Delay of stimulus presentation after the saccade in visual search

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Delay of stimulus onset after each saccade in visual search decreased oculomotor and manual reaction times, with a greater effect occurring for the oculomotor response. The saccadic oculomotor reaction might have been facilitated in three ways: by the facilitation of reaction with a foreperiod warning stimulus, by the attenuation of saccadic suppression effects due to the stimulus onset delay, or by the use of a strategy of preprogramming fixation durations. The results support a model of visual search using preprogrammed control of visual fixation durations.

To study how the processing of visual information is related to the intermittent visual input produced by sequential fixations in search, one might simply measure the duration of a subject's fixations while he is engaged in a visual search task. However, this measurement confounds two factors that might contribute to the control of fixation duration: (1) the time required for cognitive processing of a visual stimulus, and (2) the time required for the oculomotor system to initiate the next successive saccade. Independent estimates of the time required for each of these two factors have been obtained in situations devised to hold the other factor constant in some way. For example, the time required for processing has been estimated independently of eye movements by rapidly presenting a sequence of stimuli at a single retinal location. Under these conditions, the minimum exposure time required for the correct identification of each stimulus is 200 to 300 msec, very close to the range of fixation durations normally observed in skilled readers (Kolers & Katzman, 1966; Travers, 1973, 1975). Similarly, the time between two saccades has been measured, and again about 200 msec appears as the minimum time that must elapse between saccades (Tinker, 1958; Westheimer, 1954). Both perceptual and oculomotor factors appear to influence the minimum fixation duration.

The present experiment was designed to clarify how these factors determine fixation duration in search. A delay was imposed between the beginning of a fixation and the onset of the visual stimulus to be observed during that fixation. It was expected that processes that depend on or are limited by the

rate of information processing would be synchronized with stimulus onset, and thus be affected by the imposition of a delay, whereas processes primarily under other oculomotor influences would be more affected by the beginning of a fixation than by stimulus onset per se. For instance, if the time required for processing is the limiting factor in the rate of visual search in this task, then imposition of stimulus onset delay should lengthen fixation durations in direct proportion to the delay imposed, since processing cannot begin until the stimulus is presented. Conversely, if it is only the rate at which one saccade can follow another that imposes a lower limit on the duration of individual fixations, then the delay imposed might have no effect on fixation duration.

METHOD

Subjects

Six subjects (three female) with normal vision (corrected if necessary) served in the experiment. Half the subjects were paid volunteers; the others, salaried laboratory staff.

Apparatus

The horizontal electrooculogram (EOG) was recorded using Grass E4S silver-silver chloride electrodes applied with Grass EC2 cream as close as possible to the external canthi. Electrode-to-electrode resistance was established at 3,000 ohms or less. An ear-clip electrode served as ground reference. The amplified (Grass, P-18 dc amplifier) EOG was recorded by a PDP-8/e computer. Subjects were lightly restrained in a headrest with a chin support.

The computer generated stimuli on a Tektronix oscilloscope with P31 phosphor (32 msec delay time) located 27 cm in front of the subject. The two stimulus locations were located 7.5 deg on either side of the center of the oscilloscope screen, and were presented on a background of about 0.6 cd/m². Two stimuli were made up of 12 dots from a 4 by 6 dot matrix, forming the characters "X" or "O." Stimuli subtended an angle of 0.6 deg horizontally and 1.0 deg vertically, and their brightness was adjusted for comfortable visibility without persistence on the oscilloscope screen. The subject operated a microswitch with the thumb of the preferred hand. Red and green pilot lights mounted below the oscilloscope provided performance feedback after each trial.

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Procedure

Figure 1A shows the stimulus display as it appeared to the subject, and the apparatus that was used. Each trial began with the screen blank except for a central fixation point. To begin a trial, the subject pressed the microswitch and stared at the center fixation point. This fixation was used by the computer to recalibrate the recording received from the subject at the beginning of each trial (necessary because of drift in the EOG signal, particularly in the early part of each session). After a fixation of 450 msec, the center fixation point was extinguished and two other fixation points were presented at the stimulus locations. The subject then searched for the target stimulus (which was "O" throughout), by alternately fixating the two stimulus locations.

