7 A LEARNING MODEL FOR FORCED-CHOICE DETECTION EXPERIMENTS[1]

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Several signal detection experiments employing a forced-choice procedure are analysed in terms of a model that incorporates two distinct processes: a sensory process and a decision process. The sensory process specifies the relation between external signal events and hypothesized sensory states of the subject. The decision process specifies the relation between the sensory states and the observable responses of the subject. The sensory process is assumed to be fixed throughout an experiment, whereas the decision process is viewed as varying from trial to trial as a function of the particular sequence of preceding events. The changes in the decision process are assumed to be governed by a simple stochastic learning model. There are several ways of formulating the learning model and the experiments reported here were designed to select among these alternative approaches. The empirical results favour a linear-operator process with trial-to-trial changes in response probabilities that are a function not only of the signal and information events, but also of the particular sequence of sensory states activated.

1. Introduction

This paper examines a model for choice behaviour in a two-alternative forced-choice detection task. The model is restricted to experimental situations where the subject is given feedback on every trial regarding the correctness of his response, and to situations with a simple outcome structure. Thus the model has a limited range of applicability, but for appropriately contrived experiments it appears to provide an accurate account of the gross aspects of the data and certain sequential effects. The model represents a special case of a more general theory proposed by Luce (1963); it is also very similar in most details to a model of forced-choice behaviour proposed by Atkinson (1963). The relations of the model developed in this paper to these other theories of detection behaviour are examined in some detail by Atkinson, Bower and Crothers (1965, Chapter 5); they also discuss the relation of the model to various theories that have been proposed for probability learning experiments.

The model postulates that the observable relations between stimulus events and responses are a product of two processes: a sensory process and a decision process. The sensory process specifies the relation between the external stimulus event and hypothetical sensory states of the subject. The decision process

[1]Support for this research was provided by the National Aeronautics and Space Administration, no. NGR-05-020-036.

specifies the subject's response in terms of his current sensory state and information that he has acquired during the course of a given experiment. The two processes interact as follows: the stimulus is fed into the sensory process which converts the pattern of external energy changes into sensory information (sensory events); the decision process then operates on the sensory information to determine a response. Some theories of detection have assumed a continuum of sensory states (Green, 1960; Swets, 1961; Tanner and Swets, 1954), whereas others have argued for a finite representation (Atkinson, Carterette and Kinchla, 1962; Fechner, 1860; Luce, 1963; Norman, 1964). Further, some have proposed that the sensory process is static over trials, whereas others have assumed that it varies within certain fixed limits from trial to trial as a function of preceding events (Atkinson, 1963). One point of agreement among all theories is that the decision process is dynamic, and undergoes change when the experimenter manipulates the presentation schedule or outcome structure. However, for a given experimental schedule some theories treat the decision process as fixed (independent over trials), whereas others represent it as changing from trial to trial as a function of the particular sequence of preceding events. This latter way of representing the decision process is an important feature of the model considered in this paper. The subject is viewed as adopting a pattern of decision making in each experimental situation by means of a simple stochastic learning mechanism. The learning mechanism that will be examined is similar to those proposed by Bush and Mosteller (1955).

As noted above, the type of psychophysical situation that we shall consider is a two-alternative forced-choice detection experiment. On each trial two temporal intervals are defined and the subject is instructed to report which interval contains a signal. It is a forced-choice task in that on each trial the subject must select one of the two intervals as containing a signal even if he is uncertain as to what occurred. The presentation of a signal plus noise in the first interval and noise alone in the second interval on trial n will be denoted as $S_{1,n}$ and the presentation of noise in the first observation interval-followed by signal plus noise in the second observation interval as $S_{2,n}$. Further, the subject's responses will be denoted $A_{1,n}$ and $A_{2,n}$ to indicate which interval he reported contained the signal on trial n. Finally, $E_{1,n}$ and $E_{2,n}$ will denote the occurrence of an event at the end of trial n informing the subject that stimulus S_1 or S_2, respectively, was presented. Thus

$S_{i,n}$ = the presentation of stimulus S_i on trial n,

$A_{j,n}$ = the occurrence of response A_j on trial n,

$E_{k,n}$ = information event at the end of trial n indicating that stimulus S_k was presented.

Using this notation each trial can be described by the ordered triple $\langle S_i, A_j, E_k \rangle$.

In experiments of the type described above the following variables can be manipulated: (*a*) physical parameters of the signal and noise; (*b*) presentation schedule of signal events; (*c*) information feedback; and (*d*) the outcome structure

which specifies the payoffs associated with correct and incorrect responses. In this paper we shall examine how these variables influence detection behaviour, but the experiments reported here deal only with manipulations involving presentation schedules and information feedback. The presentation of signal events will be specified by a probabilistic schedule; namely, events S_1 and S_2 will form a binomial sequence with parameter γ. Further, the experiments employ a simple outcome structure. The subject is instructed to make a correct response as often as possible, and each trial terminates with an information event which tells him whether he was correct or not. There are no monetary payoffs or penalties for correct and incorrect responses as is frequently the case in detection experiments.

The major dependent variable is the probability of an A_j response on trial n, given that stimulus S_i occurred. The four outcomes can be represented by the matrix

$$\mathbf{P}_n = \begin{array}{c} \\ S_{1,n} \\ S_{2,n} \end{array} \begin{array}{c} \begin{array}{cc} A_{1,n} & A_{2,n} \end{array} \\ \begin{bmatrix} Pr(A_{1,n} \mid S_{1,n}) & Pr(A_{2,n} \mid S_{1,n}) \\ Pr(A_{1,n} \mid S_{2,n}) & Pr(A_{2,n} \mid S_{2,n}) \end{bmatrix} \end{array}. \tag{1}$$

This matrix will be called the *performance matrix*. In the literature the occurrence of an A_1 response to an S_1 stimulus is called a *hit*, and the occurrence of A_1 response to an S_2 stimulus is called a *false alarm*. We shall use this terminology, denoting them as H_n and F_n, i.e.,

$$Pr(H_n) = Pr(A_{1,n} \mid S_{1,n})$$
$$Pr(F_n) = Pr(A_{1,n} \mid S_{2,n}).$$

Fixing $Pr(H_n)$ and $Pr(F_n)$, then, completely specifies the performance matrix.

Other quantities of interest can be defined in terms of the hits and false alarms. Frequently we want to know the probability of an A_1 response on trial n independent of the stimulus event; namely,

$$Pr(A_{1,n}) = Pr(H_n)Pr(S_{1,n}) + Pr(F_n)Pr(S_{2,n}). \tag{2}$$

Also of interest is the probability of a correct response on trial n (which is denoted C_n):

$$Pr(C_n) = Pr(H_n)Pr(S_{1,n}) + [1 - Pr(F_n)]Pr(S_{2,n}). \tag{3}$$

2. Assumptions and Rules of Identification

Sensory and Decision Processes

The model assumes that one and only one sensory state can occur on each trial of the experiment. The sensory states will be denoted as $s_0, s_1, s_2, s_3, \ldots$. We do not suppose that the same sensory state necessarily results whenever a particular stimulus is presented, but rather that the state is determined by a random process. The sensory process on trial n of an experiment can be represented by the *sensory matrix*

$$\mathbf{S}_n = \begin{array}{c} \\ S_1 \\ S_2 \end{array} \begin{array}{c} \begin{array}{cccc} s_0 & s_1 & s_2 & \cdots & s_x \end{array} \\ \begin{bmatrix} a_{10}^{(n)} & a_{11}^{(n)} & a_{12}^{(n)} & \cdots & a_{1x}^{(n)} \\ a_{20}^{(n)} & a_{21}^{(n)} & a_{22}^{(n)} & \cdots & a_{2x}^{(n)} \end{bmatrix} \end{array},$$

where $a_{ij}^{(n)}$ denotes the probability of eliciting sensory state s_j on trial n given stimulus S_i on that trial. Similarly, the decision process can be represented by the matrix

$$\mathbf{D}_n = \begin{array}{c} \\ s_0 \\ s_1 \\ s_2 \\ \cdot \\ \cdot \\ \cdot \\ s_x \end{array} \begin{array}{c} \begin{array}{cc} A_1 & A_2 \end{array} \\ \begin{bmatrix} d_{01}^{(n)} & d_{02}^{(n)} \\ d_{11}^{(n)} & d_{12}^{(n)} \\ d_{21}^{(n)} & d_{22}^{(n)} \\ \cdot & \cdot \\ \cdot & \cdot \\ \cdot & \cdot \\ d_{x1}^{(n)} & d_{x2}^{(n)} \end{bmatrix} \end{array},$$

where $d_{ij}^{(n)}$ is the probability of eliciting response A_j on trial n given sensory state s_i on that trial. Then the performance matrix specified by eqn. (1) is obtained by taking the product of the sensory matrix and the decision matrix; i.e.,

$$\mathbf{P}_n = \mathbf{S}_n \mathbf{D}_n.$$

The model that we shall examine postulates three sensory states for the two-alternative forced-choice task:

s_0 = no detection

s_1 = detection in observation interval 1

s_2 = detection in observation interval 2.

