



Express saccades: is bimodality a result of the order of stimulus presentation?

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Abstract

Subjects undertook a saccadic gap task, in which the fixation target is extinguished for a period before the appearance of the peripheral stimulus. The majority showed a population of short-latency express saccades in addition to the main, slower, distribution. However, closer analysis showed that nearly all of this bimodality was due to the *order* in which trials were performed: the faster responses came almost entirely from trials in which the target was on the opposite side from the preceding trial, slower ones when it was on the same side. Further experiments using a novel two-gap task demonstrated that this inter-trial effect is due to the return eye movement of one trial conditioning the first saccade of the next. Consequently, in a two-gap task the latency of the second saccade falls into the faster category if it is in the *same* direction as the immediately preceding one: this may be the result of the oculomotor system predicting target direction, saccades in the expected direction having a shorter latency. It seems therefore that the bimodality is not primarily the result of some kind of randomising process within the oculomotor system: rather, it is a consequence of the way in which saccadic experiments are normally conducted. © 2001 Elsevier Science Ltd. All rights reserved.

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1. Introduction

When a subject looks at a target that suddenly jumps from one position to another, the eye follows with a saccade whose latency varies randomly from trial to trial, usually with a unimodal distribution around some 150–200 ms (Carpenter, 1981). In a gap task, the target is extinguished for a short period before it moves. For many subjects the distribution then becomes bimodal, a sub-population called express saccades forming an additional peak with a much shorter latency (Fig. 1, top right) (Fischer & Boch, 1983; Fischer & Ramsperger, 1984). Express saccades have been the object of intense research and speculation since their discovery by Fischer: interest has focused particularly on the anatomical pathways that might be responsible for the faster population of responses, and on possible underlying neuronal mechanisms. It is difficult to imagine a plausible stochastic process within the brain that could give rise

to a bimodal distribution of latency in such cases. For instance, it is sometimes assumed that express saccades are due to a faster pathway in parallel with the main one (Schiller, Sandell, & Maunsell 1987), the latency of each being subject to random variation; but if this were so, then the faster responses should simply pre-empt the slower ones, whereas in practice one typically sees distributions in which there are substantial numbers of slower saccades as well as the express population. What seems to be required is not a race but a random dichotomiser that first decides whether a trial is express or not, followed by a separate stochastic process generating the different latencies, and a plausible neural network model has been published (Fischer, Geleck, & Huber, 1995) that can be programmed to behave in this way. However, before investing much effort in such models it is perhaps important to see whether bimodality might be due, not to a hypothetical stochastic neural mechanism, but rather to some feature of the sequence of stimuli that have been presented. Sequence effects in reaction time studies have been noted quite often, as examples of how the time to make a response depends

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in part on the prior state: originally for manual reaction times (Laming, 1968; Remington, 1969; Falmagne, Cohen, & Dwivedi, 1975; reviewed by Luce, 1986); subsequently for smooth anticipatory responses and in passing for saccades by Kowler, Martins, and Pavel (1984) and Kowler (1989), by Jüttner and Wolf (1994) and — in a different sense — by Paré and Munoz (1996). The importance of prior state is also suggested by the observation that cortical potentials recorded before target onset may correlate with the subsequent reaction time (Everling, Krappmann, Spantekow, & Flohr, 1996, 1997). The emphasis, in other words, is not on random dichotomy of response to constant stimuli, but rather on a more long-term view of what constitutes a stimulus, that takes into account the past history. Kowler (1990) p. 54 concludes: ‘Short latency responses aren’t special reflexes; they are responses to stimuli which happen to have been correctly anticipated by the subject... Development of saccadic models based on ideas about preparatory processes, and investigation of the different internal operations that constitute ‘preparation’, may in the long run lead to more satisfactory models of saccades than speculation about special saccadic mechanisms.’ It seemed worthwhile therefore to see whether such sequence-dependant anticipations might not merely give rise to small perturba-

tions of latency, but could actually be the origin of the bimodality constituting the phenomenon of express saccades. Could expressness be a kind of artefact, arising from a particular type of experimental set-up?

