Perception & Psychophysics
1993, 54 (2), 260-273

What are human express saccades?

ALAN KINGSTONE
University of California, Davis, California
and
RAYMOND M. KLEIN
Dalhousie University, Halifax, Nova Scotia, Canada

When a fixation point is removed 200 msec prior to target onset (the gap condition), human subjects are said to produce eye movements that have a short latency (80-120 msec), that form the early peak of a bimodal latency distribution, and that have been labeled "human express saccades" (see, e.g., Fischer, 1987; Fischer & Breitmeyer, 1987; Fischer & Ramsperger, 1984, 1986). In three experiments, we sought to obtain this express saccade diagnostic pattern in the gap condition. We orthogonally combined target location predictability with the presence versus absence of catch trials (Experiment 1). When target location was fixed and catch trials were not used, we found mostly anticipations. In the remaining conditions, where responses were under stimulus control, bimodality was not frequently observed, and, whether it was or not, latencies were not in the express saccade range. Using random target locations, we then varied stimulus luminance and the mode of stimulus presentation (LEDs vs. oscilloscope) in the gap and overlap (fixation is not removed) conditions (Experiment 2). Bimodality was rarely observed, the gap effect (overlap minus gap reaction time) was additive with luminance, and only the brightest targets elicited saccades in the express range. When fixed locations and no catch trials were combined with latency feedback (Experiment 3), we observed many responses in the express saccade range and some evidence for bimodality, but the sudden introduction of catch trials revealed that many early responses were not under stimulus control. Humans *can* make stimulus-controlled saccades that are initiated very rapidly (80-120 msec), but unless catch trials or choice reaction time is used, it is not possible to distinguish such saccades from anticipatory responses that are prepared in advance and timed to occur shortly after target onset. Because the express saccade diagnostic pattern is not a characteristic feature of human saccadic performance, we urge investigators to focus their attention on the robust *gap effect*.

Saslow (1967) was the first to report that saccadic latency is strongly influenced by the presence or absence of a visual fixation stimulus (see also L. E. Ross & S. M. Ross, 1980; S. M. Ross & L. E. Ross, 1981). Turning off a fixation stimulus 200 msec before a saccadic target appears (gap condition) results in shorter latencies than keeping the fixation stimulus on (overlap condition). In work published between 1984 and 1990, Fischer and colleagues (e.g., Boch & Fischer, 1986; Boch, Fischer, & Ramsperger, 1984; Braun & Breitmeyer, 1988, 1990; Fischer, 1987; Fischer & Boch, 1983, 1984; Fischer & Breitmeyer, 1987; Fischer & Ramsperger, 1984, 1986; Mayfrank, Mobashery, Kimmig, & Fischer, 1986) reported that human subjects produce a bimodal latency distribution in the gap condition, and they used the term *express saccade* to label the eye movements with extremely short response times (RTs from 80 to 120 msec) that form the early peak of this bimodal latency distribution. The following quote provides a typical example of the empirical claim: "When human subjects are asked to execute saccades from a fixation point to a peripheral target, if the fixation point is turned off some time (200 msec) before the target is turned on, the distribution of the saccadic reaction times is bimodal. The first peak occurs at about 100 msec and represents the population of express saccades" (Fischer & Ramsperger, 1986, p. 569). Early claims such as this one led many investigators to assume that bimodal saccadic latency distributions, with an early mode around 100 msec, are ubiquitous in the gap paradigm and might be employed as an express saccade diagnostic.

We sought to duplicate the pattern described by Fischer and colleagues—that is, to obtain bimodality with an express saccade mode in the 80-120 msec range (hereafter we will refer to this as the express saccade *diagnostic pattern*). Here we present the results of that effort and the conclusions that they suggest.

EXPERIMENT 1

In several early studies in which the express saccade diagnostic pattern has been reported, the gap duration was set at 200 msec, the gap condition was often run in a sep-

This research was supported by a Natural Sciences and Engineering Research Council of Canada Operating Grant to R.M.K. and a Fellowship Award to A.K. Address correspondence to A. Kingstone, Center for Neuroscience, 1544 Newton Court, University of California, Davis, CA 95616 (e-mail: afkingstone@ucdavis.edu) or R. M. Klein, Department of Psychology, Dalhousie University, Halifax, NS, Canada B3H 4J1 (e-mail: klein@ac.dal.ca.bitnet).

arate block, and the target for a saccade appeared at the same position on every trial (e.g., Fischer & Breitmeyer, 1987; Fischer & Ramsperger, 1984, 1986; Mayfrank et al., 1986). In some of the studies in which the diagnostic pattern has rarely been observed, the gap duration was, again, set at 200 msec; but gap and overlap conditions were combined randomly within a block of trials, and target position was varied unpredictably across trials (e.g., Edelman & Heinen, 1991; Fendrich, Hughes, & Reuter-Lorenz, 1991; Kalesnykas & Hallett, 1987; Reuter-Lorenz, Hughes, & Fendrich, 1991).

Thus, we hypothesized that the express saccade diagnostic pattern might depend on whether subjects know on what trial a gap will occur and/or where a target stimulus will appear, although it might not depend on the fact that the duration of the gap was set at 200 msec (see, e.g., Braun & Breitmeyer, 1988).[1] Because a body of research suggests that subjects frequently anticipate target onset if they know when and where a target will occur (see, e.g., Findlay, 1981; Flowers, 1978), we thought that the early peak in some of the reported bimodal distributions might be produced by such an anticipatory process. The present Experiment 1 was planned to test the importance of these considerations. There were four test conditions. In each condition, target onset was always preceded by a 200-msec gap. In one condition, target onset occurred on every trial at the same location (fixed). There were no catch trials. This follows one general format used by Fischer and colleagues (e.g., Fischer & Breitmeyer, 1987; Fischer & Ramsperger, 1984, 1986; Mayfrank et al., 1986; see also Braun & Breitmeyer, 1988, 1990). In a second condition, catch trials were included; that is, occasionally a target stimulus did not occur. Therefore, although the location of the target stimulus was predictable, the gap condition did not predict target onset on every trial. In a third condition, there were no catch trials but target position was random. Here, although the gap condition predicted target onset on every trial, the location of the target stimulus was unpredictable. In a fourth condition, catch trials were included and target position was random. Now the gap condition did not reliably predict target onset and the location of the target was unpredictable.

Method

Subjects. Four naive volunteers and the second author participated in four half-hour experimental sessions. All reported having emmetropic vision.

Stimuli and Apparatus. The visual stimuli, presented at eye level, were single pixel dots displayed on a Tektronix 604 oscilloscope. Stimulus luminance was about 2.2 log units above the foveal detection threshold. The subjects sat alone at a table with their heads placed in a chinrest. The testing room was dark and subjects were dark adapted for several minutes before the experiment began. A thumb switch was used by subjects to initiate each trial. A Biometrics (SGHV2) eye-movement monitor was used to sample the horizontal position of the subject's left eye every 2 msec. The oscilloscope, the thumb switch, and the eye-movement monitor were interfaced with a PDP-11/10 computer that controlled and recorded the sequence of events in each experiment.

Design and Procedure. The subjects were run individually in four experimental conditions: (1) target position fixed/no catch trials; (2) position fixed/catch trials; (3) position random/no catch trials; and (4) position random/catch trials. Each condition corresponded with a different test session on a different day. Each condition comprised two blocks of 100 trials (for a total of 800 experimental trials/subject in all). When the position of the target for a saccade was fixed, target onset always occurred at the same location (left or right of fixation) in a block of trials. The position of the target was changed between blocks. When the position of the target was random, target location (left or right) varied randomly from trial to trial. When catch trials were employed (i.e., no target was presented after fixation offset), they made up 20% of all the trials in a block and occurred at random within a block. The order of the four conditions was determined according to a Latin square principle and balanced across subjects.

The subjects were instructed about the details of the experiment, including the nature of the condition that they were about to receive. Each block of trials began and ended with an eye-position calibration procedure; this entailed brief subject-initiated recordings of eye position for each of seven stimulus locations: one at the center of the display, and the others at 6°, 4°, and 2° to the left and right of center. After the initial calibration, the central stimulus was illuminated. The subjects were instructed to initiate each trial when they had fixated the single dot in the center of the screen; sampling of eye position was also initiated by this response. Under all conditions, the fixation stimulus was extinguished 500 msec after trial initiation; 200 msec after fixation offset, a dot could appear 4° to the left or the right of center. The subjects were instructed to fixate this target stimulus as quickly and as accurately as possible when it appeared. They did not receive any response feedback. The target stimulus was extinguished, and the trial ended, 400 msec after a saccadic response was first detected or 600 msec after target onset, whichever came first. On catch trials, a target stimulus did not appear. Here the trial ended 400 msec after a saccade was first detected or 800 msec after fixation offset. The subjects were instructed to avoid making a saccadic response during a catch trial. In all situations, the intertrial interval (ITI) was 700 msec. Presentation of the fixation stimulus signaled that the next trial was ready to begin.