Further, the activation process and the decision process are defined by the following matrices:

$$\mathbf{S}_n = \begin{array}{c} \\ S_1 \\ S_2 \end{array} \begin{array}{c} \begin{array}{ccc} s_0 & s_1 & s_2 \end{array} \\ \begin{bmatrix} 1-\sigma & \sigma & 0 \\ 1-\sigma & 0 & \sigma \end{bmatrix} \end{array} \tag{4}$$

$$\mathbf{D}_n = \begin{array}{c} \\ s_0 \\ s_1 \\ s_2 \end{array} \begin{array}{c} \begin{array}{cc} A_1 & A_2 \end{array} \\ \begin{bmatrix} p_n & 1-p_n \\ 1 & 0 \\ 0 & 1 \end{bmatrix} \end{array}. \tag{5}$$

There are several points to note about these matrices. First, the entries in $\mathbf{S}_n$ are constants independent of the trial number; thus the sensory process is assumed to be fixed over all trials of the experiment. In contrast, the decision process may vary as a function of the trial number, and this dependence is indicated by affixing the trial index n to p. Also, s_1 can occur only if S_1 is presented, and s_2 can occur only if S_2 is presented. Thus these sensory states

have an unambiguous relation to the stimulus, since the signal event can be inferred with probability 1 when they occur. In contrast, sensory state s_0 is ambiguously related to the stimulus, for it can occur following either signal event. The parameter σ characterizes this stimulus ambiguity in the output of the sensory system. Both loss of stimulus information due to external noise and loss due to limitations on the resolving power of the sensory system are summarized by σ. Thus σ may be interpreted as a measure both of the physical stimulus and of the subject's sensitivity; σ will be referred to as the *sensitivity parameter*.

The decision matrix $\mathbf{D}_n$ reflects the relative ambiguity of the sensory states. If the subject's instructions are to make an A_i response given an S_i stimulus, then the correct response is completely determined when an s_1 or s_2 sensory state occurs. However, the subject faces a dilemma if he must make a response on the basis of s_0; either stimulus could have evoked s_0, so the subject needs some strategy by which he can resolve the ambiguity and select a response. The quantity p_n is a measure of the subject's tendency to resolve the ambiguity by making an A_1 response rather than an A_2; p_n will be referred to as the *response bias* on trial n.

For the experimental variables discussed earlier it will be assumed that the presentation schedule, information feedback and the outcome structure influence p_n, but do not affect the sensitivity parameter σ. Also, it will be assumed that the sensitivity parameter, for a given subject, is determined solely by the physical aspects of the experimental situation. It is, of course, necessary to show experimentally that these interpretations are correct, and to examine how the parameters σ and p_n are related to the physical characteristics of a given experimental situation.

In order to see how the sensitivity parameter and the bias parameter interact, consider the relation between hits and false alarms as one or the other of these parameters is manipulated. Taking the product of the matrices in eqns. (4) and (5) yields the performance matrix $\mathbf{P}_n$ for this model. The entries in the first column of $\mathbf{P}_n$ are as follows:

$$Pr(H_n) = (1-\sigma)p_n + \sigma \tag{6a}$$

$$Pr(F_n) = (1-\sigma)p_n. \tag{6b}$$

If σ is held constant and p_n is manipulated, an exchange relation is established between $Pr(H_n)$ and $Pr(F_n)$. The equation of this relation can be obtained by eliminating p_n from eqn. (6) yielding

$$Pr(H_n) = \sigma + Pr(F_n). \tag{7}$$

Thus, if σ is held constant (fixed signal and noise levels) and p_n is forced to vary (manipulations in the presentation schedule, outcome structure, etc.), the relation between hits and false alarms should be a linear function with slope 1. Plots of the relation between $Pr(H_n)$ and $Pr(F_n)$ under experimental conditions where the signal-to-noise ratio is held fixed and other variables are allowed to

vary are often referred to as receiver-operating-characteristic curves, or more simply as ROC curves.

If p_n is held constant and the sensitivity parameter changed, there is a well-defined relation between hits and false alarms. Eliminating σ from eqn. (6) yields

$$Pr(H_n) = 1 - Pr(F_n)\left[\frac{1-p_n}{p_n}\right]. \tag{8}$$

Plots of the relation between $Pr(H_n)$ and $Pr(F_n)$ when p_n is constant and σ is varied are called *iso-bias curves.*

Learning Process

As indicated earlier, an important feature of the present analysis is to represent changes in the bias probability in terms of a learning process of the type proposed by Bush and Mosteller (1955). We assume that the bias on trial $n+1$ is a linear function of its value on trial n. Specifically, if s_n occurs and is followed by E_1 (i.e., the experimenter informs the subject that the signal was in the first interval) then p_n will increase. If s_0 occurs and is followed by information event E_2, then p_n will decrease. For all other contingencies no change will occur in p_n. These statements can be summarized as follows:

$$p_{n+1} = \begin{cases} (1-\theta)p_n + \theta, & \text{if } s_{0.n} \;\&\; E_{1.n} \\ (1-\theta')p_n, & \text{if } s_{0.n} \;\&\; E_{2.n} \\ p_n, & \text{otherwise,} \end{cases} \tag{9}$$

where $0 < \theta, \theta' \leq 1$. Justification for this equation is postponed until later.

We now want to derive an expression for the expected value of p_n as a function of the presentation schedule and the sensitivity parameter. Recall that γ is the probability of an S_1 signal event and $1-\sigma$ is the probability of activating sensory state s_0 given either S_1 or S_2. Hence

$$\begin{aligned} Pr(s_{0.n} \;\&\; E_{1.n}) &= \gamma(1-\sigma) \\ Pr(s_{0.n} \;\&\; E_{2.n}) &= (1-\gamma)(1-\sigma) \\ Pr(\text{otherwise}) &= \sigma. \end{aligned}$$

To compute the expected value of the bias probability on trial $n+1$, simply weight each of the possible outcomes listed in eqn. (9) by its probability of occurrence given above. That is, the expected value on trial $n+1$ given a fixed value p_n on trial n is

$$\begin{aligned} E(p_{n+1}) &= \gamma(1-\sigma)[(1-\theta)p_n + \theta] + (1-\gamma)(1-\sigma)(1-\theta')p_n + \sigma p_n \\ &= [1-(1-\sigma)\{\theta\gamma + \theta'(1-\gamma)\}]p_n + \theta\gamma(1-\sigma). \end{aligned}$$

It can be shown that p_n in the above equation can be replaced by its expected value (Atkinson, Bower and Crothers, 1965). Consequently we have a linear first-order difference equation in $E(p_n)$ which has the solution

$$E(p_n) = p_\infty - (p_\infty - p_1)G^{n-1}.$$

where

$$p_\infty = \frac{\gamma}{\gamma + (1-\gamma)\phi}, \tag{10}$$

$$G = 1 - (1-\sigma)[\theta\gamma + \theta'(1-\gamma)]$$

and $\phi = \theta'/\theta$. Note that p_∞, which is defined as $\lim_{n\to\infty} E(p_n)$, does not depend on the absolute values of θ and θ' but only on their ratio.

