



Saccadic choice with asynchronous targets: evidence for independent randomisation

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Abstract

In the LATER model, randomness of saccadic latency arises through random variation in the rate of rise of the decision signal. But does it vary independently at different locations? If so, when pairs of targets are presented asynchronously, and the participant makes a saccade to the more salient one, the choice of target should be stochastic. Further, it should be possible to predict the probabilities at different asynchronies from the latency distributions for each target on its own. This study verifies the prediction in human subjects. In the real world, independent random variation of latency at different locations will give rise to randomness of choice of target. © 2001 Elsevier Science Ltd. All rights reserved.

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1. Introduction

Like other reaction times, saccadic latency — the time between presentation of a target and the start of a gaze-shifting saccade towards it — is both surprisingly long and surprisingly variable. Both these features are economically described by a model of saccadic and other kinds of latency in which reaction time is assumed to be dominated not by such low-level factors as receptor activation, conduction time and synaptic delay, but by the time needed for higher neural structures to detect the target and decide to respond to it. The LATER (Linear Approach to Threshold with Ergodic Rate) model specifically proposes a decision signal S that rises linearly in response to the stimulus at rate r from an initial level S_0 , until it reaches a criterion or threshold level S_T , at which point the response is finally triggered (Fig. 1). If r varies in a Gaussian manner from trial to trial, we can then immediately explain an apparently universal feature of reaction-time distributions, that the reciprocal of latency is normally distributed. This *recinormal* distribution can be demonstrated most conveniently by plotting cumulative histograms of reac-

tion times using a reciprocal abscissa and probit ordinate (a *reciprobit* plot: Fig. 1), which will then yield a straight line (Carpenter, 1981).

Apart from providing a parsimonious description of reaction time distributions, requiring no more than two parameters, the LATER model has an obvious functional interpretation, if we regard S as an internal representation of the perceived likelihood of the target being present. S_0 then corresponds with the prior likelihood, or expectation of the target before it actually appears, and S_T with a level at which the stimulus is regarded as so probable as to demand the response, and equivalent to a significance level in statistics. More precisely, we expect these likelihoods to be represented on a logarithmic scale: because a given piece of evidence will then cause a constant increment in S (Edwards, 1972; Carpenter & Williams, 1995), linear rise corresponds to the steady rate of arrival of sensory information providing evidence for the existence of the stimulus.

The LATER model thus offers the possibility of explaining a puzzling feature of response times, in a way that is both economical and makes functional sense. Over the last 5 years some effort has gone into testing whether its quantitative predictions are actually borne out by actual experiments. More particularly: (a)

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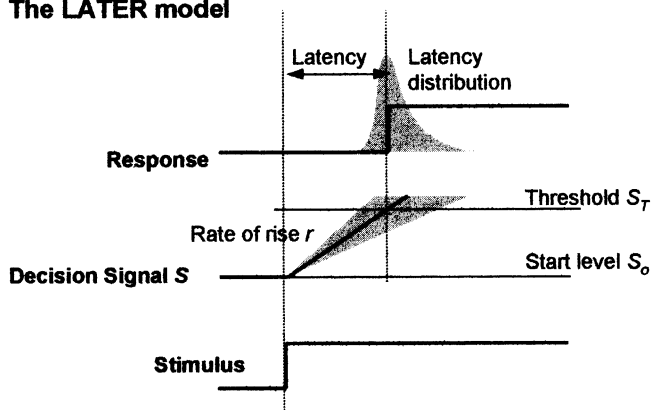
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whether actual reaction-time distributions are well described as recinormal; (b) whether changing prior probabilities of targets has the expected quantitative effects on reaction-time distributions; (c) whether instructions encouraging a user to change the criterion level S_T induce the predicted changes in distributions; and (d) whether altering the rate of provision of information has the expected effect on the rate of rise.

So far, LATER seems to have stood up to these challenges. A review of the corpus of historical data has demonstrated the accuracy with which nearly all latencies, whether evoked by vision, hearing or touch, whether the responses are saccadic or manual, and whether in humans or other species, are described by LATER (Carpenter, in preparation).