Data analysis. After the initial calibration for each test block, saccades were detected on line with a velocity criterion of >50°/sec. Specifically, a saccadic response was recorded when the left eye moved in the same direction by more than 0.1° on each of five consecutive 2-msec eye samples. The end of a saccade was recorded when the left eye failed to shift in the same direction by more than 0.1° on five consecutive samples. Saccadic amplitude was based on the initial calibration of each test block. Saccadic RT was defined as the latency required to initiate a saccade following target onset. In keeping with previous work (e.g., Braun & Breitmeyer, 1988, 1990; Fischer & Ramsperger, 1984, 1986; Mayfrank et al., 1986), saccades with RTs < 80 msec were considered anticipatory. Nonanticipatory saccades to the visual field not containing a target stimulus were recorded as directional errors. Failures to generate a saccade on trials in which a target was presented were considered misses. Saccades executed on catch trials were classified as false alarms. Blinks were excluded from the analysis and were not classified as errors.

Results

The response data, collapsed across subjects and target direction, are shown in Table 1. RT distributions (bin width, 10 msec) for each subject in each condition are shown in Figure 1. The most important results concern the comparison of anticipatory responses (RT < 80 msec) on target trials. It is immediately clear in Figure 1 that when target position is fixed and catch trials are absent, most saccades are executed less than 80 msec after target onset, that their modal latency is near 0 msec (for 4 out

Table 1
Mean Performance, Experiment 1

	Target Trials							Catch Trials		
	Anticipations (RT < 80 msec)		Correct Responses (RT > 79 msec)			% Directional		False Alarms		
Trials	%	Amplitude	%	RT	Amplitude	Errors	% Misses	%	Amplitude	% Correct
					Fixed Target Position					
Catch	7.3	3.3°	90.6	224	4.0°	1.1	1.0	17.7	3.0°	82.3
No catch	63.8	3.3°	35.9	153	3.2°	0.2	0.1			
					Random Target Position					
Catch	1.4	2.0°	98.3	209	3.9°	0.1	0.3	4.0	2.2°	96.0
No catch	0.8	1.2°	99.1	194	4.2°	0.0	0.1			

of 5 subjects), and that many saccades are released before a target is presented (i.e., saccadic latency is negative).

The data shown in Table 1 support this interpretation. Analysis of variance (ANOVA) of the percentage of anticipations revealed a significant interaction of target position predictability (fixed vs. random) × catch trial condition [$F(1,4) = 16.10, p < .05$]. The effects of position predictability [$F(1,4) = 16.67, p < .05$] and catch trials [$F(1,4) = 14.75, p < .05$] were also significant, but these two main effects derive completely from the interaction. Least significant difference (LSD) tests confirmed that when target position was fixed and there were no catch trials, the number of anticipatory responses was higher than in the other three conditions, which in turn did not differ between themselves.

Consider now the amplitude of anticipatory and correct saccades shown in Table 1. Note that anticipatory saccades are hypometric (i.e., they tend to undershoot the 4° target, a finding that is consistent with previous research, e.g., Findlay, 1981; Fischer & Ramsperger, 1986; Kalesnykas & Hallet, 1987).[2] For correct responses, there was no tendency to undershoot the 4° target position—except when target position was fixed and catch trials were absent. This is reflected by a significant interaction of target position predictability × catch trial condition [$F(1,4) = 8.67, p < .05$] and confirmed by LSD tests. Thus, in all conditions, save for the one in which target position was fixed and catch trials were absent, correct saccades tracked the position of the target stimulus with a high degree of resolution. It is interesting that when target position was predictable and catch trials were absent, the subjects' "correct" responses had the same hypometricity as did their anticipatory saccades (3.2° vs. 3.3°) [$F(1,4) < 1$]. In other words, when target position was known in advance and there were no catch trials, "correct" responses with their latency of 80 msec or greater do not appear to have been tracking the target stimulus any more than the anticipatory responses executed with a latency of less than 80 msec. The strong implication is that very few saccades in this condition were under the control of the target stimulus.

This conclusion is supported by an ANOVA of "correct" RTs. There was no main effect of position predictability [$F(1,4) = 4.73, p > .05$], but there was a significant effect of catch trial condition [$F(1,4) = 18.13, p < .05$], as well as a significant interaction [$F(1,4) = 87.36, p < .01$]. LSD tests showed that when target position was fixed and there were no catch trials, RT was faster than in the other three conditions.

Should these fast responses be considered express saccades? The evidence suggests not. Anticipatory RTs (RT < 80 msec) made up 64% of all the responses when target position was predictable and there were no catch trials. And the residual 36% "correct" responses (RT > 79 msec) possess an anticipatory topology. Moreover, the RT distributions for this condition (see Figure 1) do not suggest a unique subpopulation of express saccades within a bimodal distribution.

Inspection of the latency histograms reveals that in no case did a subject show evidence of the pattern we were looking for: bimodality, with one clear mode in the 80–120 msec range. There were some conditions under which subjects' performance suggested bi- or multimodality, but these were not identified as the diagnostic pattern because either the latencies of the early mode were not in the express range (e.g., R.K., position fixed/catch trials; J.T., position fixed/catch trials; C.G., position random/with no catch trials and catch trials) or the large number of anticipatory responses (in the position fixed/ no catch trials condition) discredits the residual "correct" saccades. The closest approximation of the pattern of bimodality that we were expecting (on the basis of latency histograms in the early papers from Fischer's laboratory, and claims like the one cited in the introduction) was shown by C.G. with unpredictable (random) target locations. The earliest peak of C.G.'s bimodal latency distributions extends from 130- to 190-msec latency, with a modal latency of 150–160 msec. Within the framework of Fischer and colleagues (Braun & Breitmeyer, 1988, 1990; Fischer, 1987; Fischer & Breitmeyer, 1987; Fischer & Ramsperger, 1984, 1986; Mayfrank et al., 1986), this first peak would be more closely identified with "fast regular saccades" (which range from 150 to 200 msec, with a modal latency of about 180 msec) than with "express saccades."

ANOVAs of directional errors and misses on target trials were nonsignificant. For catch trials, there were more false alarms [$F(1,4) = 15.95, p < .05$] when target position was fixed.

Discussion

Two findings from this study should be emphasized. The first is the absence, in all subjects, of the express sac-