Combining the results in eqns. (6) and (10) yields

$$Pr(H_n) = \sigma + (1-\sigma)[p_\infty - (p_\infty - p_1)G^{n-1}] \tag{11 a}$$

$$Pr(F_n) = (1-\sigma)[p_\infty - (p_\infty - p_1)G^{n-1}]. \tag{11 b}$$

From these equations it is clear that hits and false alarms will depend on p_1 at the start of an experimental session; however, over trials the subject's performance changes at a rate controlled by the quantity G, and approaches an asymptote determined by σ and p_∞. The change in performance predicted by eqn. (11) is a well-known experimental phenomenon. Generally, however, most research workers have tended to ignore the changes that occur at the beginning of an experimental session, and instead have concentrated on an analysis of data after performance has settled down to a stable level. For the experiments analysed in this paper we shall adopt this policy; to do so makes matters simpler because fewer parameters need to be estimated. Since asymptotic performance will be stressed in subsequent discussions, the following notation will be useful:

$$\lim_{n\to\infty} Pr(H_n) = Pr(H)$$

$$\lim_{n\to\infty} Pr(F_n) = Pr(F).$$

That is, asymptotic expressions will be indicated by simply deleting the trial subscript. Making the appropriate substitutions in eqn. (11) yields

$$Pr(H) = \sigma + \frac{(1-\sigma)\gamma}{\gamma + (1-\gamma)\phi} \tag{12 a}$$

$$Pr(F) = \frac{(1-\sigma)\gamma}{\gamma + (1-\gamma)\phi}. \tag{12 b}$$

Similarly, for the asymptotic proportion of correct responses (see eqn. (3))

$$Pr(C) = \sigma + (1-\gamma)(1-\sigma) + \frac{(1-\sigma)\gamma(2\gamma-1)}{\gamma + (1-\gamma)\phi}; \tag{13}$$

and for the asymptotic proportion of A_1 responses (see eqn. (2)),

$$Pr(A_1) = \gamma\sigma + \frac{\gamma(1-\sigma)}{\gamma + (1-\gamma)\phi}. \tag{14}$$

3. Experimental Manipulation of the Presentation Schedule

We now examine data collected from eight subjects in a forced-choice acoustic detection experiment. In this study the signal and noise levels were held

constant throughout the experiment and the subject was always given information at the end of each trial regarding the correctness of his response. The only experimental manipulation involved the use of three different presentation schedules. The probability, γ, of an S_1 event took on the following values:

Schedule A: $\gamma = 0{\cdot}25$

Schedule B: $\gamma = 0{\cdot}50$

Schedule C: $\gamma = 0{\cdot}75$.

METHOD

Test sessions of 350 trials each were run on consecutive days. Each day a subject ran on one of the three schedules for the entire session. In successive 3-day blocks a subject ran one day on each of the three schedules; within each 3-day block the order was randomly determined. The experiment involved 15 experimental sessions and therefore each schedule was run on five separate days.

Band-limited Gaussian noise was presented binaurally in the subject's headphones throughout a test session and the signal was a 1,000 c.p.s. sinusoidal tone; the tone was presented for 100 msec, including equal fall and rise times of 20 msec. The subject was seated before a display board. On each trial three lights flashed on briefly in succession: a red light, an amber light, and another amber light. Each light was on for 100 msec with a 500 msec delay between each successive on-period. The red light was simply a warning light, while the amber lights defined two observation intervals. The onset of the signal occurred simultaneously with the onset of one of the amber lights. After the second amber light went off the subject had 2·5 sec to indicate his response by pressing a push-button located under the appropriate amber light. At the conclusion of the response period a green light flashed on for 700 msec above the correct response button. There was a 1·5 sec intertrial period, thus each trial lasted for 6 sec.

RESULTS

Table 1 presents the proportion of A_1 responses on both S_1 and S_2 trials over the last 250 trials of replications two through five of each presentation schedule; thus each estimate is based on $250 \times 4 = 1{,}000$ trials. The first replication of each presentation schedule has been deleted, because we view the subject as adapting to the detection task on early days of the experiment and want to treat his data only after he clearly understands the experimental routine and is well practised. Also, the first 100 trials of each of the subsequent experimental sessions were deleted because, as noted earlier, our analyses are going to be restricted to asymptotic performance.

In this experiment the signal and noise levels were constant over all sessions and only the presentation schedule varied. Therefore, σ should be fixed throughout the experiment, but p_∞ should vary with changes in γ. It has already been shown that hits and false alarms should fall on the straight line $Pr(H) = \sigma + Pr(F)$. We now wish to fit this equation to the three data points corresponding to presentation schedules A, B and C. Figure 1 presents plots of $Pr(H)$ and $Pr(F)$ for individual subjects. In order to fit the above equation to the three points for each subject we use the method of least squares, i.e., σ is selected so that it minimizes the sum of squared deviations between observed values and those

TABLE 1. PREDICTED AND OBSERVED PROPORTIONS OF $Pr(H)$, $Pr(F)$, $Pr(C)$ AND $Pr(A_1)$. (The observed proportions are in parentheses)

Subject	*Schedule A*				*Schedule B*				*Schedule C*			
	$Pr(H)$	$Pr(F)$	$Pr(C)$	$Pr(A_1)$	$Pr(H)$	$Pr(F)$	$Pr(C)$	$Pr(A_1)$	$Pr(H)$	$Pr(F)$	$Pr(C)$	$Pr(A_1)$
1	0·601	0·154	0·785	0·266	0·744	0·297	0·724	0·521	0·877	0·430	0·800	0·765
	(0·622)	(0·163)	(0·783)	(0·278)	(0·714)	(0·260)	(0·727)	(0·487)	(0·890)	(0·462)	(0·802)	(0·783)
2	0·543	0·125	0·792	0·229	0·680	0·262	0·709	0·471	0·832	0·414	0·771	0·727
	(0·529)	(0·136)	(0·780)	(0·234)	(0·654)	(0·249)	(0·702)	(0·451)	(0·854)	(0·397)	(0·791)	(0·740)
3	0·597	0·106	0·820	0·229	0·716	0·225	0·746	0·470	0·849	0·358	0·797	0·726
	(0·626)	(0·107)	(0·826)	(0·237)	(0·707)	(0·210)	(0·748)	(0·459)	(0·842)	(0·384)	(0·786)	(0·728)
4	0·529	0·127	0·787	0·227	0·669	0·267	0·701	0·486	0·825	0·424	0·763	0·725
	(0·517)	(0·122)	(0·788)	(0·221)	(0·649)	(0·242)	(0·703)	(0·446)	(0·857)	(0·454)	(0·779)	(0·756)
5	0·520	0·120	0·790	0·220	0·658	0·258	0·709	0·458	0·816	0·416	0·758	0·716
	(0·546)	(0·142)	(0·780)	(0·243)	(0·650)	(0·240)	(0·705)	(0·445)	(0·799)	(0·413)	(0·746)	(0·703)
6	0·542	0·141	0·780	0·241	0·689	0·287	0·701	0·488	0·841	0·440	0·771	0·741
	(0·547)	(0·139)	(0·783)	(0·241)	(0·650)	(0·279)	(0·701)	(0·479)	(0·847)	(0·451)	(0·772)	(0·748)
7	0·618	0·125	0·810	0·249	0·744	0·252	0·746	0·498	0·872	0·379	0·809	0·749
	(0·627)	(0·136)	(0·805)	(0·259)	(0·742)	(0·251)	(0·746)	(0·496)	(0·864)	(0·369)	(0·806)	(0·740)
8	0·570	0·125	0·799	0·236	0·704	0·258	0·723	0·481	0·847	0·401	0·785	0·735
	(0·552)	(0·108)	(0·807)	(0·219)	(0·687)	(0·244)	(0·722)	(0·465)	(0·887)	(0·438)	(0·806)	(0·775)
Average	0·565	0·128	0·795	0·237	0·703	0·263	0·719	0·482	0·845	0·408	0·782	0·735
	(0·571)	(0·132)	(0·794)	(0·241)	(0·685)	(0·247)	(0·719)	(0·466)	(0·855)	(0·421)	(0·786)	(0·746)

