Target Size Modulates Saccadic Eye Movements in Humans

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The authors investigated mechanisms involved in transformation of spatially extended targets into saccadic eye-movement vectors. Human subjects performed horizontal saccades to targets of varying diameter, which contained no conspicuous elements within the target shape. With increasing target size, express saccades and saccades with fast regular latencies decreased in frequency, whereas frequency of saccades with slow regular latencies increased. For all targets, saccade amplitude distributions showed a peak close to the geometric center of the targets. However, with large targets, increased scatter of saccade amplitudes and increased undershoot of the target center was observed. These effects may reflect distinct subprocesses involved in sensorimotor transformation to spatially extended targets, and may result from modulation of neuronal activity in the superior colliculus.

Saccadic eye movements bring images of objects of interest onto the fovea. Under natural conditions, targets for saccades are mostly spatially extended objects. The saccadic system must therefore transform complex spatial information into a single eye-movement vector. Little is known about how the properties of perceived spatial information modulate this transformation process. In particular, it is unclear how target size is processed into a distinct motor program and whether this transformation process affects latencies and metrics of saccadic eye movements. A further open question is where in the brain these sensorimotor transformations occur. In the saccadic system, multiple areas are candidate regions for such computations. On the cortical level, the posterior parietal cortex (PPC) and the frontal eye fields (FEF) show both stimulus- and saccade-related activity (Andersen, Snyder, Bradley, & Xing, 1997; Schall & Thompson, 1999). In the midbrain, the superior colliculus (SC) processes visual stimuli and is a key structure for the preparation of saccades (Sparks & Hartwich-Young, 1989). It has not yet been studied whether target size modulates neuronal activity in these regions. However, studies have investigated whether the SC is involved in the representation of multiple saccade targets under conditions in which a single saccade is performed to the average of their locations (Edelman & Keller, 1998; Glimcher & Sparks, 1993; Van Opstal & Van Gisbergen, 1990). The results of these studies have been consistent with sensorimotor transformations upstream (Glimcher & Sparks, 1993), within (Van Opstal & Van Gisbergen, 1990), and downstream (Edelman & Keller, 1998) of saccade-related neurons in the SC. It can therefore be concluded that the transformation of complex visual information into a saccade vector is not necessarily always complete at the level of the cortical eye fields.

In the present study, we examined the relationship between target size and saccadic eye movements. Healthy human subjects were requested to quickly and accurately perform saccades to targets of varying diameter. We investigated whether target size affects latencies and metrics of saccades. We aimed to infer, from possible size-related changes of saccade parameters, the properties of the sensorimotor transformation process preceding saccades to spatially extended targets.

Method

Subjects

A group of 12 healthy human subjects (3 women and 9 men) with a mean age of 27.2 ± 0.9 years participated in the experiment. None of the subjects had a history of neurological or psychiatric disorders. All subjects had normal vision. Informed consent was obtained from all subjects before their participation in the study, which was approved by the local ethics committee and conducted in conformity with the Declaration of Helsinki (World Medical Association, 2002).

Stimulus Presentation

Stimuli were generated with Experimental Run Time System-Software, Version 3.32 (ERTS; Berisoft, Frankfurt/Main, Germany). Stimuli were presented on a computer monitor, with the screen subtending 40° horizontally and 30° vertically, at a distance of 50 cm from the subject’s eyes. Stimuli had a luminance of 2 cd/m² seen against a homogeneous gray background. Except for the illumination from the screen, subjects were seated in complete darkness.

Paradigm

At the beginning of a trial, subjects fixated on a small red cross (1° of visual angle) for either 2,000, 2,500, 3,000, 3,500 or 4,000 ms. Then, the fixation cross was switched off and a stimulus was presented for 50 ms in a horizontal position at either 5.0, 7.5, 10.0, 12.5, or 15.0° eccentricity, in either the right or left visual hemifield. A brief stimulus presentation was used to avoid visual feedback after saccade execution and reduce learning of stimulus positions. Stimuli were round red disks, subtending either 1.5, or 10° of visual angle. After 1,000 ms, the fixation cross was reilluminated, and the next trial began. Stimulus size, stimulus position, and duration of fixation were varied pseudorandomly across trials. Each stimulus size was presented 50 times, balanced across eccentricities. Thus, a total of 150 trials was performed by each subject. Stimuli were presented in two blocks.
of 75 trials, separated by a break of 10 min to avoid fatigue. Subjects were instructed to look quickly and accurately at the stimulus as soon as it appeared. No further instructions were given.

**Eye-Movement Recordings**

Eye movements were recorded by horizontal infrared oculography of the right eye (AMTech Eyetracker, Weinheim, Germany). Data were sampled at a frequency of 200 Hz. The system had a spatial resolution of 0.3° and a horizontal linear range of more than 20° bilaterally. The subject’s head was fixed to the recording system by means of a bite-bar with individual dental impressions. Calibration trials with two lateral targets, presented horizontally at 15° eccentricity, were regularly performed during recording sessions.