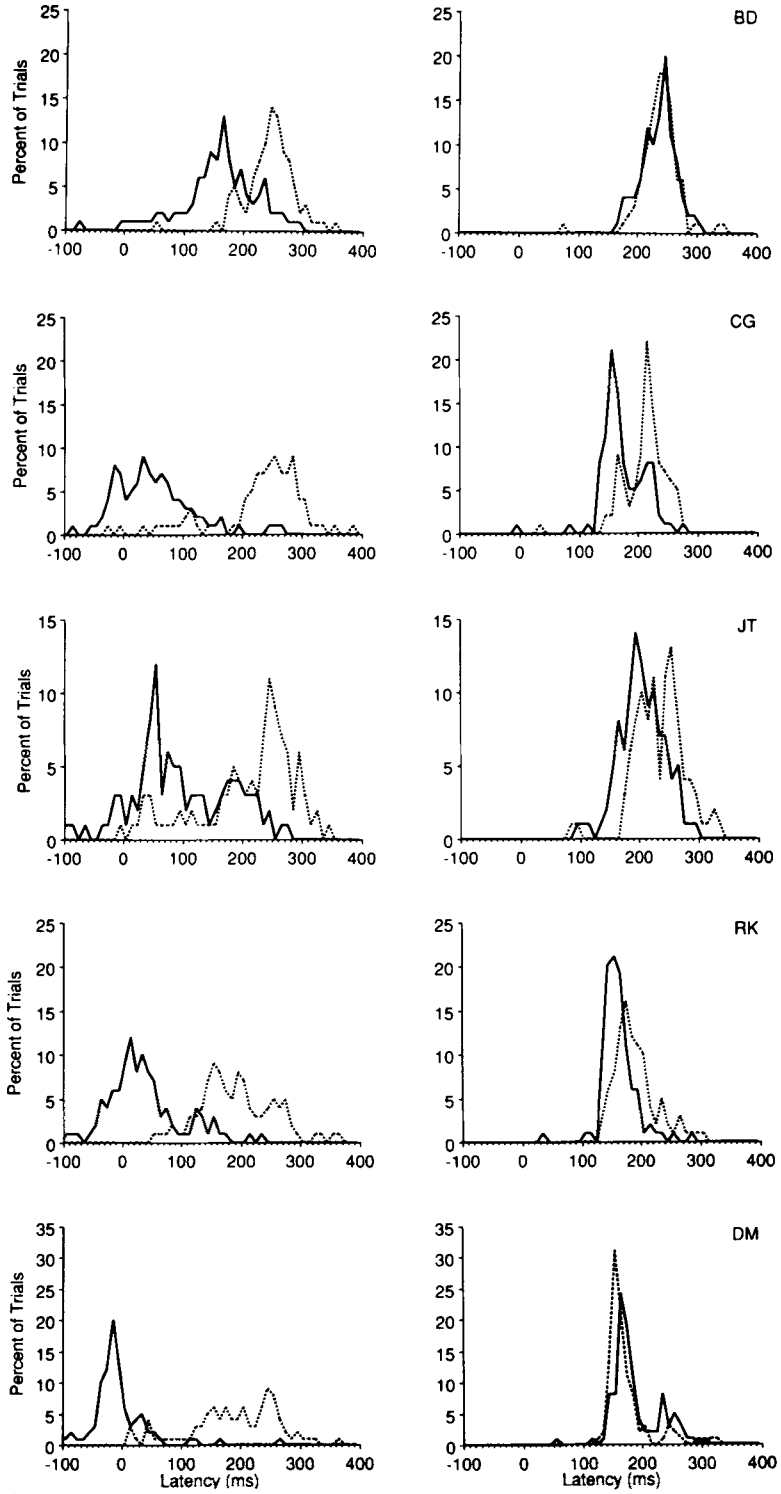

Figure 1. Frequency distributions of correct saccades from all subjects in Experiment 1. Each row represents the data from 1 subject. Data from blocks with target location fixed are shown on the left; data from blocks with target location randomized are shown on the right. In each figure, data from blocks without catch trials are plotted with a solid line and data from blocks with catch trials are plotted with a dotted line. Gap duration = 200 msec.

cade diagnostic pattern. Bimodality was clearly not the rule, and, when it was observed, the latencies of the early modes in these distributions fell outside the latency range for express saccades. Second, when target position was known in advance and catch trials were absent, anticipations (RT < 80 msec) occurred on 64% of all the trials. The 36% nonanticipatory saccades were in the express saccade latency range; but their topology was the same as that of the anticipatory saccades, and they failed to cluster into a unique subpopulation.

The data also show that response performance is essentially the same, whether one employs catch trials and/or target position randomization. However, presenting a target at the same position on every trial results in most responses' being anticipatory. This agrees with a wide number of reports that subjects will frequently anticipate target onset if they know when and where it will appear on every trial (see, e.g., Findlay, 1981; Flowers, 1978; Horrocks & Stark, 1964; Polidora, Ratoosh, & Westheimer, 1954; Stark, Vossius, & Young, 1961).

Our results contrast sharply with the findings of several previous studies of Fischer and colleagues, in which, under similar conditions, few anticipatory responses were observed[3] and the RT distributions were clearly bimodal (e.g., Braun & Breitmeyer, 1988, 1990; Fischer & Breitmeyer, 1987; Fischer & Ramsperger, 1984; Mayfrank et al., 1986). This difference might be due to the fact that in most of Fischer's experiments, RT was reported to the subject on each trial, whereas we did not provide our subjects with feedback on their saccadic performance. This possibility was tested in Experiment 3.

We are aware of one other study in which the presence and the absence of catch trials were compared. Varying the proportion of catch trials from 0% to 90%, and providing trial-by-trial feedback, Jüttner and Wolf (1991, 1992) tested 2 subjects with predictable target locations.[4] When there were no catch trials, one subject (E.B.) produced a unimodal latency distribution (peaking between 70 and 100 msec); the other subject (C.S.) produced a multimodal distribution (with the largest peak from 80 to 100 msec); and both subjects showed a number of anticipatory responses with latencies less than 80 msec. The introduction of catch trials eliminated the anticipatory responses, markedly reduced the number of saccades in the express latency range, and slightly increased the latency of the remaining "express" saccades. The express saccade diagnostic pattern was observed in several conditions (particularly with 25% and 50% catch trials). Jüttner and Wolfe report an interesting analysis with the data from some of these conditions. They sorted the saccadic responses as a function of the type of preceding trial. The express saccade diagnostic pattern was especially evident for responses following target trials. However, for responses following catch trials, the early mode (between 80 and 115 msec) was greatly reduced or eliminated. This difference is reminiscent of the increase in choice RT that follows an erroneous response (Rabbit, 1966). The subject may adopt a conservative or lax criterion for initiating a response to a target. Following target trials, there is a relatively high likelihood that the decision criterion will be lax; following a catch trial, the decision criterion becomes conservative. Jüttner and Wolfe's conclusion that bimodality may reflect "different modes of the decision processes" is consistent with this description (see also Kalesnykas & Hallett, 1987; and Kowler, 1990).

EXPERIMENT 2

In Experiment 1, the nonanticipatory saccadic latencies were generally slower than the latencies normally reported in express saccade research. Perhaps bimodality would have been more consistently obtained, and the early mode would have been in the express range, if latencies had been decreased by the use of more salient stimuli. To test this hypothesis, we ran subjects with bright dots or dots set at the same luminance as in Experiment 1 (dim dots). Also, because light-emitting diodes (LEDs) are often used in express saccade studies that report latencies in the express saccade range (80–120 msec), we also tested subjects with bright and dim LEDs. Experiment 2 differed from Experiment 1 in two other ways. First, the position of the target stimulus was always random, and catch trials were not included. Second, an overlap condition was run in conjunction with a gap condition. The overlap condition provided a baseline against which the effects of fixation offset (gap) on saccadic latency and on the shape of the latency distribution could be measured.

Method

Subjects. Five subjects, 3 of whom had participated in Experiment 1, took part in Experiment 2.

Stimuli and Apparatus. The only change from Experiment 1 was that the visual stimuli could be single-pixel dots or red LEDs that measured 5 mm in diameter. In the dim-dot condition, each dot was plotted once and refreshed every 10 msec. In the bright-dot condition, each dot was plotted 10 times and refreshed every 10 msec. The luminance of the dots on the oscilloscope was approximately 2.2 (dim dots) or 3.2 (bright dots) log units above the foveal detection threshold. Luminance of the LEDs was controlled by varying the current applied to each LED via resistors. The input current for a bright LED was 15 mA. The input current for a dim LED was 0.5 mA. The luminance of the LEDs was about 2.2 (dim LEDs) or 4.3 (bright LEDs) log units above the foveal detection threshold.

Design and Procedure. The calibration procedure remained unchanged from Experiment 1, except that when the test condition involved LEDs, these were used for calibration. There were four experimental conditions: two with LEDs (bright and dim), and two with dots (bright and dim). Each condition (e.g., bright LEDs) corresponded with a different half-hour test session. In each session, subjects received two blocks of 120 trials. Within each block, gap and overlap trials were equiprobable and were randomly combined. The order of the test conditions was counterbalanced across subjects and sessions. The subjects were instructed about the details of the experiment, including the nature of the condition they were about to receive. Each trial began with the illumination of a single central stimulus. Once subjects had fixated this stimulus, they initiated the trial and eye sampling began. Measured from trial initiation, target onset occurred after a randomly selected and equiprobable delay of 700, 850, or 1,000 msec. In the gap condition, the fixation stimulus was extinguished 200 msec before a saccadic tar-

get was presented. In the overlap condition, the fixation stimulus was not extinguished until the end of the trial. Target position, 4° to the left or right of central fixation, varied randomly and with equal probability across trials. The visual display was extinguished 400 msec after a saccadic response was first detected or 600 msec after target onset, whichever came first. The ITI was 700 msec.

Results

The response data, collapsed across subjects and target direction, are shown in Table 2. RT distributions (bin width, 10 msec) for all subjects and conditions are shown in Figure 2.

With the exception of Subject J.T., and the data of all subjects in the dim dot stimulus condition, Figure 2 shows that many responses in the gap condition were in the express saccade latency range. Interestingly, however, these very fast responses usually did not appear as part of a bimodal distribution. The express saccade diagnostic pattern, as described by Fischer and colleagues, was observed, but only by 1 of the subjects (G.B.) in three of the four stimulus conditions (R.K. shows clear bimodality in one condition, bright dots, but in that condition his fast mode is not in the express range). Comparing the *shapes* of the latency distributions for gap and overlap conditions, one is struck by their overall similarity. There is a trend for the distributions in the gap condition to be slightly "tighter"—with more responses packed into a smaller latency window. An interquartile range analysis was used to test this possibility. We found that the latency spanned by the middle 50% of responses (25%-75%) was marginally smaller for the gap condition (36 msec) than for the overlap condition (55 msec) [$F(1,4) = 5.92$, $.05 < p < .10$].