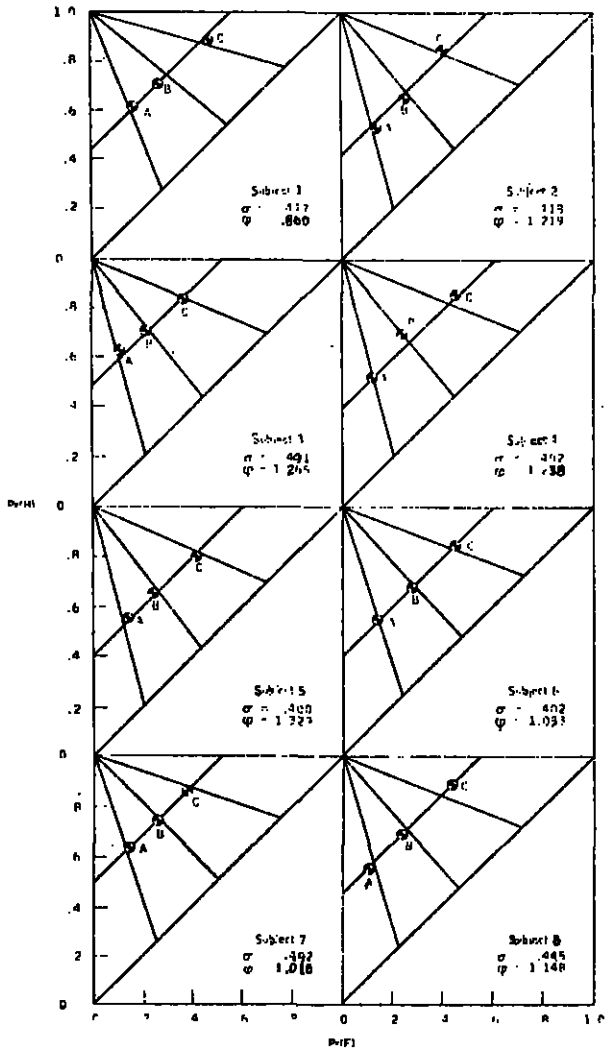

FIGURE 1. Observed and predicted values for $Pr(H)$ and $Pr(F)$.

predicted by the above equation. Applying the least squares method yields the estimates of σ that are given in Figure 1; these estimates were used to generate the ROC curves displayed in the figure. As indicated by the figures there is good agreement between the observed data points and the predicted ROC curves. Recall that the signal and noise levels were the same for all subjects

and consequently variations in σ represent inter-subject differences in sensitivity level.

Next we evaluate the proposed bias process with regard to the data presented in Table 1. Note that if γ and σ are fixed in eqn. (12) and ϕ is varied from 0 to ∞, then the point $[Pr(F), Pr(H)]$ moves along the ROC curve and approaches the lower-left point $(0, \sigma)$ as $\phi \to \infty$, and the upper-right point $(1-\sigma, 1)$ as $\phi \to 0$. Stated differently, no matter where the point may fall on the ROC curve (for fixed values of γ and σ) there exists a corresponding value of ϕ. Hence, if the three observed points $[Pr(F), Pr(H)]$ fall on a straight line with slope 1, then perfect fits of the data can be obtained by estimating separate values of ϕ for each presentation schedule.

However, obtaining an estimate of ϕ for each presentation schedule would violate the basic rationale for the model. In formulating eqn. (9) it was assumed that θ and θ' characterize trial-to-trial adjustments to stimulus and information events, and did not depend on the overall presentation schedule. The values of θ and θ' may vary from subject to subject reflecting individual differences; however, for a given subject θ and θ' are assumed to be fixed and invariant with regard to the presentation schedule and the signal intensity. Earlier it was assumed that σ was independent of the presentation schedule, and the same constraint is placed on ϕ. Thus for each subject we want a single estimate of ϕ which then can be used to make predictions for all three presentation schedules.

The observed proportion of A_1 responses given in Table 1 was used to estimate ϕ. Equation (14) gives the theoretical expression for $Pr(A_1)$; solving for ϕ yields

$$\phi = \frac{\gamma(1-\sigma)}{[Pr(A_1) - \sigma\gamma](1-\gamma)} - \frac{\gamma}{1-\gamma}.$$

For each presentation schedule we have substituted the estimated value of σ and the observed value of $Pr(A_1)$ in the above equation to obtain an estimate of ϕ. For example, for Subject 1, $\sigma = 0{\cdot}447$, $Pr(A_1) = 0{\cdot}278$, and $\gamma = 0{\cdot}25$ on schedule A; hence substituting in the above equation yields $\hat{\phi}_A = 0{\cdot}777$. Similarly $\hat{\phi}_B$ and $\hat{\phi}_C$ can be computed using the appropriate values of γ and $Pr(A_1)$. An overall

TABLE 2. ESTIMATES OF ϕ

Subject	$\hat{\phi}$	$\hat{\phi}_A$	$\hat{\phi}_B$	$\hat{\phi}_C$
1	0·860	0·777	1·099	0·705
2	1·219	1·162	1·400	1·096
3	1·265	1·155	1·390	1·251
4	1·238	1·324	1·446	0·945
5	1·329	1·065	1·449	1·472
6	1·083	1·085	1·147	1·018
7	1·016	0·914	1·028	1·105
8	1·148	1·384	1·264	0·775
Average	1·145	1·108	1·280	1·046

estimate of ϕ was obtained for each subject by taking the average of the three estimates; namely $\hat{\phi} = \frac{1}{3}(\hat{\phi}_A + \hat{\phi}_B + \hat{\phi}_C)$. The various estimates of $\hat{\phi}$ are presented in Table 2. Note that for all but one subject $\hat{\phi}$ is greater than one, indicating that $\theta' > \theta$. The interpretation of this result is that the E_2 event has a slightly greater effect on increasing the probability of an A_2 response than the E_1 event has on increasing the probability of an A_1 response.

Using the estimates of σ and ϕ, predictions can be computed for $Pr(H)$, $Pr(F)$, $Pr(C)$ and $Pr(A_1)$ from eqns. (12) to (14). These predicted values and the corresponding observed quantities are presented in Table 1. Also in Figure 1 the predicted and observed values of $Pr(H)$ and $Pr(F)$ are plotted in the ROC space. In this figure the predicted point for each presentation schedule is at the intersection of the predicted iso-bias curve and the ROC curve. Overall, the correspondence between predicted and observed values is quite good. Only Subject 8 appears to display systematic discrepancies. To a degree, this subject's performance deviated from the theoretical values in the direction of optimizing the probability of a correct response; that is, for fixed σ, to maximize the probability of a correct response the subject should set the bias parameter at unity when $\gamma > \frac{1}{2}$, and at zero when $\gamma < \frac{1}{2}$ (see eqn. (13)). If the subject adopted this strategy, then the ROC curve would reduce to three points; one at $(0, \sigma)$ for $\gamma < \frac{1}{2}$, another at $(1-\sigma, 0)$ for $\gamma > \frac{1}{2}$, and a third point for the presentation schedule where $\gamma = \frac{1}{2}$. Undoubtedly if monetary payoffs for correct responses and penalties for incorrect responses were introduced into the experimental situation, more subjects would deviate from the theoretical values in the direction of optimization. We shall return to a discussion of this point later.

Time-order Effect

In the forced-choice detection task the term *time-order effect* is used to refer to the fact that subjects generally are more accurate in detecting signals embedded in the second observation interval than in the first. For example, on schedule B (which has S_1 and S_2 events occurring equally often), every subject had a higher probability of being correct when the signal was in the second interval than in the first interval. In terms of the present analysis there are two explanations for this time-order effect. One is that the bias parameter tends to favour the A_2 response. Hence when sensory state s_0 is activated, the subject makes the A_2 response more frequently, which insures that he will have a higher probability of being correct on S_2 than on S_1 trials. Another possibility is that the time-order effect occurs because the subject's sensitivity level changes from one observation interval to the next; specifically, that there are two sensitivity parameters σ_1 and σ_2 associated with the two intervals and $\sigma_2 > \sigma_1$. Thus a time-order effect can be accounted for by postulating a bias process that tends to favour the A_2 response, or by postulating a sensory mechanism that is more sensitive to stimuli presented in the second observation interval.

Both of these explanations are tenable and one would like to have some means for selecting between them; fortunately the model makes quite different

predictions depending on which explanation is offered. If the explanation is in terms of the bias function (as was the case in our analysis of these data) then the ROC curve has slope 1 and the time-order effect is simply due to the fact that $\phi > 1$. If, however, the effect is explained in terms of different sensitivity levels, then

$$Pr(H) = \sigma_1 + (1 - \sigma_1)p$$
$$Pr(F) = (1 - \sigma_2)p.$$

Under these conditions the ROC curve is

$$Pr(H) = \frac{1 - \sigma_1}{1 - \sigma_2} Pr(F) + \sigma_1.$$

If $\sigma_2 > \sigma_1$ the slope of the ROC curve is greater than one. Thus to decide whether the time-order effect is due to the bias process alone, or whether it also may be due to differential sensitivity levels, we must determine whether the ROC curve has slope greater than one. Inspection of Figure 1 indicates that there is no evidence (except possibly for Subject 2) to suggest that the observed points would be better fit by a line with slope greater than one. Therefore, for this experiment, the conclusion is that the time-order effect is due to the bias process, and there is no need to postulate changes in sensitivity over the two observation intervals.