One important exception is that in larger data sets it is clear that a very small proportion (typically around

The LATER model



A reciprobbit plot

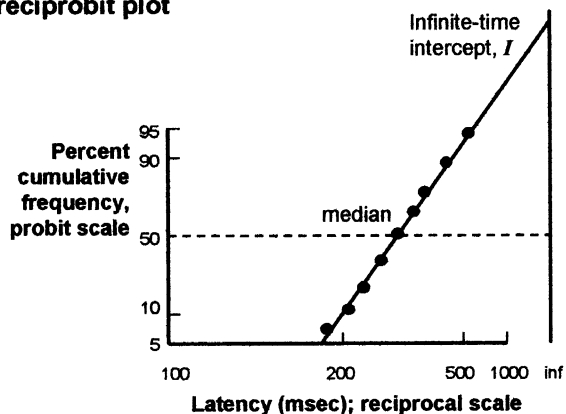


Fig. 1. Saccadic latency and the LATER model. Above, a stimulus (bottom) causes a decision signal S to rise at a constant rate r until it reaches a threshold level S_T , whereupon it triggers the response. Because r varies in a gaussian manner from trial to trial, the distribution of latencies has a characteristic form: its reciprocal is normally distributed (a *recinormal* distribution). Below, this property can be tested by plotting latency histograms cumulatively on a cumulative, probit, ordinate with a reciprocal abscissa: in this *reciprobbit plot*, *recinormal* distributions become straight lines.

3–4%) of the fastest responses occur significantly more often than predicted by LATER, generally lying on a second, shallower line than the main *recinormal* distribution, that extrapolates to 50% at infinite time. Because they form so small a proportion of the main distribution, it is difficult to be certain of the mathematical form of their distribution, which might for instance be Poisson rather than *recinormal*, but is certainly distinct from classical express saccades, whose distribution is typically bimodal (Fischer & Boch, 1983; Fischer & Ramsperger, 1984). In reciprobbit plots these fast responses have a visual prominence that greatly exaggerates their significance, since in truth they represent only the extreme tail of the distribution, and in studies such as the present one they can probably safely be neglected. Experiments in which prior probability is manipulated (Carpenter & Williams, 1995) strongly support the identification of S_0 with prior probability, and provide evidence that the decision signal is indeed represented on a logarithmic scale. Similarly, a recent study (Reddi & Carpenter, 2000) has shown that instructions to subjects to concentrate either on speed or alternatively on accuracy cause changes, as predicted, in the criterion level, S_T . Results currently in preparation have in addition shown that systematically altering the amount of information available to a subject alters latency distributions in the way expected from LATER if it is indeed the mean rate of rise r that is affected.

Further support for LATER has come from single-cell recording in animals carrying out saccadic tasks. Saccadic movement-related cells in monkey frontal cortex demonstrate patterns of firing behaviour that are closely similar to what would be expected from LATER (Hanes & Schall, 1996; Schall & Thompson, 1999). In response to a visual stimulus, the activity in such cells rises roughly linearly after presentation of a stimulus, at a rate that varies randomly from trial to trial: initiation of a saccade occurs in association with a level of firing that is constant across trials. These features are the essence of LATER. More recent experiments have shown that the mean rate of rise alters in the expected way when the amount of information provided is altered (Bichot & Schall, 1999; Kim & Shadlen, 1999; Gold & Shadlen, 2000).

However, there is one aspect of LATER with very important functional implications that has not yet been mentioned. In the real world, faced with not just one LED, but a host of possible stimuli competing for the privilege of being a saccadic target, what LATER implies is a sort of race between decision signals corresponding to the different possibilities, the one reaching threshold first being the one that captures the saccade. Variation of rate of rise will then translate into variation of *choice* of response: whatever mechanism generates randomness of timing would then also generate randomness of choice, for even under identical condi-

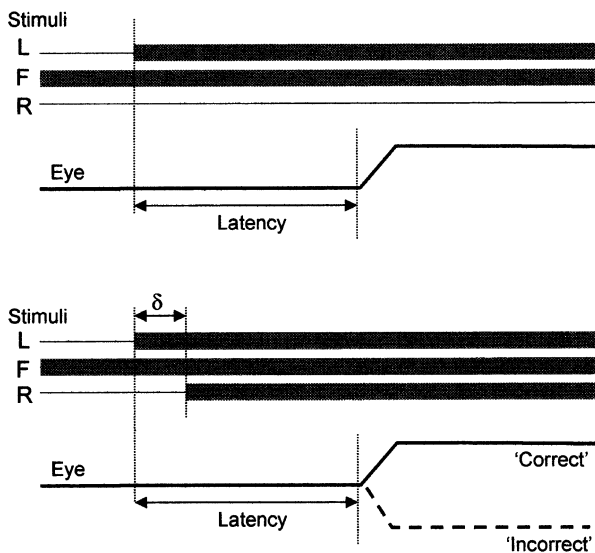


Fig. 2. Stimulus paradigms. Above, control trials: only a single target L or R appears, in addition to the fixation light F that is always on. Below, test trials: targets appear on both left and right, their onsets separated by an interval δ , and the subject may make a saccade to the first or to the second target. In practice, appearances to left or right are equally probable, though for simplicity only half the possible cases are shown here.

tions, given the same set of alternatives, the system would not always make the same selection each time. The randomiser might, in other words, underlie the spontaneous unpredictability so characteristic of decision behaviour, that no doubt contributes to our sense of having free will (Carpenter, 1999).