In detail, the presentation of the stimuli during each fixation was as follows (see Figure 1B): After the beginning of a fixation on one side, the screen was blank for the period of stimulus onset delay (0 to 150 msec). Then the stimulus appeared at the location of the subject's fixation for the stimulus duration (100 to 300 msec). Following stimulus offset, if the subject was still looking at that location, the stimulus was replaced by a masking noise pattern of 12 randomly placed dots. The presentation of the stimulus or mask was terminated when the subject looked away from it.

If the stimulus presented during a fixation was the nontarget ("X") stimulus, the subject continued his search by making a saccade to the other side; his oculomotor latency was recorded from the onset of that nontarget stimulus to that saccade. If the stimulus presented was the target ("O") stimulus, the subject released the microswitch as quickly as possible, and manual reaction time was recorded from the onset of that target stimulus to the release of the microswitch. An error was recorded on a trial if the subject released the microswitch before the target onset, or made any additional saccades after the target onset; errors were reported to the subject by illumination of the red pilot light for 1 sec, while correct responses were reported by illumination of the green light for 0.5 sec.

A complete session comprised 240 trials during which the number of nontarget stimuli preceding the target was varied from 2 to 6 (there were 48 trials with each number); the stimulus onset delay was varied from 0 to 150 msec in steps of 30 msec, and the stimulus duration was selected from the values 100, 150, 200, and 300 msec. Values for these variables were combined in a counterbalanced order; the values were the same within each trial. Each subject received practice until accuracy was 90% or above; then five additional sessions were conducted.

RESULTS

Overall mean fixation duration, oculomotor latency, and reaction time are shown in Table 1 for each stimulus onset delay.

Figure 2 shows the effect of stimulus onset delay on oculomotor latency and reaction time for the four stimulus durations used. Both oculomotor latency and reaction time were facilitated by a preceding delay, but the effect was greater for the oculomotor latency than for reaction time. A 2 by 4 by 6 analysis of variance showed significant main effects for the response measure [reaction time vs. oculomotor latency: $F(1,5) = 44.26$; $p < .002$], stimulus duration [$F(3,15) = 16.08$; $p < .001$], and stimulus onset delay [$F(5,25) = 189.68$; $p < .0001$]. The two-way interactions of Response Measure by Stimulus Duration [$F(1,5) = 16.74$; $p < .01$] and Response Measure by Stimulus Onset Delay [$F(1,20) = 46.88$;

Figure 1. (a) The stimulus display (left) and apparatus used. (b) The sequence of stimuli presented in a typical trial. A fixation on a nontarget stimulus on the left was followed by a fixation on a target stimulus on the right. Stimulus onset delay was 90 msec and stimulus duration was 200 msec. Note that the oculomotor latency and reaction time are measured from stimulus onset, and that oculomotor latency plus stimulus onset delay equals fixation duration.

$p < .001$] were significant using a conservative test for interactions in repeated measures designs (Winer, 1962, p. 323). Given this analysis, Table 1 and Figure 2 may be interpreted as follows: Stimulus duration and stimulus onset delay affected oculomotor latency and reaction time; the effects were greater for oculomotor latency than for reaction time. Figure 3 shows the oculomotor latency for each individual subject for the two most extreme durations, 100 and 300 msec. The data of five subjects show increasing divergence between the 100- and 300-msec curves as stimulus onset delay increases. The single exception (subject D.H.) is also the fastest subject in the group, with minimum oculomotor latencies of 200 msec; it is plausible that this subject was more affected by a physiological "floor" effect than the others.