2. Methods

Saccadic stimuli consisted of yellow LEDs subtending 14×23 arcmin viewed against a yellow background of 9 cd m^{-2} , normally (except where noted otherwise) at a contrast of 100%. A trial began with a warning tone, followed by a random wait of 0.5–1.0 s during which the central fixation LED was illuminated. In a *single-gap* task, the LED was extinguished at the end of this period for 130 ms, and then jumped 3° randomly to the left or right (Fig. 1, left). In a *two-gap* task a trial began as before, but after jumping 1.5° to left or right the target remained for 1 s at its new position before being extinguished for 130 ms and then making a second jump of 4.5° randomly to left or right of its new position (Fig. 3, left). Unpredictability of direction or of waiting times was achieved by a pseudo-random number generator using the congruence method, with a period of over 64 000. In each case, subjects were instructed to follow the apparent movement of the

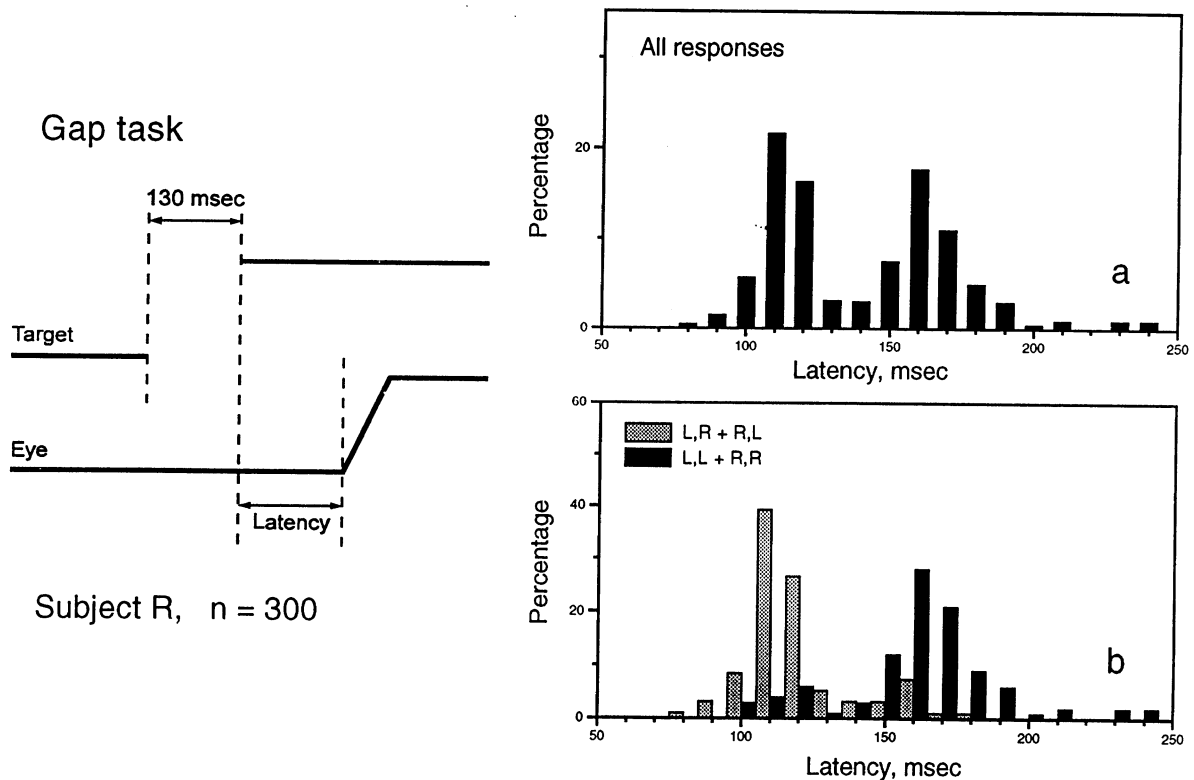


Fig. 1. Distribution of latencies in a saccadic gap task, with express saccades. Latency distributions are shown at right for one subject in a single run of 300 trials, in the form of a histogram with 10-ms bins; (a) for all responses pooled; (b) separated according to whether the trial was preceded by one in the same direction (L,L + R,R) or in the opposite direction (L,R + R,L).

stimulus without being concerned about the speed of their responses, resulting in a high degree of accuracy in the response: only seven of the more than 5000 saccades making up the study were incorrect in direction.

