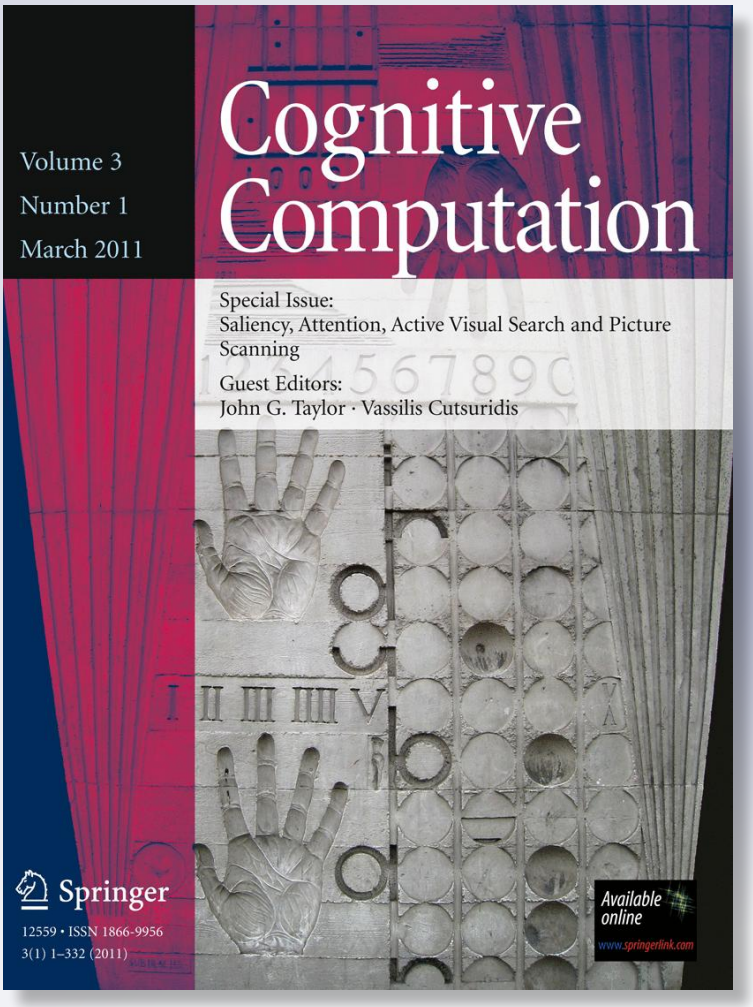
Insights into the Function and Mechanism of Saccadic Decision Making From Targets Scaled By an Estimate of the Cortical Magnification Factor

Cognitive Computation

ISSN 1866-9956
Volume 3
Number 1

Cogn Comput (2011) 3:89-93
DOI 10.1007/s12559-010-9075-y



Springer

Cogn Comput (2011) 3:89–93
DOI 10.1007/s12559-010-9075-y

Insights into the Function and Mechanism of Saccadic Decision Making From Targets Scaled By an Estimate of the Cortical Magnification Factor

David J. Yates · Tom Stafford

Received: 30 April 2010 / Accepted: 6 October 2010 / Published online: 26 October 2010

Abstract Here we address the shape of the saccadic latency–eccentricity function. Previously it has been proposed that the bowl-shaped nature of this function is a by-product of diminished stimulus representation in the periphery. A direct prediction of this theory is that saccadic latencies in the periphery should be speeded if stimuli are increased in size in proportion to the cortical magnification factor (*M*-scaling). Using a target-elicited saccade paradigm, ten subjects were shown *M*–scaled and unscaled Gaussian targets over a horizontal range of ± 40°. Saccadic latencies increased at an equal rate for peripheral targets regardless of whether targets were *M*–scaled or not. This suggests that the changes of latency with eccentricity are not a by-product of resources devoted to stimulus representation, but instead are a functional adaptation which takes account of the likelihood of saccades of each amplitude in the natural environment.