Table 1
Mean Fixation Duration, Oculomotor Latency, and Reaction Time at Each Stimulus Onset Delay

Stimulus Onset Delay	Nontarget Stimuli (X)		Target Stimuli (O)
	Fixation Duration	Oculomotor Latency	Reaction Time
0	371	371	434
30	366	336	398
60	369	309	383
90	381	291	381
120	394	274	373
150	415	265	378

Note—The first fixation of each trial and all error trials have been excluded. All times are milliseconds.

Figure 2. Oculomotor latency and reaction time at each stimulus onset delay, for each stimulus duration used. The dashed line has a slope of -1 . The first fixation of each trial and all error trials have been excluded.

Because each number of nontarget stimuli appeared in an equal number of trials, the conditional probability of a target stimulus varied over the course of each trial; however, this did not appear to affect the overall appearance of the oculomotor latency function (Figure 4). Though the function was shifted downwards for fixations when the probability of a target stimulus was zero, all curves have about the same shape, regardless of the ordinal number of the fixation within a trial or the conditional probability of the target on that fixation.

DISCUSSION

The major effect observed is the steeper slope for oculomotor latency than for reaction time, as stimulus onset delay is increased. This result appears to rule out the possibility that the reaction time and oculomotor latency functions are determined by a common mechanism, either saccadic suppression or warning stimulus foreperiod effects.

Saccadic Suppression

Saccadic suppression influences ought to occur only in the earliest stages of processing a stimulus. These initial stages of processing (perception and encoding) are common to the fixations of both target

and nontarget stimuli. Saccadic suppression should, then, equally effect the perception of both target and nontarget stimuli. Thus, if the presentation of the stimulus is delayed until the time of suppression is completed, we should find quantitatively similar effects of the delay of stimulus onset for both oculomotor latency and reaction time. However, the effect of stimulus onset delay was larger for oculomotor latency than for reaction time, and extended (for oculomotor latency) far beyond the 30 to 40 msec after saccade onset during which saccadic suppression has typically been observed psychophysically (e.g., Volkman, Schick, & Riggs, 1968). Thus the steep slope of the oculomotor latency curve beyond the 30-msec delay cannot be attributed to saccadic suppression during a stage common to the determination of oculomotor latency and reaction time.

Foreperiod Effects

In the paradigm used here, the saccade serves to

Figure 3. Oculomotor latency for each subject, at the 100- and 300-msec stimulus durations.

Figure 4. Oculomotor latency and reaction time for stimuli appearing on the first through sixth fixations of each trial. The probability of a target after zero or one nontarget stimuli is 0.0; after 2, 3, 4, 5, or 6 nontargets, it is 0.20, 0.25, 0.33, 0.5, or 1.0, respectively.

signal the subject that the stimulus is about to be presented as well as to expose new information on the retina. Warning stimuli have been observed to decrease disjunctive reaction time in a number of situations, presumably by facilitating attentional or preparative processes. For instance, Bertelson and Tisseyre (1969) presented a warning click or flash 0 to 750 msec before a visual signal calling for a choice reaction. Both auditory and visual warning signals facilitated reaction time to the visual stimulus, as in the reaction time data of the present experiment. We might suppose, then, that the effect of stimulus onset delay on oculomotor latency is simply such a foreperiod effect where the saccade serves as a warning stimulus. However, the magnitude of the facilitation of oculomotor latency by stimulus onset delay in the present experiment is much larger than that observed for other reaction time tasks, which have involved finger movements with auditory or visual warning stimuli. In several

studies, the slope relating reaction time to the duration of the warning foreperiod ranges from -0.16 to -0.26 over a 0-to-150-msec range (Bertelson, 1967; Bertelson & Tisseyre, 1968, 1969; Davis & Green, 1969). Of particular interest is a study by Saslow (1967), who measured saccadic reaction time to a displaced target preceded by a visual warning stimulus. The slope of reaction time as a function of foreperiod duration was -0.22 over the 0-to-150-msec range. By comparison, the average slope of oculomotor latency in the present data over this range was over twice these values (about -0.60), with a slope of nearly -1 for stimulus onset delays of 60 msec or less. Thus, the effect observed on oculomotor latency is much greater than known warning stimulus effects.¹