**Data Analysis**

Data were analyzed offline with EYEMAP-software (AMTech). Latencies and amplitudes of saccades were measured. Metrics of saccades were expressed as gain, that is, the ratio of saccade amplitude to target eccentricity, defined as the geometric center of the stimuli. Thus, a gain of 1 indicates a precise saccade to the target center; a gain > 1, hypermetria; and a gain < 1, hypometria, with respect to the stimulus center. Statistics were done with reference to Altman (1991). For each subject, medians were used to describe systematic targeting errors (hypo- or hypermetria of saccade endpoints) and interquartile ranges were used to describe variable targeting errors of saccades (scatter of saccade endpoints; Ploner, Gaymard, Rivaud, Agid, & Pierrot-Deseilligny, 1998; Ploner, Rivaud-Péchoux, Gaymard, Agid, & Pierrot-Deseilligny, 1999). Friedman’s analysis of variance and two-tailed Wilcoxon’s signed-rank tests were used for statistical analysis. Spearman’s correlation coefficients were used for correlation analysis.

**Results**

For saccade latencies, analysis of variance revealed clear differences between stimuli, with average saccade latencies increasing with stimulus size, \( \chi^2(2, N = 12) = 9.22, p = .01 \) (see Figure 1). Next, we plotted pooled latencies of saccades from all subjects as histograms separately for each of the three stimuli (Figure 2). In all histograms, a trimodal distribution of saccade latencies was apparent, with a first peak occurring at latencies of approximately 120 ms, a second peak at 160–170 ms, and a third peak at approximately 250 ms. The first peak and the second peak were separated by a gap at latencies of about 140 ms, that is, at the transition between the “express” and “fast regular” modes of

![Figure 1. Effects of target size on group mean (± SEM) saccade latencies. *** p = .004, significantly different from 1° stimuli.](image)

![Figure 2. Effects of target size on saccade latencies, pooled data from 12 subjects. Histograms show latency distributions separately for 1° stimuli (A), 5° stimuli (B), and 10° stimuli (C). Dotted line at 140 ms indicates transition between “express” and “fast regular” latencies. Dotted line at 200 ms indicates transition between fast regular and slow regular latencies. Percentages indicate frequencies of express, fast regular, and slow regular saccades. Fit curves were calculated for each histogram assuming three superposed Gaussian latency distributions. Note that the height of peaks changes with stimulus size, whereas the position of peaks remains fairly constant.](image)
saccade latencies (Becker, 1989; Fischer et al., 1993; Gezeck, Fischer, & Timmer, 1997). The second and the third peak were separated by a gap at 190–200 ms, that is, at the transition between the fast regular and slow regular mode of saccade latencies (Becker, 1989; Fischer et al., 1993; Gezeck, Fischer, & Timmer, 1997). The position of the three peaks was similar for all three stimuli (Figure 2). However, with increasing stimulus size, the express peak and fast regular peak decreased in height, whereas the peak for slow regular latencies increased (Figure 2). Thus, the significant increase in saccade latencies appeared to be due to a lower frequency of saccades with latencies < 200 ms to larger stimuli, that is, express saccades and fast regular saccades, rather than to a shift of the entire latency distribution toward longer latencies. This effect was not due to a limited subset of subjects performing saccades with latencies < 200 ms, as there also was a significant effect of stimulus size on individual frequency of these saccades, χ²(2, N = 12) = 13.50, p = .001. In addition, compared to 1° stimuli, the individual frequencies of both express saccades and fast regular saccades to 10° stimuli were significantly decreased (p = .008 and p = .005). Next, we calculated the frequency of saccades with latencies < 200 ms from the pooled latency data separately for each stimulus size and eccentricity. The plotted values (see Figure 3) show that differences in frequencies of these saccades occurred mainly with eccentricities of up to 10°. However, these latency effects were obviously not due to an overlap of 10° targets at 5° eccentricity with the fixation point, as they were also observed with larger eccentricities and also occurred with 5° targets. Furthermore, these effects are unlikely to be due to different distances between fixation point and medial stimulus margins, as the frequency of saccades with latencies < 200 ms to 1° targets at 5° eccentricity was considerably higher than the frequency of these saccades to 10° targets at 10° eccentricity.