Keywords Saccadic latency · Eccentricity · Cortical magnification factor · Saliency

Introduction

There is a distinctive relationship between target eccentricity and saccadic latency [1, 2, 3, 4, 5, 6, 7, 8]. There is a general consensus within the literature that this function is bowl-shaped: there is a sharp increase in latency to targets very close to fixation ($<\approx 1°$) and a gradual increase in latency to targets in the periphery ($>\approx 20°$) (although see [8, 9, 10] for exceptions). For example, Kalesnykas and Hallett [4] found that subjects took approximately 50 ms longer to respond to a point-target at 40° compared to the same target at around 20°.

What is the origin of this distinctive latency–eccentricity function? It seems unlikely that intrinsic differences in stimulus conduction times between the retina and the lateral geniculate nucleus (LGN) are responsible for the changes in saccadic latency with eccentricity. If anything, conduction times for peripheral afferent signals are faster than for central afferent signals [11, 12].

One proposal has taken inspiration from the observation that more salient targets can reduce saccadic latency, e.g. those with greater contrast [13, 14] or luminance [4, 14, 15, 16]. Bell and colleagues have recently provided evidence that the saccadic latency–intensity relationship is due to different processing times along the visual pathway, which means that a high intensity stimulus reaches the superior colliculus faster than a low intensity stimulus [17].

Given that stimulus salience can affect saccadic latency, it has been suggested that the increase in saccadic latency with increasing target eccentricity is due to a reduction in target salience in the visual periphery [18, 19]. Consistent physiological differences between the fovea and periphery mean that a stimulus further away from fixation has less 'neural hardware' devoted to processing it: the density of cones reduces, the ratio of photoreceptors to ganglion cells increases past 10° and the average receptive field size increases [20]. Furthermore, when the signal reaches the LGN, the central representation is preferentially magnified on route to the visual cortex [20]. This change in scale of the retinotopic map from retina to cortex is known as the 'cortical magnification factor'.

A quantification of the cortical magnification factor, based on some or all of these physiological differences, is

D. J. Yates · T. Stafford (✉)
Department of Psychology, University of Sheffield,
Western Bank, Sheffield S10 2TP, UK
e-mail: t.stafford@sheffield.ac.uk

known as *M*–scaling. For example, Rovamo and Virsu [21, 22] estimated the value of *M* for the four principle meridians of the visual field from the density-distribution of retinal ganglion cells and central most cones. These *M* values can be used to adjust the size of stimuli according to their eccentricity, and so excite roughly the same amount of cortex. When this is done, many of the eccentricity-related decrements disappear (for a review see [23, 24]). For example, *M*–scaling sinusoidal gratings removes differences found in contrast threshold [22] and the decrement in visual search performance is neutralised when the search display is *M*–scaled [25].

If *M*–scaling can neutralise the speed related eccentricity-effect in visual search tasks, it is reasonable to suppose that it might also neutralise the peripheral eccentricity-effect on saccadic latencies. If *M*–scaling of targets flattens the latency–eccentricity function then this confirms the hypothesis that its curve, for unscaled targets, is due to a diminished representation of targets in the periphery. If, however, *M*–scaling of targets does not alter the shape of the latency–eccentricity function then an alternate explanation for the changes in latency with eccentricity must be sought.

Methods

Participants

Ten subjects aged between 18 and 29 ($M = 20.4$ years, $SD = 3.7$ years) participated in the experiment (7 Female, 3 Male). Eight of the subjects took part in return for course-credit towards their undergraduate Psychology degrees. All subjects had normal or corrected-to-normal vision and were naïve as to the purpose of the experiment. The subjects all gave their informed consent to take part in the experiment and the procedures were in accordance with the ethical standards of the Department of Psychology Ethics Sub-Committee and British Psychological Society Guidelines.

Apparatus and Stimuli

The centre of the left pupil of the subject was tracked at a sample rate of 60 Hz using a head mounted, infra-red video-based eye tracker (ISCAN RK-500). Stimuli were presented on a custom-built immersive dome, which wraps around the subject at a distance of 150 cm from the subject's head, and allows images to be projected over a horizontal range of 240° and a vertical range of 100°. Head movements were restricted during the experiment using a custom-built, height adjustable chin rest.