Eye-movements were recorded by infra-red scleral oculometry (Carpenter, 1988), and saccadic latencies measured automatically by an on-line computer system (Carpenter, 1994) and assigned to 10-ms bins; trials with abnormal responses (unsteady fixation, false or premature movements, blinks) were discarded. Seven subjects participated with informed consent (the general procedures having approval from the local Ethical Committee); four were male and three female, and none with known visual defects. Whole runs consisted entirely either of one-gap trials or of two-gap, divided into blocks of 100, with optional rests in between, and preceded by a training period, normally of 200 trials of exactly the same kind as in the subsequent experimental runs, in which the subjects became accustomed to the procedure.

3. Results

Fig. 1a shows the distribution of saccadic latencies for one subject in an experiment consisting of 300 single gap trials. The histogram is obviously bimodal, with a typical express population of saccades peaking at around 110 ms, and a slower population at around 160 ms. But looking back at the original sequence of stimuli, and creating separate histograms for those trials where a stimulus to the right (R) was preceded by a trial to the left (this can be notated as L,R) or vice-versa (R,L), as opposed to those in which the preceding trial was in the same direction (L,L and R,R), it becomes apparent that nearly all the early peak is due to (L,R + R,L) and nearly all the later peak to (L,L + R,R) (Fig. 1b). The bimodality, in other words, is essentially the result of adding together statistics from two kinds of trials that are not in fact equivalent. Fig. 2 shows comparable separations in three other subjects, with some variation of conditions. For each of these four subjects, the (L,L + R,R) and (R,L + L,R) distributions were compared with each other, and with the overall distribution, using the Kolmogorov–Smirnov two-sample test (Siegel & Castellan, 1988): in all cases the distributions were significantly different at the 5% significance level, and in fact for all but one at the 1% level. This effect is qualitatively similar to what was noted in passing for a single subject by Jüttner and Wolf (1994). Though there is individual variation in the degree of separation, it is clear that in each case any bimodality is to a very large extent due to the conditioning effect of one trial upon the next. But it is also clear that the bimodality is not *entirely* due to this effect, since the separated histograms still show some

residual traces of being composed of two separate populations.

It is not difficult to think of processes that might favour target alternation in this way. A second saccade to the same position as a preceding one might, for instance, be delayed because the visual mechanisms in that particular part of the visual field have not yet recovered from the effects of the previous stimulus, perhaps because of an after-image. Preliminary experiments suggested that this was not the case: targets of low contrast, seen against bright backgrounds, still exhibit the effect, as do targets of extremely brief duration; and changing the eccentricity of the target from trial to trial has no noticeable influence either.

In any case, there is a complication in these conventional experiments that makes interpretation of this kind less applicable than might at first be thought, for one must consider not just the movement *to* the peripheral target whose latency is measured, but also the *return* movement at the end that refixates the original central target. Thus a pair of trials designated as (R,L) actually consists of four movements in all: (R,L,L,R). If the return movements are taken into account, then the conclusion is reversed: movements made in the same direction as a preceding one are shorter, in the opposite direction, longer.

To test this, a novel experimental paradigm was devised, the *two-gap* task (Fig. 3), designed to test the effect of direction sequence more explicitly. Each trial consisted of two target movements: the central target was extinguished for 130 ms, then jumped randomly to right or left; then, after a delay of a second, it was again extinguished for 130 ms and moved randomly to right or left of its new position. Each trial was then of the form (RL), (RR), (LL) or (LR), with the second component succeeding the first immediately without an intervening refixation. Under these conditions, the dichotomy is again evident (Fig. 3), but reversed: latencies are now shorter for the second saccade in the (RR + LL) conditions compared to (RL + LR). For each of these subjects, the (LL + RR) and (RL + LR) distributions were examined with the Kolmogorov–Smirnov two-sample test and found to be significantly different ($P < 0.01$). This effectively rules out the after-image hypothesis: saccades to a retinal locus previously a target are shorter, not longer.