4. Blank Trials and False Information

We now examine two modifications of the forced-choice detection task used in the previous experiment. One involves the introduction of blank trials and the other the use of false-information feedback. By *blank trials* we mean that on occasion a trial will occur on which the signal has been omitted entirely; the subject is not told that blank trials are being introduced and (because of the forced-choice nature of the task) continues to make A_1 and A_2 responses. A blank trial will be denoted as S_0. By *false-information feedback* we mean that on some trials the subject will be told that a signal occurred in a particular observation interval when in fact it did not. The introduction of these two modifications in the detection task permits us to make some sharp predictions that differentiate this model from others with similar assumptions.

In the present experiment the subject was given the same instructions that were used in the first experiment, i.e., he was told that a signal would occur on every trial and that the information events at the end of each trial indicated the interval in which the signal occurred. Actually, however, the presentation schedule involved S_1, S_2 and S_0 type trials; on S_1 trials an E_1 always occurred, on S_2 trials an E_2 always occurred, and on S_0 trials sometimes E_1 occurred and sometimes E_2. The presentation schedule used in this study can be characterized by the parameters γ, π and x as follows: (a) with probability $x\gamma$ a signal was presented in the first interval and, after the response, E_1 occurred, (b) with probability $x(1 - \gamma)$ a signal was presented in the second interval and followed by

E_2, and (c) with probability $1-x$ a blank trial was presented and an E_1 occurred with probability π and an E_2 event with probability $1-\pi$. Thus, the probability of presenting a signal in the first interval was $x\gamma$; but the probability of telling the subject that the signal occurred in the first interval was $Pr(E_{1,n}) = x\gamma + (1-x)\pi$. Similarly, the probability of presenting the signal in the second interval was $x(1-\gamma)$; however, the probability that the subject was told that the signal occurred in the second interval was $Pr(E_{2,n}) = x(1-\gamma) + (1-x)(1-\pi)$. The model presented earlier is directly applicable to this experiment. No new assumptions are necessary; we need only apply the axioms and carry out the appropriate derivations. First of all, consider the sensory matrix for this experiment. In terms of the assumptions

$$\mathbf{S}^* = \begin{array}{c} \\ S_1 \\ S_2 \\ S_0 \end{array} \begin{array}{c} \begin{array}{ccc} s_0 & s_1 & s_2 \end{array} \\ \begin{bmatrix} 1-\sigma & \sigma & 0 \\ 1-\sigma & 0 & \sigma \\ 1 & 0 & 0 \end{bmatrix} \end{array}.$$

Using the matrix $\mathbf{S}^*$ and the decision matrix $\mathbf{D}_n$ specified by eqn. (5), a performance matrix $\mathbf{P}_n^*$ can be derived whose rows are the events S_1, S_2 and S_0 and whose columns are the responses A_1 and A_2. The entries in the first column of the matrix $\mathbf{P}_n^*$ are

$$Pr(H_n) = Pr(A_{1,n} \mid S_{1,n}) = \sigma + (1-\sigma)p_n \tag{15a}$$

$$Pr(F_n) = Pr(A_{1,n} \mid S_{2,n}) = (1-\sigma)p_n \tag{15b}$$

$$Pr(A_{1,n} \mid S_{0,n}) = p_n. \tag{15c}$$

From eqns. (15a) and (15b) it is clear that the ROC curve is the same as one given in eqn. (7) for the first experiment. Also, from eqns. (15a) and (15c) it follows that $Pr(H_n)$ and $Pr(A_{1,n} \mid S_{0,n})$ are linearly related as follows:

$$Pr(H_n) = \sigma + (1-\sigma)Pr(A_{1,n} \mid S_{0,n}). \tag{16}$$

Equation (9) presented the axioms describing possible changes in p_n. These axioms are directly applicable to the present experiment. Given eqn. (9) we need only to compute the probability of the events ($s_{0,n}$ & $E_{1,n}$) and ($s_{0,n}$ & $E_{2,n}$). The tree in Figure 2 describes the possible events that can occur on a given trial. From the figure we obtain

$$Pr(s_{0,n} \,\&\, E_{1,n}) = x\gamma(1-\sigma) + (1-x)\pi$$

$$Pr(s_{0,n} \,\&\, E_{2,n}) = x(1-\gamma)(1-\sigma) + (1-x)(1-\pi)$$

$$Pr(\text{otherwise}) = x\sigma.$$

Given these results an expression can be derived for $E(p_n)$. We shall not carry out the derivation, for it involves precisely the same arguments that were employed in developing eqn. (10). Invoking these arguments yields the following equation:

$$E(p_n) = p_\infty - (p_\infty - p_1)G^{n-1}.$$

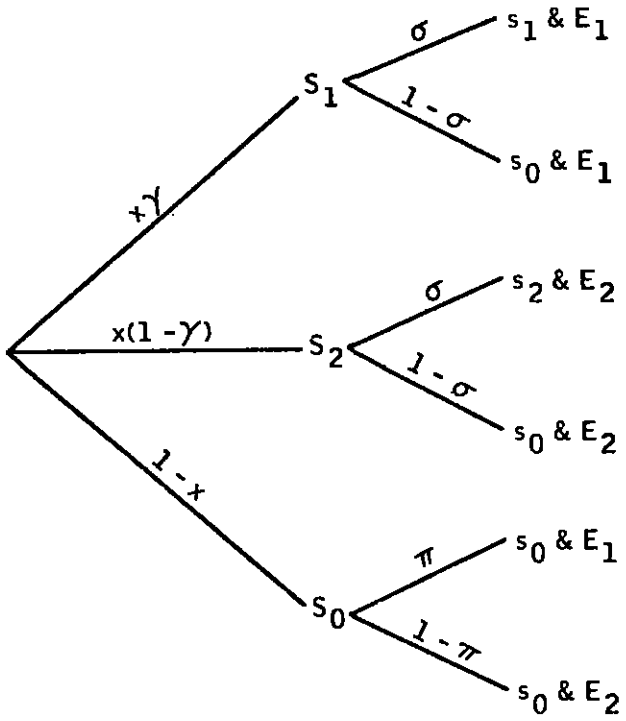

FIGURE 2. A tree describing possible events and their related probabilities for the blank-trial experiment.

Here

$$G = 1 - \theta[x\gamma(1-\sigma) + (1-x)\pi] - \theta'[x(1-\gamma)(1-\sigma) + (1-x)(1-\pi)],$$

and

$$p_{\cdot 0} = \frac{x\gamma(1-\sigma) + (1-x)\pi}{[x\gamma(1-\sigma) + (1-x)\pi] + [x(1-\gamma)(1-\sigma) + (1-x)(1-\pi)]\phi}, \tag{17}$$

where $\phi = \theta'/\theta$.

METHOD

The same experimental procedures were employed in this study as in the first one except for the pretraining phase. Pretraining took three days and involved running each subject on the schedule B routine used in the first experiment. The signal intensity was held fixed throughout the experiment, but during pretraining the experimenter manipulated the noise level in an attempt to establish a signal-to-noise ratio for each subject that yielded a correct response percentage of approximately 79; the rationale for selecting this particular value will be given later. The manipulation of the noise was done strictly by trial and

error, but the procedure proved to be quite successful for by the end of pretraining a level had been established for each subject that yielded a correct response probability fairly close to the desired value. During the remainder of the experiment the noise level was fixed for each subject at the value determined for him during pretraining. Also, any subject who tended to strongly favour one response over the other, during pretraining, was eliminated from the experiment. Only subjects whose overall proportion of A_1 responses was between 0·40 and 0·60 for the second and third days of pretraining were included in the main experiment. Four subjects from a group of 18 were eliminated on this basis. Pretraining, therefore, involved two special features: (*a*) noise levels were determined individually for each subject, and (*b*) subjects were eliminated from the experiment who showed a strong preference for one of the response alternatives. The first requirement guaranteed that the sensitivity parameter σ was approximately the same for all subjects. The second insured that ϕ was fairly close to 1 for all subjects. Thus, in a rough sense, a homogeneous group of subjects was formed by using this pretraining procedure; *homogeneous in the sense* that all subjects were characterized by approximately the same values of σ and ϕ.