The fixation point was a green cross (+) of diameter 2.56°, which was located straight ahead of the subject at 0° horizontal and 0° vertical. All target stimuli consisted of white Gaussian targets (see inset of Fig. 1) with a peak luminance at their centre of 6 cd/m^2. Gaussian targets were used to avoid participants fixating on stimulus boundaries. The target stimuli were presented along the horizontal axis only at 5° intervals from −40° (40° left of fixation) to +40° (40° right of fixation) giving a total of 16 eccentricities. All stimuli were presented on a black background (0.3 cd/m^2) and luminance was kept constant across all eccentricities.

Design and Procedure

There were two experimental conditions. In Condition 1 (unscaled) all the targets were 2.56° in diameter. In Condition 2 (*M*–scaled) the targets were scaled in proportion to the *M* values obtained by averaging the following equations for the nasal (M_N) and temporal (M_T) visual fields:

$$M_N = (1 + 0.33E + 0.00007E^3)^{-1} M_0 \quad (1)$$

and

$$M_T = (1 + 0.29E + 0.000012E^3)^{-1} M_0 \quad (2)$$

Here, E refers to the eccentricity in degrees and M_0 is the magnification value (7.99 mm/°) for the central fovea [21, 22]. A graphical representation of the *M*–scaling used in this experiment can be seen in Fig. 1.

The dark-adapted subjects undertook a target-elicited saccade paradigm. After a variable period of fixation (2000–4000 ms) the fixation point was extinguished and a target would appear immediately at one of the 16 target locations. Subjects were instructed to move their eyes as quickly and as accurately as they could to the centre of the target. The two conditions were interleaved and the order

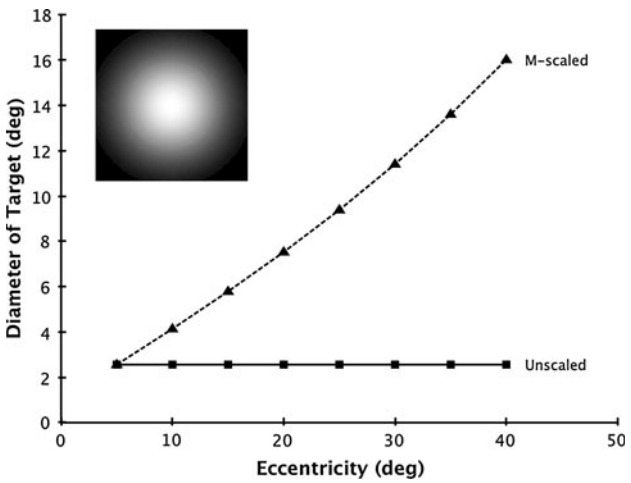


Fig. 1 The size of targets used in Condition1 (*unscaled*) and Condition 2 (*M-scaled*) and the Gaussian target (*inset*)

of presentation was randomised for each subject, meaning that the targets were both temporally and spatially unpredictable. The subjects undertook 7 cycles of the 32 target size–eccentricity combinations, and were given the opportunity to rest between each cycle. The first cycle of each subject was used as a practice run and was discarded.

Measures

Initial estimates of the onset and offset of the primary saccade were first found using a velocity threshold algorithm. These values were then displayed on the horizontal eye movement trace, and each point was checked for accuracy by eye. A total of 91 (4.73%) saccadic onsets could not be located due to the subject blinking or the onset of the saccade not being clear due to erratic eye movements. The mean saccadic latency was then calculated for each eccentricity, and any latencies above or below three times the standard deviation were classed as outliers and removed. This resulted in 22 extra data points being removed for a total of 113 (5.89%).

The amplitude of the primary saccade was also calculated to check if there were any differences in saccadic amplitudes between the two conditions.

Results

Saccadic Latency

Figure 2 shows the mean latencies of the primary saccades as a function of eccentricity, for unscaled and *M*–scaled targets. A repeated measures ANOVA was conducted with three factors of *condition* (unscaled and *M*–scaled), *direction* (left and right) and *eccentricity* (eight eccentricities from 5° to 40°). The only significant effect was that of eccentricity, $F(7, 63) = 17.94$, $P < 0.001$.