The foreperiod effect on reaction time (that is, the shape of the reaction time function) appears to be independent of target stimulus probability, which increases as the trial progresses (Figure 4). Similarly, the shape of the oculomotor latency fixation is unchanged as nontarget probability decreases, though both oculomotor latency and reaction time tend to be faster as stimulus probability (nontarget or target, respectively) increases. Oculomotor latencies become longer as the probability of a nontarget stimulus decreases over the course of a trial, while reaction times decrease as the probability of a target increases. Nevertheless, the essential observation of the experiment (the near -1 slope of the oculomotor latency function at the shorter stimulus onset delays) is not changed by this expectation effect.

Preprogramming of Fixation Durations

In a visual search task such as this one, the subject might use a serial information processing strategy, with several stages executed one after the other. The subject would first perceive the stimulus presented, then encode it, and finally decide whether to produce a nontarget response or a target response. In such a model, the stages prior to response selection would be common to both target and nontarget fixations. Within this model, it is difficult to see how stimulus onset delay might have different effects on oculomotor latency and reaction time.

Alternatively, the subject might use a strategy in which the control of fixation duration occurs in parallel with, and independent of, the processes which identify the stimuli and initiate target reporting responses. In this parallel model, the function of the fixations in the search task is to present to the subject the visual stimuli necessary to perform on the task. This might be done without stimulus control of the duration of each fixation, unless the stimulus fixated required an extraordinarily long processing time. Suppose that in each trial of the search task the subject initiated a series of fixations of roughly

constant, predetermined duration. If this predetermined duration were greater than the maximum amount of time (including the stimulus onset delay, if any) required for processing a stimulus, the information processing requirements of the stimulus would have no effects on the duration of fixations, and the function relating the oculomotor latency to stimulus onset delay would have a slope of -1 . If, on the other hand, the predetermined duration of the fixations were less than that required to process the stimulus, then the oculomotor latency function would have a slope of zero. In this case, the next saccade could only be initiated when the information presented in the preceding fixation has been processed, regardless of the delay. By the foregoing analysis, the slope of the oculomotor latency curve ought to approach -1 if the fixation duration time exceeded the time required for processing (including delay), and approach 0 if the processing time were greater than the preprogrammed fixation duration. Now, both stimulus duration and stimulus onset delay varied, and this ought to affect processing time.

The time required for the processing of a stimulus ought to increase if the stimulus quality were impaired by decreasing its duration (if it is followed by a mask). Furthermore, the time available for processing in a fixation of constant duration would be less if the stimulus onset delay were greater. In either of these cases, it is more likely that the information processing requirement would exceed a predetermined fixation duration. Thus, we would predict that the slope of the function relating oculomotor latency and stimulus onset delay ought to be flatter when the stimulus duration was short, or the delay was long. Just these trends are evident in Figure 2. It remains to be seen whether other manipulations of the stimulus that ought to increase processing time will show similar effects on this function—for instance, stimulus degradation, or increase in the number of stimulus alternatives. Finally, the model proposed here depends on the subject's estimation of the time required for the processing of the stimulus. This estimation could be made more accurately in a blocked (constant-delay) design than in a variable-delay design like the present one, in which the delay was different for each trial. Whether this prediction is borne out will depend on further experimentation.

In summary, the relation between saccade-synchronized stimulus onset delay and the oculo-

motor patterns of subjects suggests that under some conditions the duration of fixations in search may be independent of the visual information presented in each of those fixations.

REFERENCE NOTE

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NOTE

1. A control experiment (Graefe & Vaughan, Note 1) failed to detect any difference between finger and oculomotor responses in a reaction-time paradigm, observing a slope of about -0.25 over the 0-to-150-msec range in both cases. Thus, while a warning-stimulus effect might account for the reaction-time data of the present experiment, it does not plausibly account for the difference between reaction time and oculomotor latency.

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