However, this argument is only valid if the randomisation is specific to individual decision units, and not, for instance, applied commonly to all of them. In the latter case, on an occasion when one signal happens to rise particularly fast, all of them will, so that the outcome will be no different. It thus becomes a matter of some interest to determine whether the randomiser is indeed specific to each decision unit, or whether it is common to all. Unfortunately, this is a question that is difficult to address neurophysiologically, for it demands simultaneous recording from at least two cells, over some period of time. But it can quite easily be addressed behaviourally. All we have to do is present not one but two alternative targets, as near identical as possible, and instruct a subject to look at whichever of them seems to appear first. We present them asynchronously, one preceding the other by a short period δ . It is clear that if δ is sufficiently large the subject's response will always be to the first. But what will happen as δ is reduced? If r varies independently for each unit, then there will be occasions when the second decision signal happens to reach threshold before the first, and as δ is reduced this outcome will become more

and more likely: when $\delta = 0$, both will be equally attractive. But if variation in reaction time is common to all decision units, then the subject will always respond to the first-appearing target whatever the value of δ : as explained above, if one rate of rise happens to particularly fast, so will the other, but the winner will be unchanged. Which actually happens?

2. Methods

2.1. Participants, stimuli and protocols

The six participants took part with informed consent, and the general procedures used had received local ethical committee approval. All were 20–22 years of age, two male and four female: they could all focus on the targets, with optical correction if necessary, and none suffered from apparent visual defects apart from one who was amblyopic and performed the task monocularly.

The stimuli consisted of a horizontal row of three red LEDs, 90 cm from the participant, each $2 \text{ mm} \times 2 \text{ mm}$ in size and separated by 4° , mounted directly behind a translucent screen uniformly illuminated with light of matching colour at an intensity of 7 cd/m^2 , the target lights being 2.8 log units above increment threshold intensity, and in dark surroundings.

The central light remained on as a fixation point throughout the experimental runs. Each trial began with a brief auditory tone and a random wait of 700–1700 ms. In control trials only one target then appeared, randomly on the left or right (Fig. 2), and the participant was instructed to look at it as soon as it appeared. In test trials, both targets appeared, but one (chosen randomly) preceded the other by an interval δ , which had a value of 20, 40, 60 or 80 ms. Participants were asked to look at whichever target caught their attention. Target lights were normally extinguished 100 ms after detection of a saccade response, with a new trial beginning automatically 150 ms after that. A control run consisted of 200 trials. Test runs were made with a single value of δ or with mixed values of δ chosen at random with equal probability. Random numbers were derived from a congruence sequence (Abramowitz & Stegun, 1965) with a period greater than 65000.

Participants normally performed 100 practice trials to get accustomed to the task before a control, single δ or mixed δ experiment, and were instructed to look, as quickly as possible, at whichever target caught their attention. A minimum of 200 control trials and 200 trials of each value of δ were recorded and analysed for each participant.

2.2. Recording and analysing eye movements

Horizontal eye movements were recorded with a head mounted infra-red oculometer (Carpenter, 1988), linear within 1% over $\pm 10^\circ$ and with a flat frequency response to 500 Hz. Participants used either a chin rest or a bite-bar mounted on a vertical support. Eye movements were stored and analysed with a computer system running SPIC (Carpenter, 1994) which as well as controlling the presentation of stimuli, monitors eye position at 10 kHz and detects in real time the occurrence of saccades (using velocity and position criteria), whose times and directions are stored in 10 ms bins along with the raw records. At the end of a run, records were thoroughly reviewed to check that saccades had been correctly identified and to discard those responses due to blinks, lack of attention or other irregularities; in addition, all saccades with latencies outside the range 50–700 ms were automatically rejected.

For all participants, data from saccades in both directions were initially separated and compared and any discrepancies noted. Test experiments were not carried out until there was no appreciable difference between latency distributions of saccades to the right and those to the left. Care was taken during this experiment to ensure that both target lights were similar in every way possible, since the contrast strongly affects saccadic latency. Participant A had a significant problem eliminating left/right differences in latency and underwent considerable training. The latency distributions were analysed using SPIC, and a line of best fit was plotted by computer analysis by minimisation of the Kolmogorov–Smirnov statistic (Kolmogorov, 1941); all were compatible with the recinormal distribution at $P = 0.05$. In test runs, the proportion of ‘successes’, i.e. saccades in the direction of the first stimulus to appear, was calculated.