Saccadic Amplitude

Figure 3 shows the mean amplitudes of the primary saccades as a function of eccentricity, for unscaled and *M*–scaled targets. There is a statistically significant ($t(9) = 3.85$, $P < 0.01$) difference in saccadic amplitude between the unscaled and *M*–scaled targets. Although it is clear that subjects made slightly shorter saccades when the targets were *M*–scaled, this difference is small: on average, saccades to *M*–scaled targets were 96.34% of the magnitude of saccades to unscaled targets. It is important to note that although this difference exists, its size suggests that, given the normal saccadic latency–eccentricity function, it is not an important factor in determining our results. Any effect would only be to increase the power of our analyses to reveal a speeding of saccadic latency in the *M*–scaled condition. As discussed above, this was not seen.

Discussion

The experiment was successful in replicating the bowl-shaped function using spatially extended Gaussian targets. It is also clear that saccadic latencies increased at an equal rate, regardless of whether the targets were *M*–scaled or not (Fig. 2). This suggests that the peripheral increases in saccadic latency cannot be counteracted by increasing the spatial representation of the targets. It would seem that for saccadic latencies, stimulating more neurons does not equate to more salient, in the same way that increasing the luminance of the target does [4, 14, 15, 16].

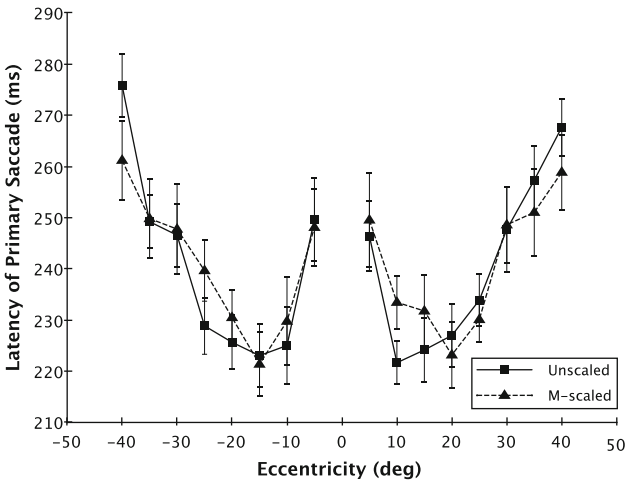


Fig. 2 The latency of the primary saccade as a function of eccentricity, for unscaled and *M*-scaled targets. *Points* and *error bars* represent the group means ± SEM of 10 subjects

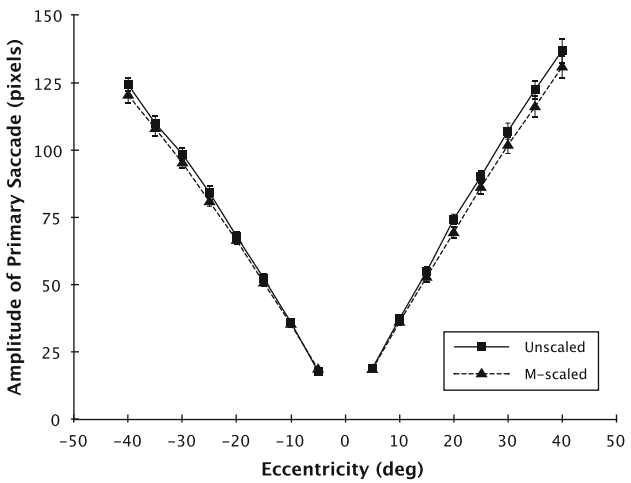


Fig. 3 The amplitude of the primary saccade as a function of eccentricity, for unscaled and *M*-scaled targets. *Points* and *error bars* represent the group means ± SEM of 10 subjects

There is a potential confound in our results, however. Most studies of saccadic eye movements use point-like saccade targets where the target itself designates the saccade endpoint. In this study, spatially extended targets were used, where an additional computation is required to locate the saccade endpoint within the target [26, 27]. If target size has a significant impact on saccadic latency then this could have influenced the results in the *M*–scaled condition where eccentricity *and* target size were manipulated.