One might speculate that the dichotomy arises in the single-gap trials because in the RL and LR cases the eye is returning to the central fixation spot, and that there is some kind of long-lasting inhibition associated with locations in space (as opposed to retinal locations) that have been previously fixated, perhaps equivalent to the well-known ‘inhibition of return’ (IOR) that can also be demonstrated merely by prior exogenous shifts of visual attention (Vaughan, 1984; Abrams & Dobkin, 1994; Taylor & Klein, 1998; Dorris, Taylor, Munoz, &

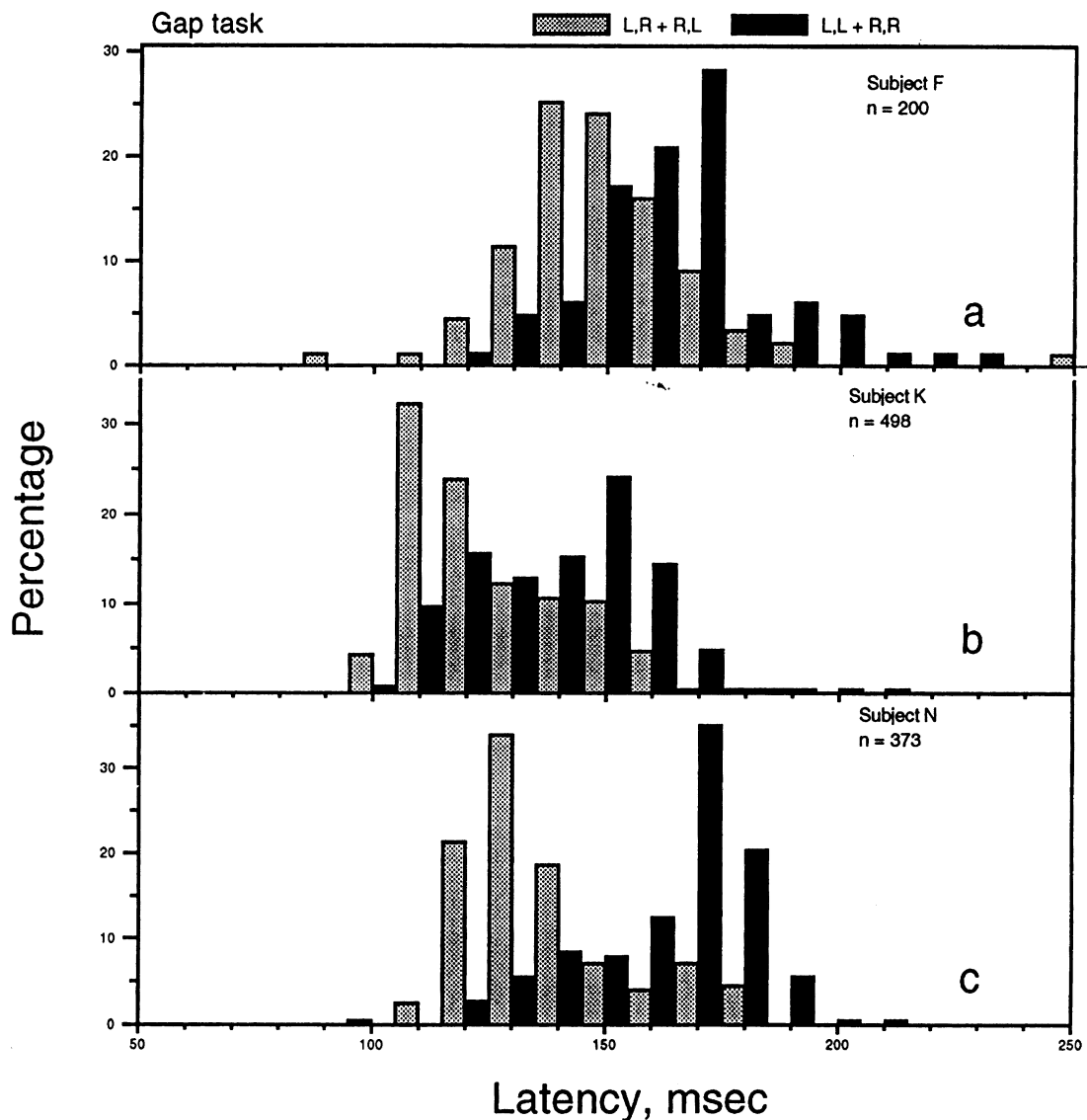


Fig. 2. Latency distributions separated as in Fig. 1, for three other subjects. For subject N the contrast was 30%, and the gap was 150 ms; for subject F the gap was 160 ms, and for K it was 120 ms.