2.3. Simulations

SPIC also ran the computer simulations, comprising pairs of competing units whose rates of rise to threshold varied in a Gaussian fashion between trials, with the LATER parameters, intercept and slope, adjusted to correspond with control data. A parameter could be included in the model to represent lateral inhibition between the two units (Section 4). Simulations of 2048 trials were run with values of δ corresponding to test runs, and measurements of latency distributions and proportions of successes calculated in the usual way. For the purpose of calculating the significance of differences in proportions of successes between participants and simulations, these were treated as binomial processes.

3. Results

3.1. Probabilities

Fig. 3 shows how the probability of ‘success’ in any trial (i.e. of responding to the target first presented) varies with the interval δ between the two targets. It is clear that for these four subjects this probability increases monotonically with δ , and indeed on a probit ordinate the relationship is almost linear. It is also evident that there is a certain amount of variability between subjects in respect of the steepness of this function, from 80–95% at $\delta = 60$ ms. For the two other subjects this variation was more extreme: they are considered separately, below. While LATER predicts that the probability will increase with increasing δ , and that the relation is nearly linear when a probit scale is used as ordinate, the relationship is not analytically simple; the easiest way to estimate expected probabilities is to run Monte-Carlo simulations of the LATER process, but this demands information about the actual latency distributions in each case.

3.2. Latency distributions

Latency distributions were calculated for all subjects under control conditions (runs with just one target) and experimental conditions: three examples are shown as reciprob plots in Fig. 4. Best-fit LATER parameters were estimated for each control distribution, using the Kolmogorov–Smirnov statistic; in no case did the observed distribution deviate significantly from the expected distribution (Kolmogorov–Smirnov, $P = 0.05$). Distributions for experimental runs did however differ significantly from control runs for every subject (Kolmogorov–Smirnov, $P = 0.05$), with medians that for each participant exceeded those of the controls, by some 10–30 ms. However, those for different values of δ did not differ significantly amongst themselves, whether in mixed or single- δ trials (Fig. 5, top). For this reason, we pooled together latencies for all values of δ in mixed runs in subsequent analysis, both of experimental data and simulations. It is these pooled distributions that are shown with open circles in Fig. 4. In addition to the main recinormal distribution, subjects generally also showed evidence for the faster group of saccades mentioned in Section 1. As can be seen in Figs. 4 and 5, they seemed to show the usual property of lying along a second line of shallower slope going through the 50% point at infinite time. However, the number of these faster responses was too small to be able to perform statistical analysis of significance. Since they never formed more than 4% of the total population, they were excluded from subsequent analysis; estimates of the parameters of the main recinormal distribution were not significantly affected by their ex-

clusion. Similar analysis of distributions resulting from simulations that explicitly included a second component designed to correspond with the faster population confirmed that their contribution to estimates of the main recinormal distribution was insignificant.

3.3. Lateral inhibition

The latency distributions of Fig. 4 show a feature that is immediately puzzling. If we run a race between two stochastic processes, then it is clear that whichever process happens to be faster on any particular occasion determines the overall outcome. It is thus evident that the latency distribution for two targets ought to be shifted to the left relative to that for one alone. We have seen that in fact it is shifted to the right, with test median latencies being on average longer than controls by anything from 15 to 40 ms.

One possible explanation is to invoke lateral inhibition, a ubiquitous phenomenon at both sensory and motor levels of the brain (Section 4). One can well

imagine that decision units may not only race against one another, but also compete more aggressively, with activity in one tending to reduce the activity of others. To see whether mutual inhibition between decision units might account for the slowing of responses in test trials, Monte-Carlo simulations were performed in which the activity in each of the units, multiplied by an attenuating factor p , was subtracted from that of the other before being compared to threshold. This inhibition was of the feed-forward kind: more specifically,

$$u_1 = S_0 + r_1 t; \quad u_2 = S_0 + r_2 t;$$

$$v_1 = u_1 - p u_2; \quad v_2 = u_2 - p u_1;$$

where u_i is the intrinsic activity of the i th unit, t is time and v_i is the corresponding value after lateral inhibition, which is then compared with S_T .

Responses under these conditions were indeed slower on average than with single targets, the distributions being shifted to the right by an amount that depended on the value of p (Fig. 5, bottom). By choosing simulation parameters corresponding to experimental control