There have been two studies which both suggest that target size does not play a significant role in saccadic latency. Firstly, Kowler and Blaser [27] found that saccadic accuracy, precision and latency did not vary as a function of target size (up to 4° in diameter). In the second study, target size *and* eccentricity were manipulated [28]. This study found that target size only affected saccadic latency when the targets were presented at low eccentricities (<7.5°). These findings suggest that target size only plays a significant role when the target is large enough to activate the 'fixation zone' of the superior colliculus, which has been estimated to extend 10° around the fixation point [29]. This finding could also explain why we found a central latency peak at 5°: because the targets there were 2.56° in diameter, they were large enough to activate the fixation zone and so slow saccadic latencies.

Our findings suggest that the saccadic latency–eccentricity function is not a by-product of diminished peripheral representation, but a central and robust feature of the saccadic system, which has arisen during evolution due to the different frequency of saccades of different amplitudes. Tatler and Vincent [30] provide crucial evidence in favour of this conjecture. They recorded eye movements during free-viewing of colour photographs of natural scenes and so derived a saccadic amplitude–frequency function. Figure 4 shows their findings for horizontal (black) and vertical (grey) saccades.

Comparing with our results (Fig. 2), readers will immediately note the superficial similarity between the two functions: the latency–eccentricity function looks like the inverse of the amplitude–frequency function. There are similarities of at least two non-trivial features, which suggest that the shape of one may be core in determining the shape of the other. Firstly, they both share a non-monotonicity, which is unusual for psychometric functions. Note also that the cortical magnification factor is monotonic, which means it is unable on its own to explain both the central and peripheral rises in saccadic latency. Secondly, in line with earlier estimates [31], the vast majority of naturally occurring saccades (around 86% according to [31]) have an amplitude of 15° or less. This figure is in broad agreement with the stationary point of the saccadic latency–eccentricity function, which lies in the region of 1–15° [1–7]. This suggests that we are fastest to make the saccades we make most often.

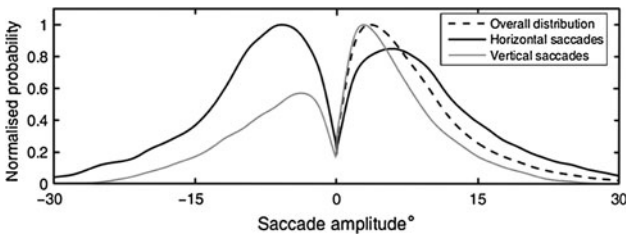


Fig. 4 The amplitude–frequency distribution of saccades. Reprinted from [30]

If it is true that the latency–eccentricity function is derived from the amplitude–frequency function, then Tatler and Vincent's results allow for the prediction of saccadic latency for targets of different *vertical* eccentricities. Of particular interest here is the asymmetry in frequency of vertical saccades, with the distribution of positive (i.e. upward) saccades being far more peaked than for negative (i.e. downward) saccades. We predict, therefore, that the vertical latency–eccentricity function will be a similar inverse of the amplitude–frequency function, and that upward saccades will be quicker than downward ones for the mid-range. There is already some evidence for this latter point: a number of studies have found that upwards saccadic latencies are significantly faster than downward saccadic latencies [32, 33]. The full vertical saccadic latency–eccentricity function, however, remains to be determined.

Conclusion and Summary

The experiment presented here tests and falsifies an explanation of the shape of the saccadic latency–eccentricity function, which is based on the idea that it is driven by diminished stimulus representation in the periphery. Instead we find that saccadic latency is unaffected by target size and so follows the same distinctive non-monotonic function with eccentricity for consistently sized and *M*–scaled targets. We suggest that this latency–eccentricity function is embedded architecturally in the circuitry controlling eye movements as a result of the natural history of eye movements, particularly the amplitude–frequency function as revealed by [30].

Acknowledgments Thanks to Jim Austin at the University of York, Kevin Gurney, Jon Chambers, Alex Cope. Martin Thirkettle and Mahmood Javed at the University of Sheffield, Jeff Ferguson at Inition, Nicol Harper at the Redwood Center for Theoretical Neuroscience, University of California and Benjamin Tatler at the University of Dundee for advice during the setup of our eye tracking facilities. Thanks also to the two anonymous reviewers of this paper for their

comments. David Yates is funded by the White Rose University Consortium.