An additional important finding is illustrated in Figure 3. We see that RTs were fastest when the LEDs were bright, and slowest when the dots were dim. And latencies were slowed when the LEDs were dimmed, and speeded when the dots were brightened. However, in all cases, latencies in the gap condition were about 65 msec faster than in the overlap condition. In other words, the gap effect was the same for all types of stimulus display—bright or dim, LEDs or dots.

A two-way ANOVA confirmed that the gap effect was significant [$F(1,4) = 60.18$, $p < .01$], that there was a reliable difference between the four types of stimulus display [$F(3,12) = 3.66$, $p < .05$], and that there was no interaction [$F(3,12) < 1$]. LSD tests revealed that in the gap and overlap conditions, response latencies were significantly shorter for bright than for dim stimuli, and shorter for LEDs than for dots.

Two-way analyses of the accuracy data in Table 2 revealed that the proportion of correct [$F(3,12) = 7.65$, $p < .01$] and anticipatory saccades [$F(3,12) = 7.14$, $p < .01$] varied significantly across stimulus displays. LSD tests revealed that with bright dots there were more correct saccades and fewer anticipations than with any other stimulus display. No other significant result was obtained.

To summarize the important findings: (1) latencies were faster in the gap than in the overlap condition; (2) latencies were faster with LEDs than with dots, and faster with bright than with dim stimuli; (3) the gap effect remained constant at about 65 msec; (4) although there were many RTs in the 80- to 120-msec express saccade range in the gap condition, latency distributions only rarely (3 out of 20 times) conformed to the express saccade diagnostic pattern.

Discussion

Experiment 2 agrees with the findings in Experiment 1, in showing that the express saccade diagnostic pattern is not robust. We had hypothesized that bimodality might become a common occurrence in the gap condition if response latencies were speeded by brightening the dots on the oscilloscope or running subjects with LEDs. Although these manipulations had dramatic effects on saccadic RTs—with the bright LEDs, most modal latencies were in the 80- to 120-msec range—bimodality remained an atypical result. Experiment 2 also revealed that the shape

Table 2
Response Accuracy, Experiment 2

Stimulus Condition	% Correct	Amplitude	% Anticipations	% Directional Errors	% Misses
			Oscilloscope Presentation		
Dim					
Overlap	91.6	4.1°	6.7	0.2	1.5
Gap	87.3	4.0°	11.4	0.7	0.7
Bright					
Overlap	94.1	4.0°	5.5	0.0	0.3
Gap	89.1	3.9°	11.0	0.0	0.0
			LED Presentation		
Dim					
Overlap	93.5	3.9°	5.9	0.0	0.7
Gap	93.5	3.8°	4.5	1.3	0.0
Bright					
Overlap	99.5	4.1°	0.5	0.0	0.0
Gap	98.7	3.9°	0.5	0.3	0.2

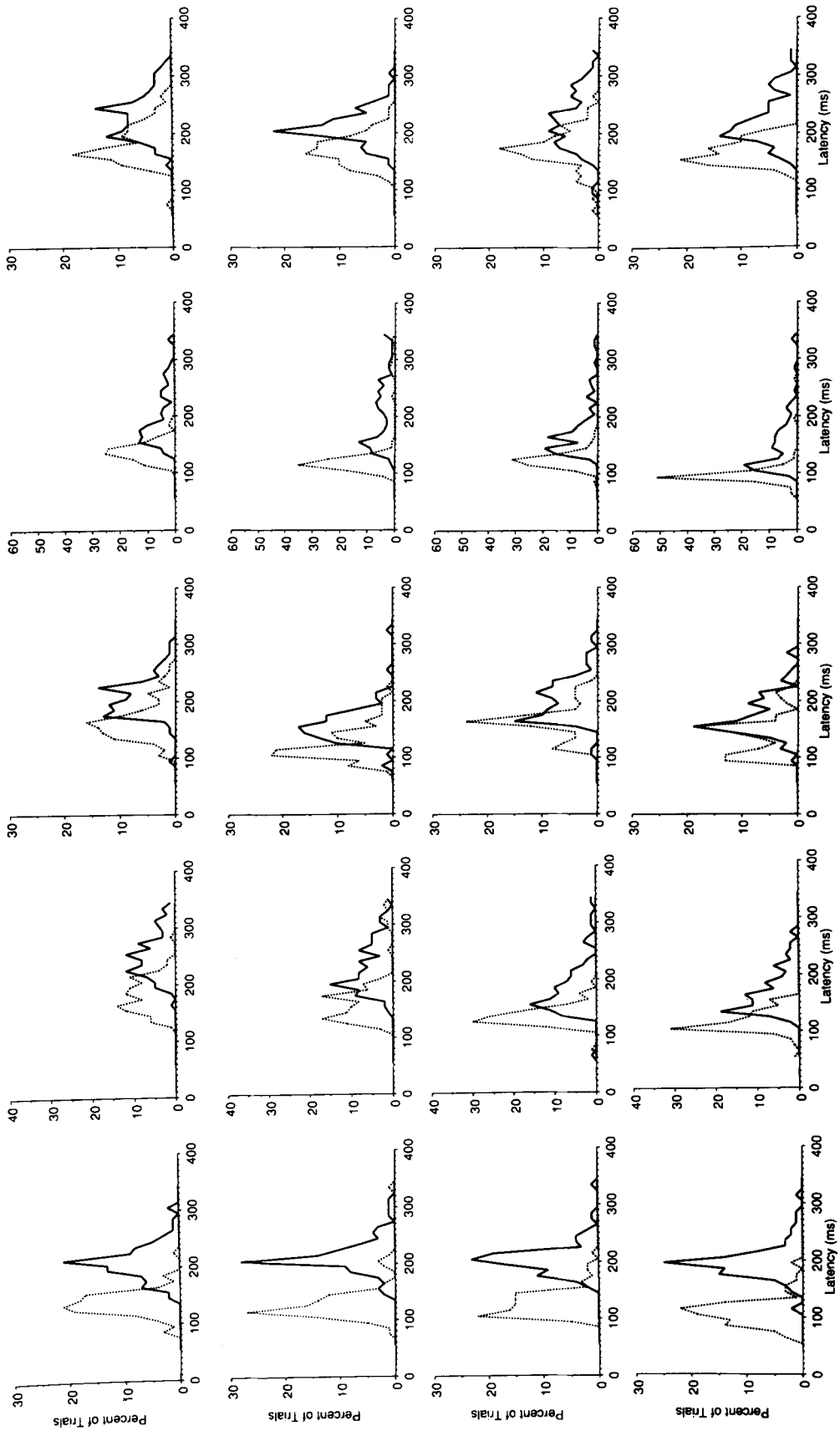

Figure 2. Frequency distributions of correct saccades from all the subjects in Experiment 2. Each column represents the data from a single subject. Each row shows data from a particular stimulus condition (from top to bottom: dim scope, bright scope, dim LEDs, bright LEDs). In each figure, data from the overlap condition are plotted with a thick/solid line and data from the gap (200-msec) condition are plotted with a thinner/dotted line. Note that the scale on the ordinate varies between subjects.

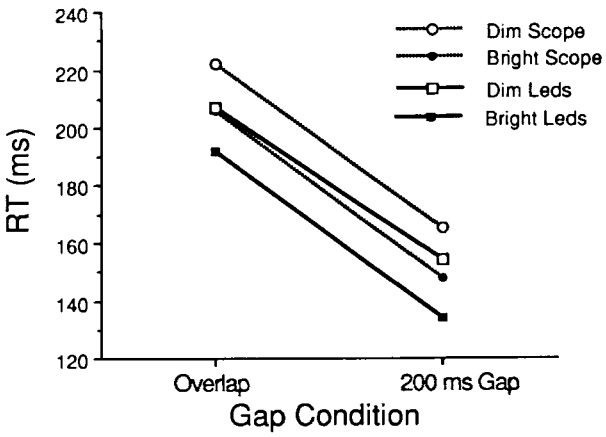

Figure 3. Mean reaction times from Experiment 2.

of the latency distributions in the gap condition did not differ markedly from the shape of the latency distributions in the overlap condition.