Klein, 1999; Hooge & Frens, 2000). However, the results of making a simple modification of the task — altering the size of the second saccade — suggests that IOR is unlikely to be the explanation. It makes no noticeable difference whether the second target-jump is the same size as the first, or whether, as was the case in all the two-gap trials (Fig. 3), it is arranged to be a different size. This implies that the phenomenon is essentially to do with motor direction and that the end-point of the saccade is not particularly significant. However, recent work on IOR (Klein & Taylor, 1994; Reuter-Lorenz & Rosenquist, 1996; Reuter-Lorenz, Jha, & Rosenquist, 1996; Dorris et al., 1999; Hooge & Frens, 2000) while suggesting that it too may have a motor as well as a sensory component, implies that the end-point of the saccade is not necessarily critical in

determining whether IOR is observed or not. If so, the present phenomenon may indeed be closely related to IOR, though manifested in a more dramatic form.

Of the seven subjects examined, two (J and M) showed neither bimodality in their gap response nor divergence of the distributions when split into the two sub-populations: correspondingly, in the two-gap task there was a complete lack of distinction between the two cases (Fig. 4) (all populations being indistinguishable with the Kolmogorov–Smirnov test at the 5% level, all but one at 10%). This provides further evidence that the phenomenon demonstrated in the two-gap task is in fact the origin of the bimodality in ordinary gap tasks, since it is clearly just as important to show that subjects lacking sequential effects also lack bimodality as to show the contrary. Another subject (F,

Fig. 2a) showed an overall distribution in the gap task that was not itself bimodal, but nevertheless broke down into two clearly separated peaks when analysed (significant at the 1% level, Kolmogorov–Smirnov test). This suggests that estimates of the proportion of people who show express saccades, based simply on bimodality in the overall distribution, may in general be too low (see also Kingstone & Klein, 1993).

4. Discussion

These results appear to suggest that bimodality of saccadic latency is a phenomenon exhibited in conventional paradigms using randomised sequences of target directions. It may well explain an earlier finding, that express saccades are reduced in number when the preceding trial evokes no saccade at all (Jüttner & Wolf, 1992), but the long time-scale implies that this phenomenon is

distinct from the directional effects observed between saccades evoked in rapid succession (Becker & Jürgens, 1979; Carpenter, 1988; McPeck, Skavenski, & Nakayama, 2000), that seem to represent a dichotomy between refractoriness and concurrent processing. The effects seen here appear very much greater than have been described in comparable manual tasks: the reduction in latency observed by Remington (1969) for two identical manual responses in succession, compared with two different ones, was little more than 10 ms, and far too small to give rise to bimodality. They are also much bigger than the effects observed in a study published while this paper was in preparation (Dorris et al., 1999) on saccades in monkeys using a paradigm similar in some respects to the second, two-gap, experiment, in which three of the four monkeys showed some 10 ms increase in saccadic latency for a saccade of the same amplitude and direction as its predecessor, compared with one returning to the previously-fixated locus. This is puzzling, as it seems contrary