For both parameters of saccade metrics—average gain and gain variability—analysis of variance showed a significant effect of stimulus size, χ²(2, N = 12) = 10.67, p = .005, and χ²(2, N = 12) = 17.9, p = .0001 (see Figure 4). Average gain decreased slightly but significantly with increasing stimulus size, that is, saccades to large targets were more hypometric with respect to the stimulus center than saccades to small targets. At the same time, gain variability increased significantly with stimulus size, that is, saccades to large targets showed an increase in scatter around their average landing position. Although gain variability for 10° stimuli was high (M = 0.26 compared to 0.14 for 1° stimuli, Figure 4), its 95% confidence interval (0.21–0.31) did not include 0.5. This value would have been expected if saccades had been targeted to random positions within the 10° stimuli. It can therefore be concluded that even for targets with large diameters, a distinct landing position close to the geometric center of the target is computed, albeit with lower precision than for small stimuli. This is also evident from the pooled gain values from all subjects, plotted separately for each stimulus size (Figure 5). Inspection of the histograms shows that for 5° and 10° stimuli the distribution of saccade gains remained unimodal, with a peak close to 1. Saccades were obviously not directed to the inner or outer margins of these stimuli. No correlation was found between average gain and gain variability, calculated from a subject’s pooled gain values, and individual frequency of saccades with latencies < 200 ms (p = .10 and p = .41).

Figure 3. Eccentricity-dependent frequency of saccades with latencies < 200 ms, that is, express saccades and fast regular saccades. Percentages were calculated from pooled latency data from 12 subjects.

Figure 4. Effects of target size on average gain (A) and gain variability (B) of saccades. Data are group means (± SEM). ** p = .005, *** p = .002, significantly different from 1° stimuli.
Discussion

The findings from our experiment show that target size affects latencies and metrics of saccadic eye movements. Frequency distributions of saccade latencies changed with stimulus size, leading to an increase in average latencies for larger targets. At the same time, scatter of saccade endpoints increased and saccades became more hypometric with larger targets. In the following, we discuss how these findings relate to previous studies of oculomotor control and to the sensorimotor transformation process preceding saccades to spatially extended targets.

The observed latency effects differ from those seen in previous studies, in which no effects of target size on latencies of saccades were found (Kowler & Blaser, 1995; McGowan, Kowler, Sharma, & Chubb, 1998). However, in those studies, subject instructions clearly emphasized accuracy over reaction time. In the present study, no priority was given to either accuracy or reaction time. Our results show that under such conditions, reaction time is significantly modulated by target size. Because even saccades to 10° stimuli were mostly targeted to the stimulus center, target size was clearly taken into account in our paradigm. Hence, an increasingly complex spatial pooling or selection process may have determined a target center from an increasing number of stimulus-activated neurons before saccades were executed (McGowan et al., 1998). However, if such mechanisms had been responsible for the observed size-dependent latency effects, we would have expected that an increasing amount of processing time was added before execution of saccades to 5° or 10° stimuli (Schall & Thompson, 1999). A shift of the entire latency distribution toward longer latencies would have resulted, which was not the case in our experiment, in which changes in average saccade latencies resulted from a discrete process (i.e., changes in the frequency distribution of saccade latencies).

It has been shown previously that frequency distributions of saccade latencies critically depend on neuronal activity in the SC (Schiller, Sandell, & Maunsell, 1987; Schiller & Lee, 1994). After lesion or deactivation of the SC in monkeys, express saccades are no longer obtained, and saccades with regular latencies occur at significantly longer latencies (Schiller et al., 1987). Hence, both types of saccade latencies appear to be controlled by the SC (Schiller et al., 1987). Recent single-neuron studies have suggested that the distribution of saccadic reaction times may result from the distribution of neural activity of saccade-related neurons in the SC (Dorris, Paré, & Munoz, 1997; Edelman & Keller, 1996; Sparks, Rohrer, & Zhang, 2000). These neurons are reciprocally connected with fixation neurons in the SC, whose deactivation has been shown to increase the frequency of express saccades (Munoz & Wurtz, 1992, 1993a, 1993b). The target size-dependent changes in frequency of express saccades and fast regular saccades in our experiment may thus indicate higher activation of fixation neurons and lower activation of saccade-related neurons in trials with large saccade targets compared to trials with small targets. As stimuli were randomized and appearance of any of the three stimulus types was equally probable at any stimulus position, motor preparation or target uncertainty do not explain a possible differential modulation of these neurons (Basso & Wurtz, 1997, 1998; Dorris et al., 1997; Edelman & Keller 1996). It is thus likely that target size itself modulated activity of fixation and/or saccade-related neurons in the SC. The spatial distribution of size-related differences be-

Figure 5. Effects of target size on metrics of saccades, pooled data from 12 subjects. Histograms show gain distributions separately for 1° stimuli (A), 5° stimuli (B), and 10° stimuli (C). Dotted line indicates perfect precision, (i.e., gain = 1). Fit curves were calculated for each histogram assuming Gaussian gain distributions.
tween frequencies of saccades with express and fast regular latencies lends further support to this hypothesis, as they were particularly observed with eccentricities corresponding to the distribution of fixation cells in the SC (Gandhi & Keller, 1997, 1999). These neurons occur mainly in rostral regions of the SC, where saccades with amplitudes of up to 10° are represented (Gandhi & Keller, 1997, 1999). Our results are therefore in keeping with previous behavioral research postulating a “fixation zone” extending up to 10° from the actual fixation point (Walker, Deubel, Schneider, & Findlay, 1997).