In all conditions, mean latencies in the gap condition were about 65 msec faster than in the overlap condition. Thus, the gap effect did not interact with changes in absolute response time caused by stimulus type, or stimulus luminance. This finding replicates a result reported recently by Reuter-Lorenz et al. (1991, Experiment 1). They found that for bright and dim LED targets, the gap effect remained constant at approximately 70 msec. This was true despite the fact that a number of saccadic responses were observed to occur in the express saccade range, and despite the fact that none of the 10 subjects produced a bimodal latency distribution. Interestingly, these authors concluded that they did not replicate Fischer and colleagues' bimodality result because of differences introduced into their experimental procedure—"i.e., the presence of catch trials, inclusion of an auditory warning signal, and position uncertainty" (Reuter-Lorenz et al., 1991, p. 169). The data from our first two experiments show that these methodological differences are not essential: when catch trials were excluded, auditory warnings omitted, and position uncertainty of the target eliminated, we rarely obtained the express saccade diagnostic pattern.

EXPERIMENT 3

Fischer and Ramsperger (1984, 1986) and other investigators (Braun & Breitmeyer, 1988, 1990; Jüttner & Wolf, 1992; Mayfrank et al., 1986) who have reported consistently obtaining the express saccade diagnostic pattern usually provided their subjects with latency feedback on every trial.[5] In contrast, some investigators who have not consistently obtained the express saccade diagnostic pattern did not provide latency feedback (e.g., Experiments 1 and 2 of the present study; Fendrich et al., 1991; Kalesnykas & Hallet, 1987; Reuter-Lorenz et al., 1991; Wenban-Smith & Findlay, 1991).

Fischer and colleagues discard saccadic RTs of about 80 msec or less as anticipatory. We thought it possible that their subjects might have learned (perhaps implicitly) to avoid executing saccades in this latency range. This raises the possibility that because subjects know when and where a target will appear, and because subjects know their RT on every trial, demand characteristics of the experiment might encourage them to occasionally prepare and execute a saccade that anticipates the onset of the target stimulus, while keeping the RT close to, but rarely faster than, the latency criterion that defines an anticipation (see Snodgrass, 1969; Snodgrass, Luce, & Galanter, 1967, for studies demonstrating subjects' ability to accurately place responses within a narrow latency window). The operation of such a strategy on a proportion of trials could be responsible for a mode just above the criterion for anticipations.

Once subjects are trained with the latency feedback procedure and learn how to prepare and time their saccades so that the latencies are frequently close to the experimenter-defined criterion for anticipations (e.g., RT $>$ 80 msec), one would predict that removing latency feedback would have little effect on performance (as was reported in some control conditions of Fischer & Ramsperger, 1984, 1986). Moreover, these trained subjects should, obviously, produce few anticipations, whereas untrained subjects produce many (as demonstrated by Fischer & Ramsperger, 1986, Figure 3; and Mayfrank et al., 1986, Figure 2B; and the present Experiment 1).

In Experiment 3, we explored the possibility that simple RT without catch trials, coupled with a latency feedback procedure, might produce a bimodal latency distribution composed of an early peak of short latency movements not under visual control of the target stimulus and a later peak of longer latency eye movements triggered by the target stimulus (see Findlay, 1981, and Horroks & Stark, 1964, who report precisely this form of bimodality). If methodological differences account for our failure to obtain the express saccade diagnostic pattern consistently, we should see this pattern more often by altering our methods to dovetail more closely with those described in the prototypical studies of Fischer and Ramsperger (1984, 1986). Thus, our stimuli were made larger (.25°), subjects no longer initiated each trial, there were only gap trials, the gap duration was fixed at 200 msec, the target appeared at the same location on every trial, and reaction time feedback was provided on each trial. In addition, we discouraged subjects from executing saccades with a latency less than 80 msec. We introduced catch trials after several training sessions without them. If, as hypothesized, the early peak of a bimodal latency distribution reflects saccades that are not under stimulus control, the introduction of catch trials should result in a number of false alarms or the elimination of the early peak and bimodality.[6]

Method

Subjects. Four naive subjects took part in Experiment 3.

Stimuli and Apparatus. The apparatus was the same as in Experiment 1. The calibration, fixation, and target stimuli were larger

than before, consisting of .25° circles composed of 12 evenly spaced dots with an additional dot in the center.

Design and Procedure. Two subjects received four test sessions, and 2 subjects received five test sessions, spread out over a 1-week period. Each session lasted about 1 h and was composed of a short warmup block of 20 trials, followed by three blocks of 100 test trials. The first 100-trial block on the 1st day was considered a practice block. For half the subjects, the target was always presented in the left visual field. For the remaining subjects, the target was always presented in the right visual field. Target direction was counterbalanced with number of sessions.

For each subject, there were no catch trials, until the final test session. In the last session, catch trials occurred at random on 20% of the trials.

Each block of trials began with the word *ready* displayed on an oscilloscope. The subjects used a thumb switch to initiate a block. Each trial began with the display of a circle (the fixation point) at the center of the screen. The subjects were instructed to fixate this stimulus when it appeared. The fixation point was removed 1.8 sec after its appearance.

On target trials, 200 msec after the fixation point was extinguished, a target circle was displayed for 1 sec, 4° to the left or right of the previous fixation stimulus. If subjects saccaded to the target, then, immediately after target offset, the RT (in milliseconds) was displayed for 1 sec at the center of the screen. The screen then went blank for 1 sec, followed by the onset of the fixation point signaling the start of the next trial. If subjects did not execute a saccade to the target, there was no RT feedback. Instead, after target offset, the screen went blank for 2 sec, followed by the onset of the fixation point.

On catch trials, a target was not presented. The screen went blank for 1 sec after fixation offset. The word *good* was then displayed for 1 sec at the center of the screen if subjects had avoided making a saccade; otherwise, the screen remained blank. Following this feedback interval, the screen was blank for an additional 1 sec. The onset of the fixation signaled a new trial.

The subjects were instructed to make an eye movement to the target as quickly and accurately as possible, but without anticipating its appearance. They were told that the RT would be displayed at the end of each trial when they moved in the correct direction, and that the word *good* would be displayed on catch trials if they did not move their eyes. It was explained that on spoiled trials—those with blinks, movements in the wrong direction, or eye movements on catch trials—there would be no feedback display. It was further explained that the RT would be negative if they moved their eyes before the target appeared, and that they should try to avoid making responses that were less than 80 msec. The experimenter reminded them of the instructions whenever necessary.

Eye position was sampled every 2 msec, beginning 1.3 sec after the appearance of the fixation point (500 msec prior to fixation offset) and continuing for 1.3 seconds.

Results

Response data, collapsed across subjects and target direction, are shown in Table 3. RT distributions (bin width, 10 msec) for all 4 subjects from the early sessions without catch trials and the final session with catch trials are shown in Figure 4.

Visual inspection reveals that all subjects produce a multimodal latency distribution during training without catch trials. Subject T.C.'s distribution is marked by two broad but clearly defined peaks. The early peak is centered at 150 msec (range, 70–230 msec), and the second peak is centered at 270 msec (range, 230–530 msec). Subject T.R. exhibits an early peak centered at 70 msec (range, 20–160 msec), and a second broad mode that spans 160–530 msec. Subject S.B. produces an early peak at 100 msec (range, 0–120 msec), and a second broad mode spanning from 120 to 220 msec. Subject M.K. has a small early peak centered at 100 msec (range, 0–120 msec) and a second larger peak centered at 150 msec (range, 120–230 msec). To examine the effect of training, we generated distributions from the first and second halves of the blocks with no catch trials (excluding the very first block, which was considered a practice block). Only 1 of the 4 subjects (T.C.) showed a reduction in the number of responses, with latencies in the 50–100 msec range.

Although there is considerable variability between subjects in the location of the early peaks in the latency distribution (T.C., 150 msec; T.R., 70 msec; S.B., 100 msec; M.K., 150 msec), wherever they are, these modes are abolished or markedly reduced when catch trials are introduced. To verify this conclusion statistically, we tabulated the number of responses with latencies between 70 and 150 msec. In the sessions before catch trials were introduced, subjects averaged 18.2% responses in this range, whereas after catch trials were introduced, they averaged only 2.3%—a difference that was highly significant [$F(1,3) = 19.76$, $p < .025$]. For Subjects T.R. and S.B., a bimodal latency distribution without catch trials becomes a clearly unimodal distribution when catch trials are introduced—with modes appearing at 240 and 260 msec, respectively. For Subjects T.C. and M.K., there remain signs of bimodality when catch trials are introduced. Importantly, these 2 subjects also produced a substantial number of false alarms on catch trials.