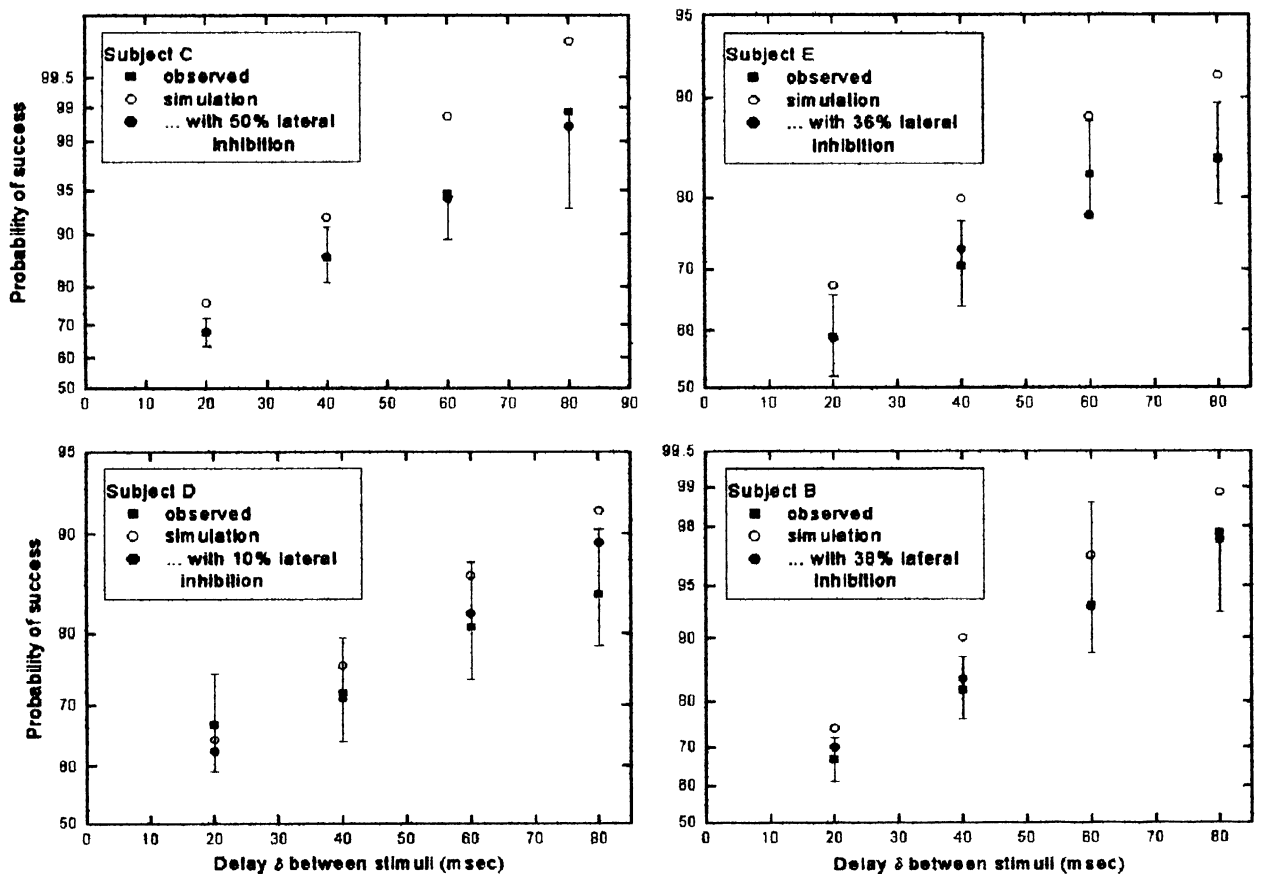


Fig. 3. Observed and simulated probability of success as a function of precedence interval, δ . The proportion of trials in which the participant made a saccade in the direction of the first stimulus to appear is plotted on a probability scale as a function of the time δ between the first and second target. Error bars represent twice the value of the standard deviation of the successes, as derived from a binomial distribution. The parameters for the simulations were calculated from control runs for the same subjects, and the percentage lateral inhibition was derived in each case from best-fit simulations of the overall latency distribution for all test trials taken together, as described in the text. ($n = 200$ for each experimental point, 2048 for simulations).