In the experiment proper, four presentation schedules were used. The probability π of a signal trial was 0·50 for all schedules, but the schedules differed in the values of γ and π as follows:

	$\pi=0{\cdot}25$	$\pi=0{\cdot}75$
$\gamma=0{\cdot}25$	Schedule A′	Schedule C′
$\gamma=0{\cdot}75$	Schedule B′	Schedule D′

Test sessions of 400 trials were run on consecutive days. Each day a subject ran on one of the above presentation schedules for the entire session. In successive 4-day blocks a subject completed one day on each of the four schedules; within each 4-day block the order of schedules was randomly determined. The experiment involved 20 test sessions and therefore each schedule was repeated on five separate days.

RESULTS

Table 3 presents the average proportion of A_1 responses conditional upon the various trial types; these averages are based on 14 subjects. Proportions

TABLE 3. OBSERVED AND PREDICTED VALUES FOR THE BIASS-TRIAL STUDY

	Schedule A′		*Schedule B′*		*Schedule C′*		*Schedule D′*	
	Obs.	*Pred.*	*Obs.*	*Pred.*	*Obs.*	*Pred.*	*Obs.*	*Pred.*
$Pr(H)$	0·641	0·672	0·755	0·734	0·820	0·820	0·903	0·886
$Pr(F)$	0·086	0·100	0·174	0·162	0·227	0·248	0·344	0·314
$Pr(A_1 \mid S_0)$	0·213	0·234	0·401	0·378	0·553	0·578	0·765	0·733
$Pr(A_1)$	0·219	0·238	0·505	0·485	0·464	0·484	0·764	0·738

were computed for each subject based on the last 350 trials of replications two through five of a given presentation schedule; thus the estimates for each subject are based on a sequence of $4 \times 350 = 1{,}400$ trials. The averages of these individual subject proportions are the quantities presented in the table. Although data were analysed for individual subjects in the first experiment, there is a theoretical rationale for treating group data in the present experiment. The rationale is based on the pretraining procedure, which was designed to insure that both σ and ϕ would be approximately the same for all subjects. By inspection of eqns. (15) and (17) we see that $Pr(H)$, $Pr(F)$ and $Pr(A_1 \mid S_0)$ depend on only

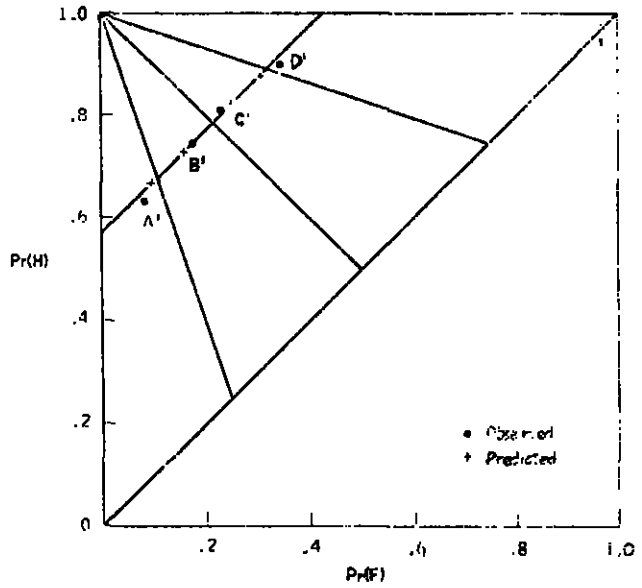

FIGURE 3. Observed and predicted values for $Pr(H)$ and $Pr(F)$.

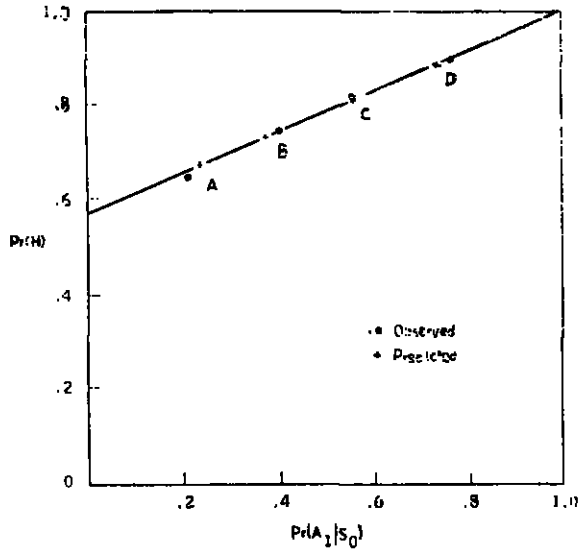

FIGURE 4. Observed and predicted values for $P(H)$ and $P(A_1 \mid S_0)$.

σ and ϕ. If σ and ϕ are identical for all subjects, then the model makes the same predictions for the group average as for individual subjects.

Figure 3 presents plots of the observed values of $Pr(H)$ and $Pr(F)$ as given in Table 3. The theory predicts that these points should fall on a linear curve with slope 1 and intercept σ. We estimated σ from these four data points by using the method of least squares and obtained $\hat{\sigma} = 0{\cdot}572$. This estimate was used to generate the ROC curve displayed in Figure 3. The four observed points (one from each schedule) fall fairly close to the predicted line.

Figure 4 presents a plot of $Pr(A_1 \mid S_n)$ versus $Pr(H)$. As indicated in eqn. (16) these points should be related by a linear function with slope $1-\sigma$ and intercept σ. The straight line in Figure 4 was generated using our previous estimate of σ. Once again the linear relation seems to be reasonably well supported.

To generate numerical predictions for $Pr(A_1 \mid S_i)$ an estimate of ϕ is required in addition to the estimate of σ. Estimation of this parameter is attained using the same method employed earlier. The overall probability of an A_1 response is

$$\begin{aligned} Pr(A_1) &= \sigma\gamma Pr(A_1 \mid S_1) + \sigma(1-\gamma)Pr(A_1 \mid S_2) + (1-\sigma)Pr(A_1 \mid S_0) \\ &= \sigma\gamma + (1-\sigma)p_{\pi}. \end{aligned} \tag{18}$$

Substituting in the expression for p_π given in eqn. (17) yields an expression in ϕ. For each presentation schedule we have substituted the estimated value of σ and the observed value of $Pr(A_1)$ in the above equation and solved for ϕ. For example, for schedule A' the observed value of $Pr(A_1)$ is 0·219; letting $\hat{\sigma} = 0{\cdot}572$, $\gamma = 0{\cdot}25$ and $\pi = 0{\cdot}25$ in the above equation yields $\hat{\phi}_{A'} = 1{\cdot}281$. Similarly, for the other schedules we obtain $\hat{\phi}_{B'} = 0{\cdot}969$, $\hat{\phi}_{C'} = 1{\cdot}229$ and $\hat{\phi}_{D'} = 0{\cdot}897$. It is interesting to note that $\hat{\phi}$ seems to be correlated more with γ than with π. Schedules A' and C' ($\gamma = 0{\cdot}25$) both yield $\hat{\phi} > 1$, whereas schedules B' and D' ($\gamma = 0{\cdot}75$) yield $\hat{\phi} < 1$. Recall that $\phi = \theta'/\theta$ and that γ is the probability of a signal in the first interval (if there is a signal). The present estimates of ϕ suggest that θ' is greater than θ if the probability of the signal being in the second interval exceeds $\frac{1}{2}$, whereas the reverse relation holds otherwise. Hence the *change in the bias parameter* p_π seems to be dominated by the interval with the higher probability of bracketing the signal. Despite this departure from independence of the parameters ϕ and γ, very little damage is done to the accuracy of the predictions from the model, as will be seen shortly.

To obtain an overall estimate of ϕ we have taken the average of the separate estimates of ϕ, i.e.,

$$\begin{aligned} \hat{\phi} &= \tfrac{1}{4}(\hat{\phi}_{A'} + \hat{\phi}_{B'} + \hat{\phi}_{C'} + \hat{\phi}_{D'}) \\ &= 1{\cdot}094. \end{aligned}$$

With these estimates of σ and ϕ, eqns. (15) and (17) can now be used to generate predictions for $Pr(H)$, $Pr(F)$, $Pr(A_1 \mid S_0)$ and $Pr(A_1)$. These predicted quantities are given in Table 3; they also are displayed in Figures 3 and 4 as cross marks on the appropriate line segments. There are no constraints on the relations

among the quantities $Pr(A_1 \mid S_1)$, $Pr(A_1 \mid S_2)$ and $Pr(A_1 \mid S_0)$, and therefore twelve independent predictions are being made on the basis of two parameters. An inspection of the array of observed and predicted quantities indicates that the correspondence between theoretical and observed values is quite satisfactory.