This combination of results—abolition of bimodality with a shift in the latency distribution to slower RTs, or continued bimodality with a number of false alarms—is precisely what one would expect if the early peak of a bimodal distribution consisted of many responses that were not under stimulus control.

Discussion

There were two main findings in Experiment 3. First, bi- or multimodality can be produced reliably across dif-

Table 3
Mean Performance, Target Position Random, Experiment 3

	Target Trials						Catch Trials		
	Anticipations (RT < 80 msec)		Correct Responses (RT > 79 msec)			% Directional	False Alarms		
Trials	%	Amplitude	%	RT	Amplitude	Errors	%	Amplitude	% Correct
No catch	22.6	3.1°	75.8	204	3.6°	0.3			
Catch	2.9	2.6°	93.2	262	3.5°	0.6	9.8	2.3°	90.2

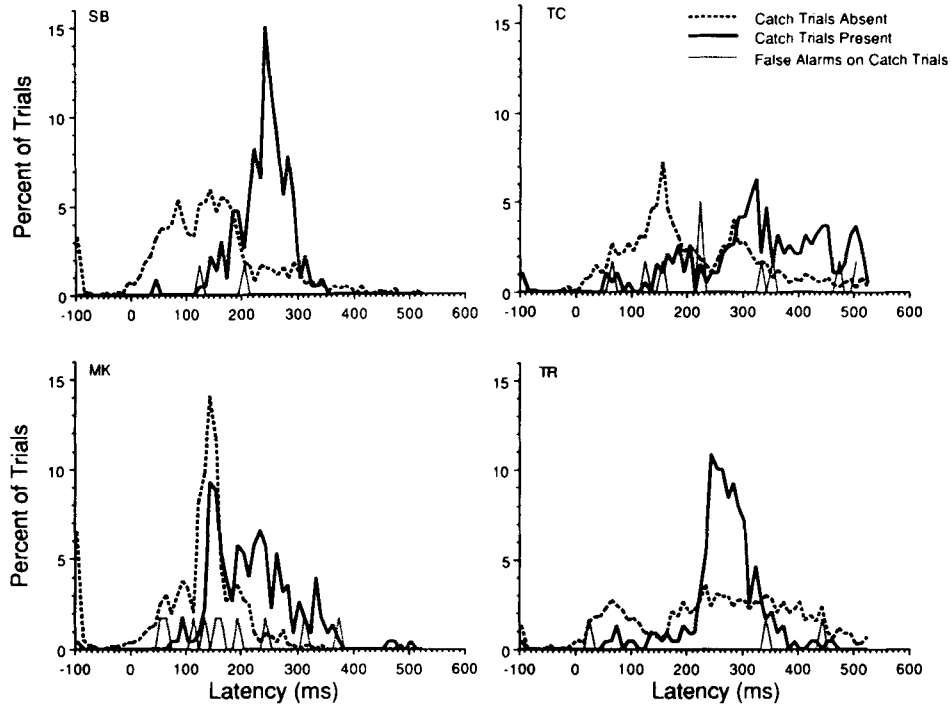

Figure 4. Frequency distributions of saccades from all the subjects in Experiment 3 (target location fixed). Each graph represents the data from a single subject. Dashed curves show data from three to four sessions when no catch trials were used. Solid curves show data from the final session, when catch trials were introduced. The dotted curves show saccades on catch trials (false alarms) from this final session. Gap duration = 200 msec.

ferent subjects. We achieved this result by providing subjects with advance knowledge of target onset time and position, by presenting a gap on every trial, by providing trial-by-trial RT feedback, and by instructing subjects that RTs faster than 80 msec would be considered anticipations. Second, introducing catch trials into this version of the task eliminates or significantly reduces the early mode of the latency distribution. Subjects who continue to show some sign of the early responses when catch trials are introduced also produce a number of false alarms on catch trials. Because catch trials ensure that responses are under stimulus control, or identify those that are not as false alarms, the data strongly argue that the early mode we have elicited in this experiment was not under stimulus control.

We believe that the conditions that we have used here (prior to the introduction of catch trials) approximate the prototypical format used by Fischer and Ramsperger (1984, 1986). With this procedure, we were able to elicit many saccades in the express range, which we subsequently showed were not under stimulus control. However, we must point out that the express saccade diagnostic pattern was not clearly seen in any of the 4 subjects of this experiment. The early mode in our data was broader within subjects and more variable across subjects than in Fischer and colleagues' studies, a discrepancy that must be attributed to remaining procedural differences.

GENERAL DISCUSSION

Eye movements with extremely short latencies (80-120 msec) that form the early peak of a bimodal latency distribution have been called *express saccades* (e.g., Fischer & Breitmeyer, 1987; Fischer & Ramsperger, 1984, 1986; Mayfrank et al., 1986). In the present paper, we tried to obtain this express saccade diagnostic pattern.

In Experiment 1 we employed conditions that have typically led to reports of this pattern. The offset of a central fixation stimulus was followed, after a temporal gap of 200 msec, by the onset of a peripheral target; the position and onset time of the target were fixed, and catch trials were not included. We found that saccades were hypometric, most were released less than 80 msec after target onset, the latency distribution was unimodal, and the mode was near 0 msec. These data converged on the conclusion that saccades were not under stimulus control. That is, subjects were generating a saccade in anticipation of, rather than in response to, the presentation of a target stimulus. To ensure that responses were under stimulus control, we also used catch trials and/or randomized

target position. Saccadic latencies now fell outside the express saccade range. Although bi- and multimodality was occasionally observed, the express saccade diagnostic pattern was not.

In Experiment 2, we sought to determine whether we could obtain stimulus-controlled saccades in the express saccade latency range (80–120 msec) by using increased stimulus salience to reduce the saccadic latencies. If we could, we expected that this might be achieved through the appearance of a new, distinct mode—the express saccade diagnostic pattern. Gap and overlap conditions were intermixed, target position was randomized, and stimulus salience (type of display and luminance) was varied between blocks. Increasing stimulus salience had a dramatic effect on RT, and in the gap condition, particularly with bright LEDs as targets, most saccadic latencies fell in the 80–120 msec range. Thus, in this condition, we consistently obtained saccades in the range one would consider very rapid (viz., *express*). Nevertheless, the express saccade diagnostic pattern was only rarely observed (1 out of 5 subjects; 3 out of 20 observations) and the gap and overlap latency distributions were remarkably similar in shape. Bimodality is not a necessary correlate of the latency reduction in the gap condition, even when this reduction brings saccadic RT into the express saccade latency range (see also Reuter-Lorenz et al., 1991).

In Experiment 3, we again used predictable (fixed) target locations and no catch trials. Recall that this paradigm produced a unimodal latency distribution of anticipatory saccades in Experiment 1. On this occasion, however, we included trial-by-trial latency feedback, which is a fundamental training procedure used by Fischer and colleagues (cf. Braun & Breitmeyer, 1988, 1990; Fischer & Ramsperger, 1984, 1986; Mayfrank et al., 1986). Results revealed that the latency distribution for all the subjects was bi- or multimodal, with the earliest mode ranging from 70 to 150 msec. We then introduced catch trials to determine whether the saccades contributing to the early mode were under stimulus control. The data fell into two patterns. Either the early mode was abolished, or a hint of bimodality was maintained at the cost of false alarms on catch trials. Both patterns converge on the conclusion that the saccades contributing to the early mode of the original bimodal distribution were not under stimulus control.

The results summarized so far suggest that subjects' knowledge about where in space a target will occur, and when it will occur, encourages predictive saccades that anticipate the onset of a target stimulus. When a latency criterion for anticipations is established, and RT feedback is provided, saccades are often prepared and executed so that they fall within a limited latency window close to criterion. The absence of catch trials permits these anticipations to go undetected.[7] We believe that reports of bimodality under these conditions reflect an early peak of short-latency saccades that are prepared in advance and anticipate target onset, and a later peak of longer latency saccades triggered by target onset.

It is crucial to note that we readily accept that saccades in the 100-msec latency range can occur when eye movements are under stimulus control, and that the express saccade diagnostic pattern (bimodality with the early mode in the 100-msec range) may be observed occasionally. Our data (e.g., Experiment 2; Kingstone & Klein, 1993) and those of other investigators (e.g., Jüttner & Wolf, 1991, 1992; Kalesnykas & Hallett, 1987; Reuter-Lorenz et al., 1991; and many studies of Fischer and colleagues) demonstrate this. However, the data also reveal that regardless of whether the fastest saccades are in the 100-msec range, when responses are under stimulus control, bimodality is not the typical finding (e.g., Edelman & Heinen, 1991; Fendrich et al., 1991; Kalesnykas & Hallett, 1987; Reulen, 1984a, 1984b; Reuter-Lorenz et al., 1991). Reuter-Lorenz et al. (1991), for example, found that only 1 of 13 different subjects produced a bimodal latency distribution when gap and overlap conditions were combined and target position was random. Similarly, Kalesnykas and Hallett (1987) obtained bimodality in 1 out of 4 subjects, and Wenban-Smith and Findlay (1991) obtained bimodality in 1 out of 3 subjects.