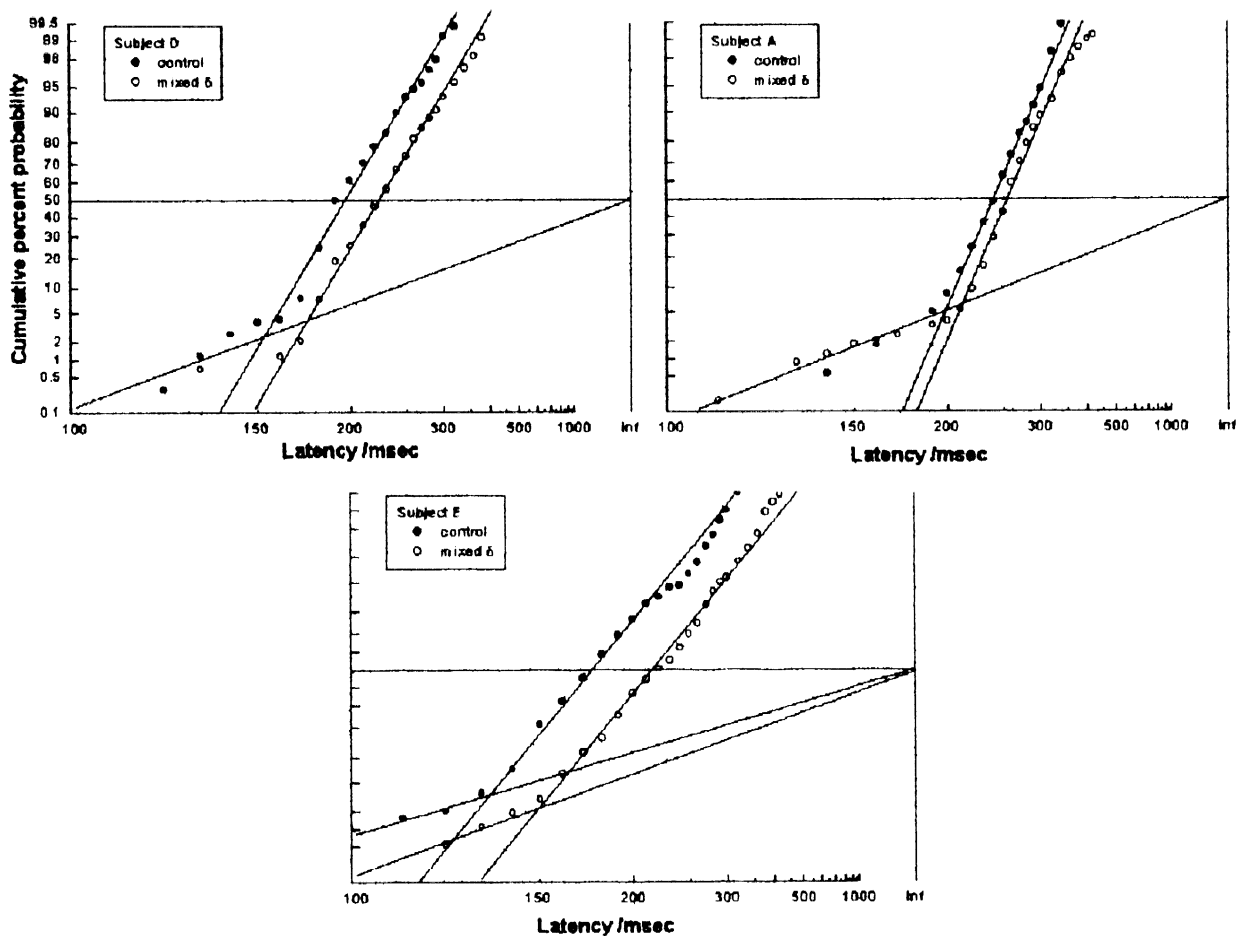


Fig. 4. Reciprobity plots (Fig. 1) showing saccadic latency distributions for both the control trials (filled circles) and for all test trials taken together (open circles), for three subjects. The lines represent best fits of the LATER model, including the lateral inhibition parameter p in the case of the test trials, minimising the Kolmogorov–Smirnov statistic; for this purpose the population of faster saccades, that tends to lie along the line of shallower slope, is ignored. ($n = 200$ for control trials, 800 for test).

runs for particular subjects, it was then possible to adjust p in each case to produce the best fit to the observed data: such simulations are shown as the right-hand lines in each pair in Fig. 4. As can be seen, the resultant distributions can then be accounted for quite satisfactorily. Furthermore, one can then proceed to use these values of p , and the other parameters for each subject, to run simulations from which the expected proportion of successes for each value of δ can be estimated. These findings are shown in Fig. 3, where the open circles show the result of simulations without incorporating lateral inhibition, and the filled circles show the result of using the values of p calculated from the latency distributions. As can be seen, in every case the incorporation of lateral inhibition improves the correspondence between the predictions and the observations. Indeed every single prediction for these subjects lies within the 5% binomial significance limits, even though the values of p were derived not from these data, but from the distributional data. For two subjects, however, the simulations did not predict perfor-

mance so well, and the possible reasons for this are discussed below.

4. Discussion

The main conclusion of this study is that the rate of rise of activity in different decision units varies independently at different sites, rather than being common to all. Consequently, under conditions of competition as experienced when viewing the kinds of scenes that actually occur in the real world, the random variation in timing is translated into randomness of choice. In addition, we provide behavioural evidence for a mechanism of lateral inhibition by which the activity of one unit tends to reduce that of another. Simulations with rather few parameters seem to provide quite adequate descriptions not only of latency behaviour but also of choice. The ability to predict choice behaviour in response to two stimuli is satisfying in that it superficially has little to do with saccadic latency distributions, and

could be regarded as further confirmation of the general applicability of the LATER model.