For both schedules B′ and C′ the E_1 and E_2 events occurred equally often, i.e., on both schedules the subject was told (via the trial-to-trial feedback) that the signal was occurring equally often in the two observation intervals. However, the signal actually occurred more frequently in the first interval for schedule B′ than for schedule C′. These experimental manipulations are clearly reflected in the data. On an S_0 trial the probability of an A_1 response was greater for schedule C′ than for schedule B′ (0·553 *vs.* 0·401), whereas over all trials the probability of an A_1 response was greater for schedule B′ than for schedule C′ (0·505 *vs.* 0·464). *Both of these relations are predicted by the model.*

Sequential Effects

The model predicts not only hit and false-alarm rates but also sequential properties of response protocols. In terms of the axioms, sequential effects in the observable response events are produced by trial-to-trial fluctuations in p_n. Such fluctuations, of course, can take place on any trial and are not restricted to pre-asymptotic data. For example, even at asymptote the likelihood of making a correct response to an S_1 stimulus depends in a very definite way on whether an E_1 or an E_2 occurred on the preceding trial. The sequential effects of particular interest deal with the influence of stimulus and response events on trial n as they influence the response on trial $n+1$; specifically

$$Pr(A_{1,n+1} \mid S_{t,n+1} A_{j,n} S_{k,n}).$$

However, we shall not examine the correspondence between these particular sequential effects and theoretical predictions, because there are 18 such independent quantities for each of the experimental conditions and the analysis would involve too much detail. Rather, we consider $Pr(A_{1,n+1} \mid E_{1,n})$ and $Pr(A_{1,n+1} \mid E_{2,n})$. For these probabilities the stimulus events on trials n and $n+1$ are suppressed, and we only ask for the overall likelihood of an A_1 response conditional on the information event of the preceding trial. The A_1 could be elicited by S_1, S_2, or S_0 on trial $n+1$; similarly the information event E_1 on trial n could follow an S_1 or S_0 stimulus, and the E_2 an S_2 or S_0 stimulus. Asymptotic expressions for these quantities can be readily obtained (see Atkinson, Bower and Crothers, 1965) and are as follows:

$$\begin{aligned}
\lim_{n\to\infty} Pr(A_{1,n+1} \mid E_{1,n}) &= Pr(A_1) + (1-\alpha x)\theta(1-p_\infty)\,\frac{\pi(1-x)+x\gamma(1-\sigma)}{\pi(1-x)+x\gamma} \\
\lim_{n\to\infty} Pr(A_{1,n+1} \mid E_{2,n}) &= Pr(A_1) - (1-\alpha x)\theta' p_\infty\,\frac{(1-\pi)(1-x)+x(1-\gamma)(1-\sigma)}{(1-\pi)(1-x)+x(1-\gamma)},
\end{aligned} \tag{19}$$

where p_∞ is given by eqn. (17) and $Pr(A_1)$ by eqn. (18).

Table 4 presents the observed values for $Pr(A_{1,n+1} \mid E_{1,n})$ and $Pr(A_{1,n+1} \mid E_{2,n})$. Estimates of these quantities were obtained for individual subjects; the average of these estimates are the quantities presented in the table. These

TABLE 4. OBSERVED AND PREDICTED SEQUENTIAL QUANTITIES FOR THE BLANK-TRIAL STUDY

	Schedule A'		*Schedule B'*		*Schedule C'*		*Schedule D'*	
	Obs.	*Pred.*	*Obs.*	*Pred.*	*Obs.*	*Pred.*	*Obs.*	*Pred.*
$\lim_{n\to\infty} Pr(A_{1,n+1} \mid E_{1,n})$	0·255	0·267	0·529	0·503	0·475	0·503	0·784	0·748
$\lim_{n\to\infty} Pr(A_{1,n+1} \mid E_{2,n})$	0·207	0·229	0·482	0·466	0·453	0·466	0·716	0·708

estimates are based on the same set of trials as the data presented in Table 3 and therefore will be regarded as asymptotic. The above equations can be used to generate predictions for these observed values. By inspection of the equations we see that values are needed for σ, θ and θ' in order to make numerical predictions. Since estimates of σ and ϕ have already been made, it is only necessary to estimate θ'; that is, if we fix on some value of θ' then θ is determined because θ'/θ must equal the previous estimate of $\phi = 1{\cdot}094$. For present purposes, one method for estimating θ' is to select its value so as to minimize the sum of squared deviations between the eight predicted and observed quantities displayed in Table 4. To carry out this minimization analytically yields unwieldy expressions, and to avoid this complication we have simply calculated the sum of the eight squared deviations for θ' ranging from 0·01 to 1·00 in successive increments of 0·01. Over this range of values the sum of squared deviations takes on its minimum when $\theta' = 0{\cdot}08$. This value of θ' was used to generate the predictions in Table 4.

In general, the correspondence between predicted and observed sequential statistics is reasonably good. In evaluating the goodness-of-fit it should be kept in mind that all of the quantities in the table are independent, and thus there are eight degrees of freedom. The model requires that $Pr(A_{1,n+1} \mid E_{1,n}) > Pr(A_1) > Pr(A_{1,n+1} \mid E_{2,n})$; and this relation is supported by all four sets of data. Also the model requires that $Pr(A_{1,n+1} \mid S_{i,n+1} E_{1,n}) > Pr(A_{1,n+1} \mid S_{i,n+1} E_{2,n})$ for $i = 0, 1, 2$. Although not presented here, a breakdown of the data into this form indicates that these inequalities hold over all four experimental conditions.

5. DISCUSSION

An alternative model for the bias process that has considerable intuitive appeal involves trial-by-trial changes in p_n that are determined solely by the information events E_1 and E_2. Formally stated, the idea is that

$$p_{n+1} = \begin{cases} (1-\theta)p_n + \theta, & \text{if } E_{1,n} \\ (1-\theta')p_n, & \text{if } E_{2,n} \end{cases}. \tag{20}$$

This formulation of the bias process (which will be called Model 2) is to be contrasted with eqn. (9) (Model 1), where changes in p_n can occur only when

sensory state s_0 is activated. In spite of the difference between these two sets of assumptions, the models yield identical predictions in the first experiment for the asymptotic probabilities of $Pr(H)$, $Pr(F)$, $Pr(A_1)$ and $Pr(C)$. Only by a detailed analysis of sequential statistics and pre-asymptotic data can it be shown that Model 1 is slightly better than Model 2.

However, the two models make strikingly different predictions in the second experiment even for asymptotic hit and false-alarm proportions. For example, applying Model 2 to the false-information study yields

$$p_{\infty} = \frac{x\gamma + (1-x)\pi}{[x\gamma + (1-x)\pi] + [x(1-\gamma) + (1-x)(1-\pi)]\phi}.$$

From this equation, we see that p_{∞} is identical for both schedules B′ and C′ of the second experiment; whereas, using Model 1, p_{∞} is greater for schedule C′ than for schedule B′. This relation, of course, is reflected in $Pr(H)$, and $Pr(F)$ and $Pr(A_1 \mid S_0)$. For Model 2

$$Pr^{B'}(H) = Pr^{C'}(H)$$
$$Pr^{B'}(F) = Pr^{C'}(F)$$
$$Pr^{B'}(A_1 \mid S_0) = Pr^{C'}(A_1 \mid S_0)$$

where $Pr^{B'}(H)$ denotes the asymptotic probability of a hit on schedule B′, etc. In contrast, for Model 1

$$Pr^{B'}(H) < Pr^{C'}(H)$$
$$Pr^{B'}(F) < Pr^{C'}(F)$$
$$Pr^{B'}(A_1 \mid S_0) < Pr^{C'}(A_1 \mid S_0).$$

The inequalities predicted by Model 1 for schedules B′ and C′ are borne out by the group averages presented in Table 3; it also is the case that the relations hold individually for all 14 subjects.

To further illustrate the differential predictions of Models 1 and 2 in the second experiment, we have plotted iso-bias curves in Figure 5 for the case where

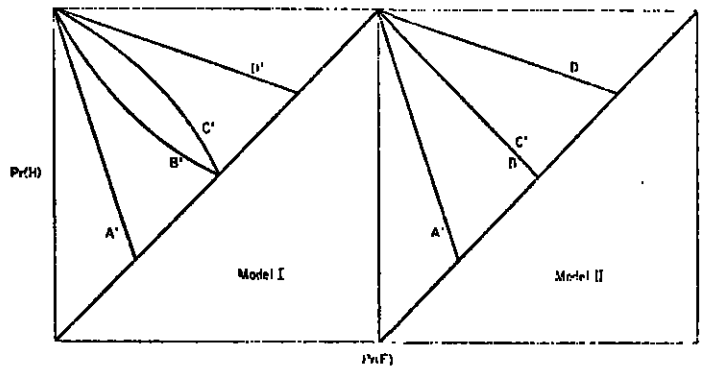

FIGURE 5. Iso-bias curves for Models 1 and 2.

