct information on each trial regarding the appropriate response; i.e.,

$$Pr(E_{1,n}|S_{1,n}) = Pr(E_{2,n}|S_{2,n}) = 1$$

It is obvious that the axioms, as stated, are directly applicable to problems in which the experimenter may give false information on some trials. We shall not go into the predictions for this type of experiment except to say that the theory gives a good account, at least at the qualitative level, of the findings reported by Carterette and Wyman (1963) and Suppes and Krasne (1961) on detection problems in which incorrect information was manipulated as an experimental variable.

Throughout this paper, we have considered psychophysical methods in which the subject is given information on each trial and have not dealt with the no information case. Under conditions of no information certain changes need to be made in Axioms A3 and L2. A discussion of this version of the theory is given in Atkinson (1963) and Atkinson and Estes (1963) and applied to some forced-choice visual detection data involving no information feedback; the detailed predictions for both asymptotic response proportions and first-order sequential statistics are excellent. However, the major difficulty with the no information condition is that it makes the mathematical predictions less manageable and increases the sampling error associated with parameter estimates. Thus, within the present theoretical framework, the study of the no information case

warrants only limited investigation until the less complicated cases have been adequately explored.

There are a number of special topics that have not been discussed. The following are of particular interest: the effect of blank trials in a forced-choice procedure; extension of the model to account for choice-time measures; and extension of the model to multiinterval forced-choice experiments where second choices are permitted. These problems can be formulated in a natural way within the framework of the theory and will be treated in later papers.

SUMMARY

In this paper we present an analysis of both yes-no and forced-choice experiments in terms of a two-process model. One process describes systematic changes that may occur over time in the subject's sensitivity level to external stimuli; the other process specifies changes in the subject's decision rule as information accrues to him. From the theory one can derive predictions regarding both gross statistics like receiver-operating-characteristic curves and detailed sequential statistics like autocorrelations based on previous stimulus-response events.

Most theories of signal detection assume that the subject's decision rule changes as a function of instructions, payoffs, stimulus presentation schedules, and other experimental variables, but to our knowledge the present paper is the first to examine the implications of postulating systematic nonrandom changes in sensitivity. Undoubtedly the detailed features of the axioms describing changes in sensitivity are going to need much revision to provide a broad

base for interpreting psychophysical phenomena. Nevertheless, it seems clear that by assuming a variable sensitivity level one can provide a highly parsimonious account of a wide array of phenomena. No suggestions have been offered regarding the mechanism that might account for changes in sensitivity (e.g., orienting responses, peripheral changes within the sensory system, or events presumed to occur at higher centers) and future exploration of the concept may require such specificity.

Another unique aspect of the present development is its emphasis on sequential phenomena. These effects can be easily estimated in most experiments and represent a source of information about detection behavior that cannot be duplicated by an analysis of gross statistics like the proportion of hits or false alarms. Within the present theory, sequential effects are accounted for in terms of trial-by-trial fluctuations in both the decision rule and the sensitivity level. Predictions regarding sequential phenomena play a crucial role in evaluating the theory. In the past, most investigators either have ignored these sequential effects or treated them as experimental artifacts to be minimized by counterbalancing, trial spacing, or by the use of trained subjects.

Much research is needed to test the general class of models suggested by the theory. However, in our opinion, there is enough evidence already available to suggest that the concept of a variable sensitivity level will be a necessary ingredient of a comprehensive theory of detection behavior. Also, it is hoped that the present paper has emphasized the importance of examining trial-by-trial sequential phenomena as a source of information about the perceptual process.

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