Lateral inhibition is a common feature of sensory systems at all levels of the CNS, and it has often been argued that this is a desirable feature that tends to sharpen the discrimination between competing perceptions. By analogy, one would equally expect to find it on the motor side of the brain, where it would serve to enhance intended patterns of response and discourage interference from alternative, less strongly evoked outputs, as perhaps in the familiar example of recurrent

inhibition from Renshaw cells on to spinal motor neurons. Such concepts have recently been discussed in quantitative detail by Wilson (1999). Lateral inhibition was evoked by Hanes and Carpenter (1999) to explain certain features of behaviour observed in human saccadic countermanding tasks. It has also been demonstrated neurophysiologically at two important sites concerned with initiation of saccades and their motor preparation. In the superior colliculus, an extensive network of inhibitory processes appears to have an important role in target selection (Munoz & Istvan,

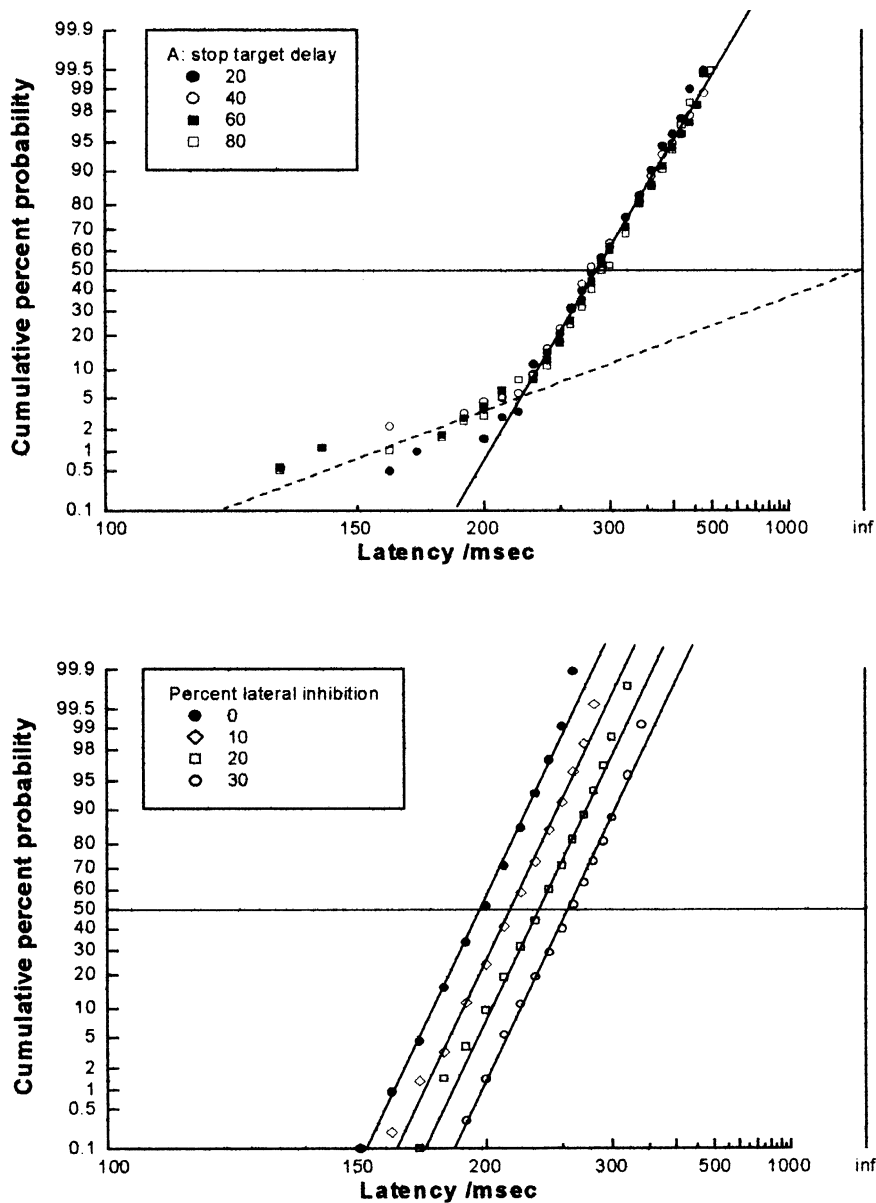


Fig. 5. Above, latency distributions for test trials with the different values of δ shown, for one subject: the distributions are statistically indistinguishable $p = 0.05$, Kolmogorov-Smirnov). Below, effect of lateral inhibition on simulated competition between a pair of LATER units. Reciprobbit plots are shown for simulations ($n = 2048$) with mixed values of the precedence interval δ as in the experiments, for different values of the lateral inhibition parameter p . In general, the effect of increasing p is to increase median latency, the distributions shifting in a roughly parallel fashion to the right. The amount of lateral inhibition used in subsequent simulations was interpolated from the relevant graph using the median latency values from combined test trials such as in Fig. 4.