Recently Fischer and colleagues (Fischer et al., 1993) have reported a comprehensive study in which target position was random and RT feedback was not provided (as in Experiment 2 of this paper). Thirty-nine naive subjects (20 adults, 7 teenagers, 12 children) and 4 trained adults were tested in the gap and overlap conditions. Examination of the histograms from their naive adult subjects in the gap condition reveals that approximately half of these subjects do not show a latency distribution consistent with the express saccade diagnostic pattern (see also Fischer & Weber, 1990).[8] A 50% occurrence rate of the diagnostic pattern is still larger than that in the studies with similar methods cited above, but less robust than Fischer and colleagues had previously reported (e.g., Fischer & Breitmeyer, 1987; Fischer & Ramsperger, 1984, 1986). Whether procedural differences or sampling error account for the remaining discrepancy between Fischer and colleagues' results and those of other investigators is an intriguing puzzle for future research.

The express saccade diagnostic pattern has been observed, but it does not appear to be a characteristic feature of human saccadic latencies in the gap paradigm. However, suppose it was a reliable result. Would this mean that express saccades were a distinct class of eye movements governed by a special module of the oculomotor system? We think not. In a penetrating analysis of the early express saccade literature, Kowler (1990, pp. 52–54) writes:

> The suggestion of bimodality in a response latency distribution is insufficient reason to posit separate classes of saccades. . . . If the stimulus presented in a trial is the one the subject had anticipated, then reaction time is drawn from one distribution; if the other stimulus appeared, reaction time is drawn from a different distribution with a higher mean value. The resulting distribution of reaction times to the presentation of each stimulus is thus a weighted mixture of the two underlying distributions, one for trials in which the subject had been prepared for the stimulus, and the other for trials in which he had not been prepared. In

> other words, short-latency responses aren't special reflexes; they are responses to those stimuli which happen to have been correctly anticipated by the subject. The important point is that the same preparatory process needed to explain distributions of manual reaction times may explain distributions of saccadic reaction times as well without proposing separate short and long-latency saccadic mechanisms. (Kowler, 1990, pp. 53-54

A similar point is made by Kalesnykas and Hallett, (1987). This line of argument suggests that the very label "express saccade" may be misleading, because it implies an oculomotor response whose programming is in some way qualitatively different from that for normal saccades. To date, little evidence supports this characterization.[9]

In several studies, findings have been presented in terms of the proportion of express saccades observed in various experimental conditions (e.g., Braun & Breitmeyer, 1988; Fischer & Weber, 1990; Mayfrank et al., 1986). Our findings, as well as accumulating evidence in the literature, point to a classificatory/definitional conundrum that questions this practice (see also Klein & Kingstone, in press): How does an experimenter know whether a particular saccade is express or not? Bimodality, even when it occurs, does not permit the classification of a saccade as "express." First, it is possible to obtain bimodality with both peaks well outside the express range. Second, it is possible to obtain a unimodal distribution in which all the saccades are in the express range. An alternative is to rely on an absolute latency criterion—say, saccades in the 80-120 msec range. This diagnostic, however, is seriously challenged by the finding that a reduction in saccadic latency occasioned by fixation offset (the gap effect) is the same for bright and for dim targets (Reuter-Lorenz et al., 1991; and Experiment 2 in this paper). This additivity of the gap condition and stimulus luminance strongly suggests that whatever mechanism decreases saccadic latency in the gap condition when a target stimulus is bright does so also when a target stimulus is dim—and by an equal amount. However, because saccadic RTs to dim targets may not be in the express range, the absolute latency criterion would preclude these eye movements from being classified as "express saccades." We find it inconsistent and illogical that saccadic responses whose latencies have been reduced by equivalent amounts by the prior removal of fixation should be classified differently, merely because the sensory processing in one case is retarded by a lower target intensity. Since neither bimodality nor absolute latency can be reliably used to determine whether or not a particular saccade is "express," we encourage investigators to focus their attention on factors that modulate the gap effect (also see Kowler, 1990, for a different analysis that supports this recommendation).

We have shown, in agreement with Fischer and others, that following the removal of a fixation stimulus very short latency saccades that *are* under stimulus control are sometimes observed. Like an "express" train," such saccades could be fast for two reasons: (1) they could involve a different programming pathway (in the train metaphor, a different kind of engine and/or tracks), and (2) they could involve fewer or shorter processing stages (as when the same train does not make all the stops or makes briefer ones). Which class of explanation applies to the gap effect? We anticipate that evidence from studies of the saccadic performance of normal human subjects (e.g., Kingstone & Klein, in press), patients with brain damage (e.g., Guitton, Buchtel, & Douglas, 1985), and animals undergoing direct manipulations of and/or measurements from their central nervous systems (e.g., Munoz & Wurtz, 1992) will converge on an answer to this question.

REFERENCES

Boch, R., & Fischer, B. (1986). Further observations on the occurrence of express-saccades in the monkey. *Experimental Brain Research*, **63**, 487-494.

Boch, R., Fischer, B., & Ramsperger, E. (1984). Express-saccades of the monkey: Reaction times versus intensity, size, duration, and eccentricity of their targets. *Experimental Brain Research*, **55**, 223-231.

Braun, D., & Breitmeyer, B. G. (1988). Relationship between directed visual attention and saccadic reaction times. *Experimental Brain Research*, **73**, 546-552.

Braun, D., & Breitmeyer, B. G. (1990). Effects of reappearance of fixated and attended stimuli upon saccadic reaction time. *Experimental Brain Research*, **81**, 318-324.

Edelman, J. A., & Heinen, S. J. (1991, May). *Directional effects of the fixation-offset/target-onset gap on saccadic latency*. Paper presented to the Association for Research in Vision and Ophthalmology, Sarasota, FL.

Fendrich, R., Hughes, H. C., & Reuter-Lorenz, P. A. (1991). Fixation-point offsets reduce the latency of saccades to acoustic targets. *Perception & Psychophysics*, **50**, 383-387.

Findlay, J. M. (1981). Spatial and temporal factors in the predictive generation of saccadic eye movements. *Vision Research*, **21**, 347-354.

Fischer, B. (1987). The preparation of visually guided saccades. *Reviews in Physiology, Biochemistry & Pharmocology*, **106**, 1-35.

Fischer, B., & Boch, R. (1983). Saccadic eye movements after extremely short reaction times in the monkey. *Experimental Brain Research*, **260**, 21-26.

Fischer, B., & Boch, R. (1984). Express-saccades of the monkey: A new type of visually guided rapid eye movements after extremely short reaction times. In A. G. Gale & F. Johnson (Eds.), *Theoretical and applied aspects of eye movement research* (pp. 403-408). Amsterdam: North-Holland.

Fischer, B., & Breitmeyer, B. (1987). Mechanisms of visual attention revealed by saccadic eye movements. *Neuropsychologia*, **25**, 73-83.

Fischer, B., & Ramsperger, E. (1984). Human express saccades: Extremely short reaction times of goal directed movements. *Experimental Brain Research*, **57**, 191-195.

Fischer, B., & Ramsperger, E. (1986). Human express-saccades: Effects of randomization and daily practice. *Experimental Brain Research*, **64**, 569-578.

Fischer, B., & Weber, H. (1990). Saccadic reaction times of dyslexic and age-matched normal subjects. *Perception*, **19**, 805-818.

Fischer, B., & Weber, H. (in press). Express saccades and visual attention. *Behavioral & Brain Sciences*.

Fischer, B., Weber, H., Biscaldi, M., Aiple, F., Otto, P., & Stuhr, V. (1993). Separate populations of visually guided saccades in humans: Reaction times and amplitudes. *Experimental Brain Research*, **92**, 528-541.

Flowers, K. A. (1978). The predictive control of behaviour: Appropri-

ate and inappropriate actions beyond the input in a tracking task. *Ergonomics*, **25**, 176-191.

GUITTON, D., BUCHTEL, H. A., & DOUGLAS, R. M. (1985). Frontal lobe lesions in man cause difficulties in suppressing reflexive glances and in generating goal-directed saccades. *Experimental Brain Research*, **58**, 455-472.