The peaked distribution of saccade amplitudes in our experiment confirms that the saccadic system has access to a reference position within spatially extended targets (Kowler & Blaser, 1995; McGowan et al., 1998). The observed changes in metrics of saccades nevertheless indicate that the associated transformation of a spatially extended stimulus into a saccade vector becomes increasingly noisy and erratic with larger saccade targets. These changes in metrics appear to reflect a stochastic process associated with target size and are therefore distinct from the discrete changes in latency distributions discussed above. The lack of correlation between both measures of saccade metrics and frequency of express saccades further shows that these effects are not directly related. Similar effects have been observed with saccades to two simultaneously presented targets, where saccades tended to land at an intermediate position between both stimuli (Findlay, 1982; Ottes, Van Gisbergen, & Eggermont, 1984). As in our experiment, averaging saccades in these studies showed both an increase in amplitude variability and a tendency to undershoot the exact average position of both targets (Findlay, 1982; Ottes et al., 1984). Furthermore, it has been shown that increasing the spatial separation between targets in double-target experiments decreases the frequency of express saccades, as observed with our spatially extended stimuli (Chou, Sommer, & Schiller, 1999). It appears possible, therefore, that the neural mechanisms involved in computation of saccade endpoints to spatially extended targets are similar to those involved in computing averaging saccades in double-target experiments. The fact that these averaging responses are facilitated when speed is stressed in instructions (Findlay 1982; Ottes et al., 1984) may also explain the discrepancy between our results and those of previous studies with saccades to spatially extended targets, in which saccade accuracy was given priority in instructions and metrics of saccades were found to be largely independent of target size (Kowler & Blaser, 1995; McGowan et al., 1998).

Several neurophysiological studies have investigated a possible role of neurons in the SC for transformation of complex visual information into saccade vectors. With double-target experiments, some authors found that averaging saccades are coded in saccade-related SC neurons similarly to saccades to single targets (Glimcher & Sparks, 1993). Others found that, at least under certain behavioral conditions, SC neurons may be involved in saccade averaging (Van Opstal & Van Gisbergen, 1990), or even represent the two saccade targets (Edelman & Keller, 1998). In this latter case, saccade averaging may occur in regions downstream of the SC. We believe that the latency effects in our experiment, in which saccade targets were not defined by conspicuous elements within the stimulus shape, support the hypothesis that the SC is involved in representation of the saccade target in its spatial extent rather than receiving an integrated pointlike representation of the stimulus center from areas upstream of the SC. This suggests that the SC or downstream areas may be involved in subsequent computation of corresponding saccade vectors. In keeping with this hypothesis, previous research has related variability of saccade endpoints to noisy variations in the population activity of saccade-related neurons in the SC (Van Opstal & Van Gisbergen, 1989). These neurons constitute a nonlinear motor map in which neurons coding small saccade amplitudes are more abundant than neurons coding large saccade amplitudes, and in which executed saccade amplitudes are determined by weighted averaging of the activity of a population of neurons coding adjacent amplitudes (Lee, Rohrer, & Sparks, 1988; Sparks & Hartwich-Young, 1989). The increasing hypometria of saccade endpoints with spatially extended targets suggests that the average of this population is increasingly biased toward small amplitudes with increasing stimulus size. Hence, with increasing spatial extent of saccade targets, nonlinear synaptic weighting of activated SC neurons (Lee et al., 1988) appears to compensate less efficiently for the nonlinearity of the saccade motor map. At the same time, neuronal activity surrounding the population average may show a lesser decrease with increasing stimulus size, leading to less sharply tuned neuronal populations with larger saccade targets. Properties of the SC motor map could thus, at least partially, account for the target-size effects on saccade metrics observed in our study. However, this inference is based mainly on the observed latency effects and may theoretically also apply to upstream regions like the FEF, for which a comparable mapping of saccade amplitudes has not yet been performed. Hence, a verification of our hypothesis would require direct recordings of saccade-related SC neurons during saccades to spatially extended targets.

In summary, the findings from the present study provide a behavioral correlate of the sensorimotor transformation process preceding saccades to spatially extended targets. Size-dependent changes in latencies and metrics of saccades appear to reflect distinct size-related processes, with the latter resulting from a low-level averaging process that may also be involved in double-target experiments. This process may complement more elaborate processes of target selection under behavioral conditions in which saccade targets are not precisely specified by salient stimulus features.

References

monkey superior colliculus related to the initiation of saccadic eye movements. Journal of Neuroscience, 17, 8566–8579.


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