Two-gap task

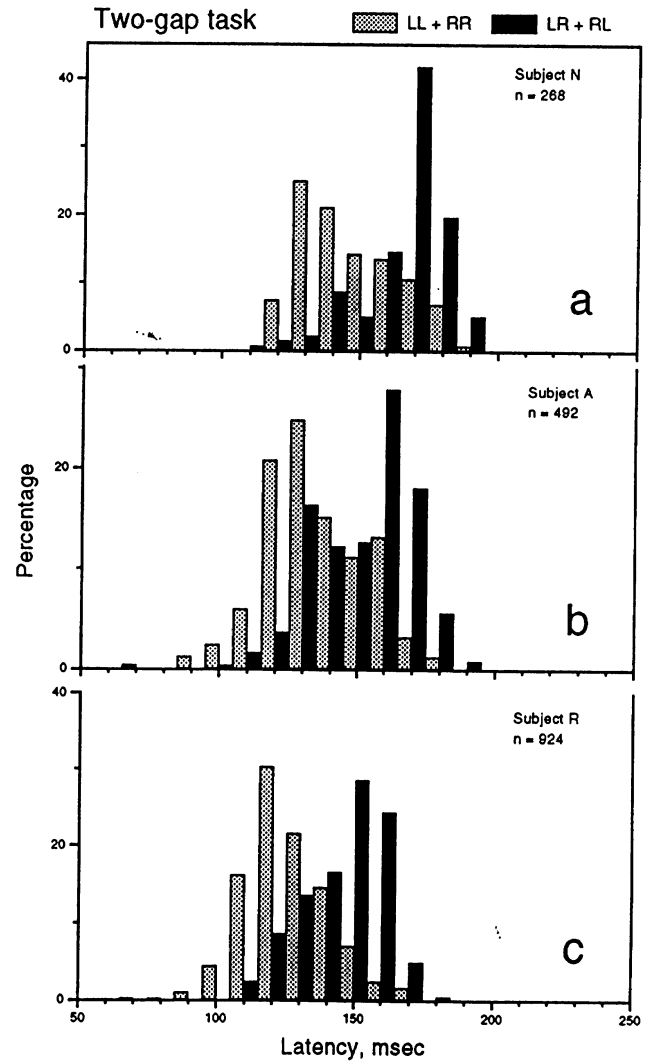
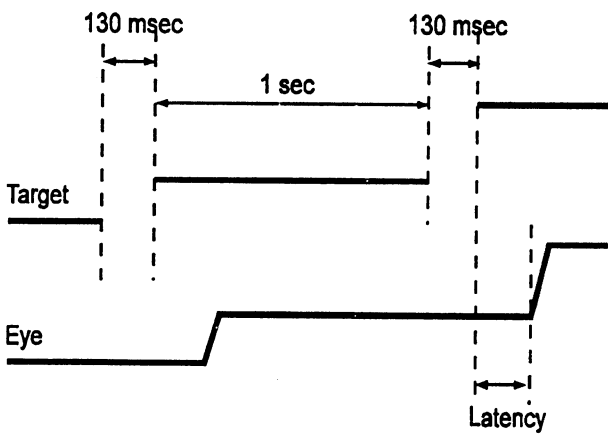


Fig. 3. Latency distributions in the two-gap task. Latencies of the second saccade were measured, and classified according as the first saccade was in the same direction (R,R + L,L) or different (R,L + L,R). Pairs of histograms are plotted as in Fig. 2 for three subjects. For subject N the contrast was 30% and the gap duration 150 rather than 130 ms; for subjects A and R and the contrast was 100%.