$\phi = 1$. Note that the iso-bias curve for Model 2 is a straight line for all four presentation schedules, and also that the iso-bias curves for schedules B′ and C′ are identical. For Model 1 the iso-bias curves for schedules A′ and D′ are the same as for Model 2; however, under the assumptions of Model 1, schedules B′ and C′ generate different, non-linear curves.

Using Model 1, a distance function can be defined between corresponding points on the iso-bias curves for schedules B′ and C′. The maximum of this function can be obtained by taking its derivative with respect to σ and setting the result equal to zero. Carrying out these operations yields

$$\sigma = 2 - \sqrt{2} \approx 0{\cdot}59.$$

Therefore, under the assumptions of Model 1, the maximum difference between corresponding points on the iso-bias functions of schedules B′ and C′ will be observed when σ is approximately 0·59. One of the principal reasons for running the second experiment was to determine whether such a difference would be observed. Therefore, to maximize the likelihood of discovering an effect if it existed, we wanted to set the signal-to-noise level at a value corresponding to a σ of 0·59. Recall that pretraining involved only S_1 and S_2 trials, and they were presented with equal likelihood; hence $Pr(C) = \sigma + (1-\sigma)\frac{1}{2}$. Consequently to fix σ at approximately 0·59 required adjusting the noise level during pretraining to yield a correct-response probability of approximately $0{\cdot}79 \simeq 0{\cdot}59 + (0{\cdot}41)\frac{1}{2}$. The pretraining procedure was fairly successful, inasmuch as the estimate of σ during the actual experiment was 0·572.

In both of the experiments reported in this paper, response times were obtained on each trial. The response-time data are reasonably orderly and are clearly affected by the presentation schedule. For example, in the first experiment the time for an incorrect response was about 50 msec longer than for a correct response. Also, the response time for an incorrect response appeared to be independent of the stimulus presentation schedule, whereas the time for a correct response decreased somewhat as γ increased. An attractive feature of the present model is that it can be easily generalized to treat response-time data. The generalization is simply to assume that response time on a given trial is determined by the sensory state activated on the trial. More specifically, we assume that if sensory state s_i $(i = 0, 1, 2)$ occurs on trial n, then the response-time distribution for that trial has probability density $f_i(t)$ with mean t_i. On the basis of this assumption a number of predictions can be derived concerning the events on the current trial (and on preceding trials) as they influence response time. For example, in the first experiment the mean asymptotic response times conditional respectively on a correct and incorrect response are as follows:

$$E(T \mid C) = \frac{\sigma[\gamma t_1 + (1-\gamma)t_2] + (1-\sigma)[\gamma p_\infty + (1-\gamma)(1-p_\infty)]t_0}{\sigma + (1-\sigma)[\gamma p_\infty + (1-\gamma)(1-p_\infty)]}$$

$$E(T \mid \bar{C}) = t_0.$$

If $t_1 < t_2 < t_0$ then these conditional response-time measures are appropriately

ordered as γ increases. We are currently analysing an experiment specifically designed to evaluate the response-time assumption outlined above. The analyses are still incomplete, but it appears that if parameter estimates are made from the time distributions conditional on correct and incorrect responses, then reasonably accurate predictions can be made for distributions conditional on responses and signal events of the current trial (and the immediately preceding trial). This approach to response times needs more exploration but appears promising.

The experiments and model analyses considered in this paper have been confined to symmetric outcome structures involving no explicit payoffs. If we were to generalize the model to situations involving manipulation of monetary payoffs then it would be necessary to offer a more general theory of the decision process. Obviously there are outcome structures that will displace the subject off the linear ROC curve specified by eqn. (7). For example, consider the payoff matrix

$$\begin{array}{c} \\ S_1 \\ S_2 \end{array} \begin{array}{c} \begin{array}{cc} A_1 & A_2 \end{array} \\ \begin{bmatrix} -1 & +10 \\ +10 & -1 \end{bmatrix} \end{array}.$$

In this case the subject is heavily rewarded for incorrect detection responses and penalized for correct ones. Undoubtedly, over time the subject would generate a point $[Pr(F), Pr(H)]$ that fell in the lower right-hand sector of the ROC space; i.e., $Pr(F) > Pr(H)$. Such effects cannot be predicted merely by generalizing the assumptions governing p_n. No matter how p_n is permitted to vary, the model still requires that performance points fall on a linear curve with intercept σ. Of course, several modifications of the theory seem able to account for experimental manipulations that generate performance points off the ROC curve. One approach is to develop a more elaborate conceptualization of the decision process. For example, one can redefine the decision matrix as

$$\mathbf{D}_n = \begin{array}{c} \\ s_0 \\ s_1 \\ s_2 \end{array} \begin{array}{c} \begin{array}{cc} A_1 & A_2 \end{array} \\ \begin{bmatrix} p_n & 1-p_n \\ d_n^{(1)} & 1-d_n^{(1)} \\ 1-d_n^{(2)} & d_n^{(2)} \end{bmatrix} \end{array}.$$

For this process experimental manipulations of the outcome structure might affect not only p_n but also the values of $d_n^{(i)}$. Thus, depending on the postulated relation of $d_n^{(i)}$ to the payoff matrix it would be possible to generate virtually any ROC curve. When this type of modification is introduced one obtains a model that is very close in structure to those proposed for discrimination learning (Atkinson and Estes, 1963, p. 238; Bush, Luce and Rose, 1964). Another possible modification of the detection model would be to develop a more general formulation of the sensory process. Pursuing this line, one might assume that the subject's sensitivity level could vary within certain fixed limits as a function of the outcome structure and other variables. Both of these alternatives represent

potential lines of theoretical development for models of this type. They raise an important question: can changes in performance induced by manipulation of the outcome structure be explained by elaborating the theory of the bias process, or do they also necessitate postulating a more complex sensory mechanism?

References

Atkinson, R. C. (1963). A variable sensitivity theory of signal detection. *Psychol. Rev.* **70**, 91–106.

Atkinson, R. C., Bower, G. H. and Crothers, E. J. (1965). *An Introduction to Mathematical Learning Theory*. New York: Wiley.

Atkinson, R. C., Carterette, E. C. and Kinchla, R. A. (1962). Sequential phenomena in psychophysical judgements: a theoretical analysis. *Inst. Radio Engineers Trans. on Information Theory*, IT-8, S155–162.

Atkinson, R. C. and Estes, W. K. (1963). Stimulus sampling theory. In R. D. Luce, R. R. Bush and E. Galanter (Eds.), *Handbook of Mathematical Psychology*, Vol. II. New York: Wiley. Pp. 121–268.

Bush, R. R., Luce, R. D. and Rose, R. M. (1964). Learning models for psycho-physics. In R. C. Atkinson (Ed.), *Studies in Mathematical Psychology*. Stanford: Stanford University Press. Pp. 201–217.

Bush, R. R. and Mosteller, F. (1955). *Stochastic Models for Learning*. New York: Wiley.

Fechner, G. T. (1860). *Elemente der Psychophysik*. Leipzig: Breitkopf and Härtel.

Friedman, M. P. and Carterette, E. C. (1964). Detection of Markovian sequences of signals. *J. acoust. Soc. Amer.* **36**, 2334–2339.

Green, D. M. (1960). Psychoacoustics and detection theory. *J. acoust. Soc. Amer.* **32**, 1189–1203. Reprinted in R. D. Luce, R. R. Bush and E. Galanter (Eds.), *Readings in Mathematical Psychology*, Vol. I. New York: Wiley, 1963. Pp. 41–66.

Luce, R. D. (1963). A threshold theory for simple detection experiments. *Psychol. Rev.* **70**, 61–79.

Norman, D. A. (1964). Sensory thresholds, response biases, and the neural quantum theory. *J. math. Psychol.* **1**, 88–120.

Swets, J. A. (1961) Detection theory and psychophysics: a review. *Psychometrika*, **26**, 49–63.

Tanner, W. P., Jr. and Swets, J. A. (1954). A decision making theory of visual detection. *Psychol. Rev.* **61**, 401–409.