HORROCKS, A., & STARK, L. (1964). Experiments on error as a function of response time in horizontal eye movements (Quarterly Research Report No. 72, pp. 267-269). Cambridge, MA: Massachusetts Institute of Technology, MIT Research Laboratories in Electronics.

JÜTTNER, M., & WOLF, W. (1991). Human express saccades: Catch trials influence the probability of their occurrence. In R. Schmid & D. Zambarbieri (Eds.), *Oculomotor control and cognitive processes* (pp. 163-176). Amsterdam: Elsevier.

JÜTTNER, M., & WOLF, W. (1992). Occurrence of human express saccades depends on stimulus uncertainty and stimulus sequence. *Experimental Brain Research*, **89**, 678-681.

KALESNYKAS, R. P., & HALLETT, P. E. (1987). The differentiation of visually guided and anticipatory saccades in gap and overlap paradigms. *Experimental Brain Research*, **68**, 115-121.

KINGSTONE, A., & KLEIN, R. M. (in press). Visual offsets facilitate saccadic latency: Does pre-disengagement of visuo-spatial attention mediate this gap effect? *Journal of Experimental Psychology: Human Perception & Performance*.

KINGSTONE, A., & KLEIN, R. M. (1993). *Visual offsets facilitate saccadic latency: There are two components to this gap effect*. Manuscript in preparation.

KLEIN, R. M., & KINGSTONE, A. (in press). Why do visual offsets reduce saccadic latencies? *Behavioral & Brain Sciences*.

KOWLER, E. (1990). The role of visual and cognitive processes in the control of eye movement. In E. Kowler (Ed.), *Eye movements and their role in visual and cognitive processes* (pp. 1-70). Amsterdam: Elsevier.

MAYFRANK, L., MOBASHERY, M., KIMMIG, H., & FISCHER, B. (1986). The role of fixation and visual attention in the occurrence of express saccades in man. *European Archives of Psychiatry & Neurological Science*, **235**, 269-275.

MUNOZ, D. P., & WURTZ, R. H. (1992). Role of the rostral superior colliculus in active visual fixation and execution of express saccades. *Journal of Neurophysiology*, **67**, 1000-1002.

POLIDORA, V. J., RATOOSH, P., & WESTHEIMER, G. (1954). Precision of rhythmic responses of the oculomotor system. *Perception & Motor Skills*, **7**, 247-250.

RABBITT, P. M. A. (1966). Errors and error correction in choice-response tasks. *Journal of Experimental Psychology*, **71**, 264-272.

REULEN, P. H. (1984a). Latency of visually evoked saccadic eye movements: I. Saccadic latency and the facilitation model. *Biological Cybernetics*, **50**, 251-262.

REULEN, P. H. (1984b). Latency of visually evoked saccadic eye movements: II. Temporal properties of the facilitation mechanism. *Biological Cybernetics*, **50**, 263-271.

REUTER-LORENZ, P. A., HUGHES, H. C., & FENDRICH, R. (1991). The reduction of saccadic latency by prior offset of the fixation point: An analysis of the gap effect. *Perception & Psychophysics*, **49**, 167-175.

ROSS, L. E., & ROSS, S. M. (1980). Saccade latency and warning signals: Stimulus onset, offset, and change as warning events. *Perception & Psychophysics*, **27**, 251-257.

ROSS, S. M., & ROSS, L. E. (1981). Saccade latency and warning signals: Effects of auditory and visual stimulus onset and offset. *Perception & Psychophysics*, **29**, 429-437.

SASLOW, M. G. (1967). Effects of components of displacement-step stimuli upon latency for saccadic eye movement. *Journal of the Optical Society of America*, **57**, 1024-1029.

SCHILLER, P. H., SANDELL, J. H., & MAUNSELL, J. R. (1987). The effect of frontal eye field and superior colliculus lesions on saccadic latencies in the rhesus monkey. *Journal of Neurophysiology*, **57**, 1033-1049.

SNODGRASS, J. G. (1969). Foreperiod effects in simple reaction time: Anticipation or expectancy? *Journal of Experimental Psychology Monographs*, **79**(3, Pt. 2), 1-19.

SNODGRASS, J. G., LUCE, R. D., & GALANTER, E. (1967). Some experiments on simple and choice reaction time. *Journal of Experimental Psychology*, **75**, 1-17.

STARK, L., VOSSIUS, G., & YOUNG, L. R. (1961). Predictive control of eye tracking movements. *IRE Transactions on Human Factors Electronics*, **HFE-3**, 52-57.

WENBAN-SMITH, M. G., & FINDLAY, J. M. (1991). Express saccades: Is there a separate population in humans? *Experimental Brain Research*, **87**, 218-222.

NOTES

1. Early reports by Fischer and colleagues (see Fischer & Weber, in press, for a review) that an express saccade diagnostic pattern is consistently obtained even when target onset is unpredictable conflicts with this hypothesis. Inadequate descriptions of their methodology and data, however, compromise any interpretation and evaluation of this claim. For instance, it is frequently unclear what order various conditions were run in, how many of the identified subjects were tested in each condition, what number of trials each subject received in each condition, what the error rate was in any given condition, what the latency distribution for each subject looked like, and whether any differences in mean saccadic latencies (when reported) are statistically significant. More recent reports by Fischer and colleagues (e.g., Fischer et al., 1993) demonstrate that when target position is unpredictable, the diagnostic pattern is not as robust as first reported, a point we return to in the General Discussion.

2. There are many anticipations when the target position is fixed, and these undershoot the target by about 18%. Anticipations with random target position appear to be much more hypometric, but they occur so infrequently that we do not think this comparison valid.

3. Fischer and Ramsperger (1986), however, did report many anticipations (RT $<$ 80 msec) when their naive subjects were at a relatively low level of practice (after only 200 trials with random targets). With increasing practice, the number of these anticipations was reduced significantly. The subjects in our experiment experienced the corresponding condition (fixed location with no catch trials) after between 0–600 trials (depending on the random order to which they had been assigned), and thus, in terms of practice level, our predictable-no-catch data are roughly comparable to those reported in Figure 3A of Fisher and Ramsperger (1986).

4. The two papers describe the same data, except that in the 1991 version, data with unpredictable target locations are presented from a single subject. The data shown in corresponding figures (Figure 3 from the 1991 paper and Figure 1 from the 1992 paper) reveal a troublesome latency discrepancy of 15–20 msec. The more recent paper does not refer to the earlier one, so it is not possible to know which representation is correct. In our discussion, the ranges for the various peaks are quite large, because we give neither version priority and have assumed that the peaks in the true distributions lie somewhere between the upper and lower limits defined by the discrepant reports.

5. Braun and Breitmeyer (1990) do not mention whether latency feedback was or was not provided. However, we assumed that feedback was provided because (1) their methodology appears to be very similar to that of Braun and Breitmeyer (1988), where latency feedback was provided, and (2) the anticipation rate was low, even though the onset position and onset time of the target were certain and catch trials were not included.

6. We would like to thank Tram Neill and an anonymous reviewer for encouraging us to conduct Experiment 3.

7. Topological analyses do not provide a reliable basis for the separation of anticipatory and stimulus-controlled saccades. For instance, when target position is certain, anticipatory and visually guided saccades may have the same amplitude or amplitude-velocity relation (cf. Findlay, 1981).

8. Fischer and Weber (1990) present their latency distribution data collapsed across subjects, which makes firm conclusions of how many subjects showed the diagnostic pattern difficult. They do report, however, that untrained adults show only a small percentage (10%) of express saccades in the gap paradigm.

9. Most often cited in support of the view that express saccades may involve a special programming machinery are studies of the brains of monkeys previously trained to perform various saccadic tasks (e.g., Schiller, Sandell, & Maunsell, 1987). Schiller et al. (1987), for example, have been cited in support of the view that express saccades are generated in the colliculus (while slower saccades are not). However, they point out that this conclusion from their lesion study, while tempting, would be an oversimplification (p. 1042). More recently, Munoz and Wurtz (1992) demonstrated that chemical deactivation of the fixation neurons in the rostral pole of the superior colliculus severely disrupts the monkey's performance in a memory-guided saccade task (because monkeys reflexively glance at the target, with latencies in the express range, instead of waiting for the fixation point to go off). They suggest that the rostral superior colliculus is necessary to *suppress* express saccades. We suggest that engaging the oculomotor system at fixation appears to be a function of the rostral superior colliculus, and that disengaging the oculomotor system from fixation is one stage in the *normal* sequence of saccadic programming. Removal of fixation or deactivation of the fixation system allows this stage to be bypassed, thus decreasing saccadic latencies.

(Manuscript received October 18, 1991;
revision accepted for publication February 15, 1993.)