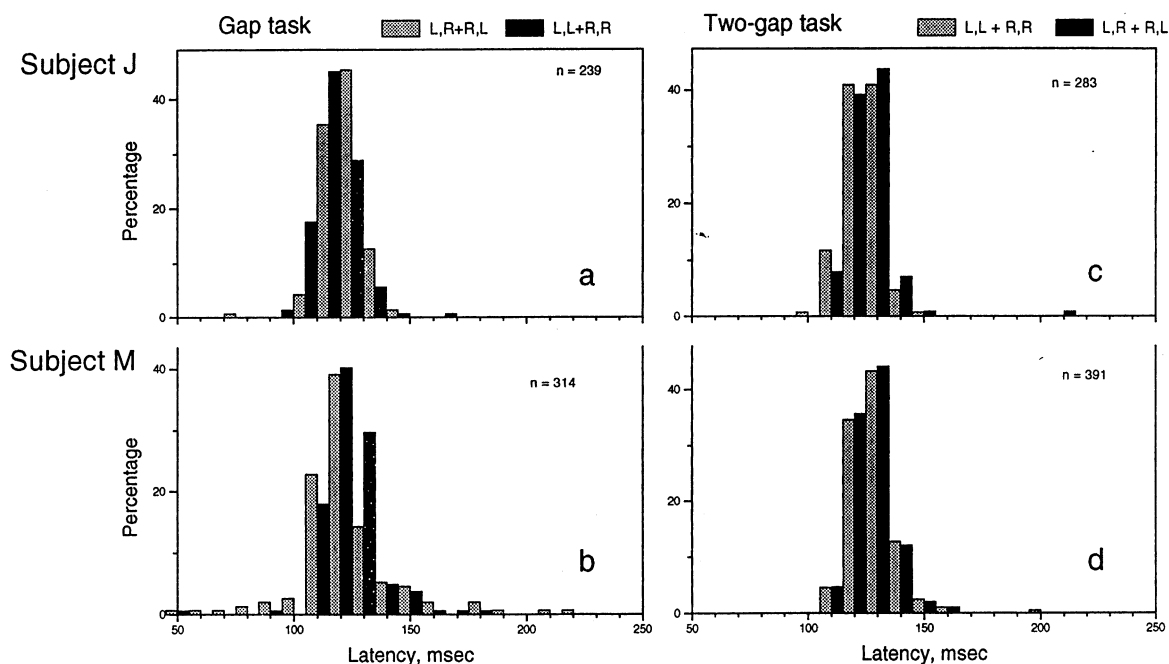


Fig. 4. Latency distributions for two subjects showing no difference in the gap task between the two conditions, and hence no bimodality, with a corresponding lack of separation of the histograms in the two-gap task (contrast 100%, gap 130 ms).

both to the present findings and to what would be expected from IOR. Since the effect appeared quite sharply localised, it is possible that it may be explained by some feature of the experimental set-up that caused localised retinal adaptation of some kind.

It is important to emphasise that while it is evident from the separated histograms that nearly all the bimodality is due to the order of presentation of targets, it is also clear that there is some 'cross-talk' in the sense that one sub-population often shows a secondary bump roughly corresponding to the main peak of the other. This might have been due to microsaccades intervening between the first and second responses, which if random in direction would have caused a degree of mixing of the two distributions; but this explanation did not appear to be borne out by examination of the raw records (though microsaccades of less than some 5 min arc would not have been seen). In any case, a previous study (Kingstone, Fendrich, Wessinger, & Reuter-Lorenz, 1995) has suggested that the influence of microsaccades on subsequent saccadic latency is negligible. It seems likely that although most of the bimodality is due to the effect of the preceding movement, there is a smaller residual effect that is independent of it.

How might the effect arise? Perhaps from the fact that expected targets evoke saccades with shorter latencies than unexpected ones (Carpenter & Williams, 1995): when a saccade is made to the left, the system might well predict that the next will also be to the left, as one might expect on theoretical grounds to occur during natural tracking. Indeed a clear correlation between the direc-

tions of successive spontaneous saccades has recently been described in a visual search task (Klein & MacInnes, 1999). It would be interesting to know whether subjects such as J and M show such a correlation, and also whether bimodality is modifiable by training such as prolonged tracking of targets jumping to left and right in strict alternating sequence. It is possible that these predictions become much more prominent in gap tasks, when there is a long period of expectation before the target actually moves, and thus more time for the system to make its estimates.

Recent neurophysiological investigations have demonstrated rather similar features in the activity of saccade-related neurons in the superior colliculus of monkeys. Neurons in deep layers which are increasingly active long before saccades to particular regions show a build-up of preparatory activity that is strongly dependant on prior probability (Basso & Wurtz, 1997; Munoz & Dorris, 1998), and similarly correlated with whether the saccade turns out to be an express one (Dorris, Paré, & Munoz, 1997). The enabling effect of a gap is easily explicable from the fact that fixation cells in the rostral pole of the colliculus tonically inhibit target cells while a target is present and fixated: the drop in their activity during the gap can thus be expected to enhance the excitability of peripheral target cells (Dorris & Munoz, 1995; Dorris et al., 1997; Everling, Paré, Dorris, & Munoz, 1998). Although, as the authors point out, this cannot in itself generate expressness, it provides the means by which the changing spatial patterns of expectation produced by particular sequences

of targets can more easily be translated into alterations in latency obvious enough to generate bimodality.

Why do a few subjects (Fig. 4) fail to show either bimodality, or sequence effects in the two-gap task? One possibility, suggested above, is that their behaviour in spontaneous visual scanning shows less correlation between successive saccades (though that begs the question why *that* should be), so that any such directional asymmetry is swamped by the generalised expectation provided by the offset of the fixation target. Idiosyncrasies of saccadic latency distribution are common both in both humans and monkeys (Schiller, Sandell, & Maunsell, 1987; Fischer et al., 1995), and — given our ignorance of the underlying mechanisms — trying to disentangle the effects of nature and nurture at this point is unlikely to be very rewarding.

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