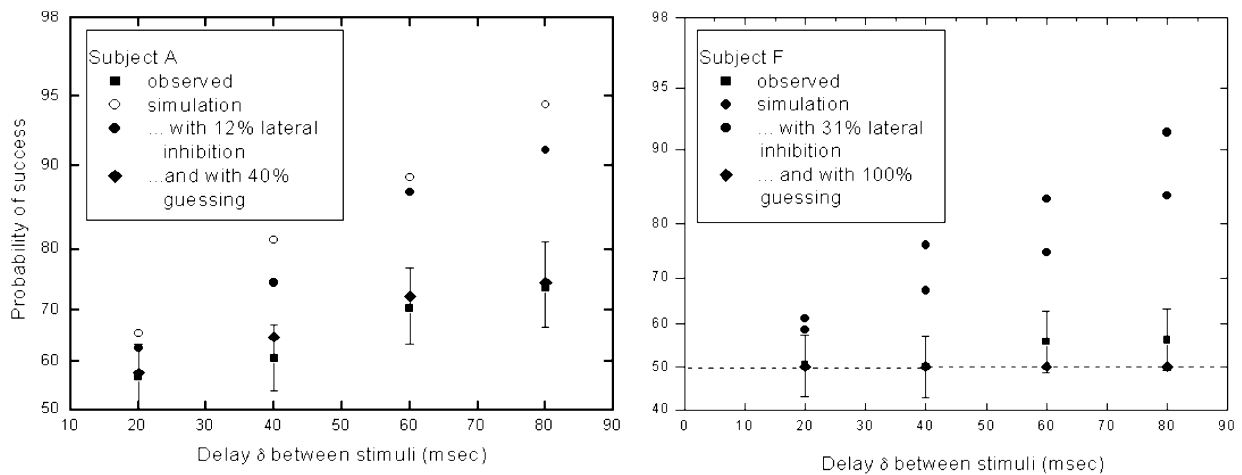


Fig. 6. Probability plots as in Fig. 3, for two subjects who performed significantly worse than predicted by the LATER model. The rhombi show expected performance if, in the stated percentage of trials, the subjects are simply guessing. ($n = 200$ for each experimental point, 2048 for simulations).

1998), and in the frontal eye fields of monkeys, similar effects of lateral inhibition can be demonstrated directly using distractor tasks (Schall & Hanes, 1993; Schall, Hanes, Thompson, & King, 1995).

4.1. Interference from idiosyncratic 'higher' factors

The asynchronous target pair protocol is at first quite disorientating for the participant. Instructions to look at the target that appeared *first* led to very long delays while the participant consciously pondered which had indeed appeared first. In an attempt to eliminate this interference of consciousness, participants were instead instructed to look, as quickly as possible, at whichever target caught their attention. Some participants reported that they found the task difficult whereas others reported that they were able to 'switch off' and think about other things whilst their eyes were subconsciously drawn to one of the targets. As a result, subjects find themselves able to make subconscious discriminations that they are unable to do consciously. As can be seen in Fig. 3, for most subjects some 30–40 ms is sufficient to achieve 75% correct responses, comparable with what has recently been described for monkeys in a similar task (Schiller & Chou, 2000). On the other hand, preliminary experiments showed that an interval of about twice that was required for conscious performance at an equivalent level in the same apparatus, a value similar to previously published findings for similar tasks (Sternberg & Knoll, 1973).

The two subjects who found the task difficult did indeed perform worse at it (Fig. 6); in fact one of them was completely unable to perform the discrimination at all, his choice of target being insignificantly different from chance level for all values of δ used. The other was able to do the task, but at a reduced level of

performance, his actual probabilities being systematically smaller than the predictions. It is possible that in such cases there is interference in some trials between the conscious and unconscious processes; subjects report that at first it is difficult not to think about the targets and one's responses, and to speculate on which will come first in the next trial. After training, most subjects get over that stage and settle down into the kind of subconscious performance described earlier, but it is possible that a subject like F never achieves this state, and that one like A manages it for some trials but cannot sustain this effort for all. To make this hypothesis more quantitative, suppose that a subject's subconscious mechanism operates in a proportion q of trials, while conscious guessing (50% success) operates in the other $(1 - q)$, and that for a particular value of δ , the underlying subconscious performance is such that the response is correct on a proportion p of trials. Then the overall proportion of success will be given by $pq + 0.5(1 - q)$. If for A we take q to be 0.6, the resultant predictions are statistically indistinguishable from his actual performance (Fig. 6), and of course F's performance can be fitted with $q = 0$. It would be interesting to find more subjects who have difficulty of this kind, and see whether the hypothesis works for them as well.

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