References

1. Bartz AE. Eye-movement latency, duration, and response time as a function of angular displacement. J Exp Psychol. 1962;64(3):318–24.
2. Biguer B, Prablanc C, Jeannerod M. The contribution of coordinated eye and head movements in hand pointing accuracy. Exp Brain Res. 1984;55(3):462–9.
3. Findlay JM. Visual information processing for saccadic eye movements. In: Hein A, Jeannerod M, editors. Spatially oriented behavior. New York: Springer; 1983. p. 281–303.
4. Kalesnykas RP, Hallett PE. Retinal eccentricity and the latency of eye saccades. Vis Res. 1994;34(4):517–31.
5. White CT, Eason RG, Bartlett NR. Latency and duration of eye movements in the horizontal plane. J Opt Soc Am. 1962;52:210–3.
6. Wyman D, Steinman RM. Letter: latency characteristics of small saccades. Vis Res. 1973;13(11):2173–5.
7. Zambarbieri D, Beltrami G, Versino M. Saccade latency toward auditory targets depends on the relative position of the sound source with respect to the eyes. Vis Res. 1995;35(23–24):3305–12.
8. Baloh RW, Honrubia V. Reaction time and accuracy of the saccadic eye movements of normal subjects in a moving-target task. Aviat Space Environ Med. 1976;47:1165–7.
9. Darrien JH, Herd K, Starling LJ, Rosenberg JR, Morrison JD. An analysis of the dependence of saccadic latency on target position and target characteristics in human subjects. BMC Neurosci. 2001;2(13):1–8.
10. Frost D, Pöppel E. Different programming modes of human saccadic eye movements as a function of stimulus eccentricity: indications of a functional subdivision of the visual field. Biol Cybern. 1976;23(1):39–48.
11. Ogden TE, Miller RF. Studies of the optic nerve of the rhesus monkey: nerve fiber spectrum and physiological properties. Vis Res. 1966;6(9):485–506.
12. Carrasco M, McElree B, Denisova K, Giordano AM. Speed of visual processing increases with eccentricity. Nat Neurosci. 2003;6(7):699–70.
13. Ludwig CJH, Gilchrist ID, McSorley E. The influence of spatial frequency and contrast on saccade latencies. Vis Res. 2004;44(22):2597–604.
14. Doma H, Hallett PE. Rod-cone dependence of saccadic eye-movement latency in a foveating task. Vis Res. 1988;28(8):899–913.
15. Doma H, Hallett PE. Dependence of saccadic eye-movements on stimulus luminance, and an effect of task. Vis Res. 1988;28(8):915–24.
16. Wheeless LL, Cohen GH, Boynton RM. Luminance as a parameter of the eye-movement control system. J Opt Soc Am. 1967;57(3):394–6.
17. Bell AH, Meredith MA, van Opstal AJ, Munoz DP. Stimulus intensity modifies saccadic reaction time and visual response latency in the superior colliculus. Exp Brain Res. 2006;174(1):53–9.
18. Chambers JM. Deciding where to look: a study of action selection in the oculomotor system. PhD thesis. University of Sheffield, Sheffield; 2006.
19. Bell AH, Everling S, Munoz DP. Influence of stimulus eccentricity and direction on characteristics of pro- and antisaccades in non-human primates. J Neurophysiol. 2000;84(5):2595–604.
20. DeValois RL, DeValois KK. Spatial vision. New York: Oxford University; 1988.
21. Rovamo J, Virsu V. An estimation and application of the human cortical magnification factor. Exp Brain Res. 1979;37(3):495–510.
22. Virsu V, Rovamo J. Visual resolution, contrast sensitivity, and the cortical magnification factor. Exp Brain Res. 1979;37(3):475–94.
23. Kitterle FL. Psychophysics of lateral tachistoscopic presentation. Brain Cogn. 1986;5(2):131–62.
24. Wilson HR, Levi D, Maffei L, Rovamo J, DeValois R. The perception of form: retina to striate cortex. In: Spillmann L, Werner JS, editors. Visual perception: the neurophysiological foundations. NewYork: Academic Press; 1990. p. 231–72.
25. Carrasco M, Frieder KS. Cortical magnification neutralizes the eccentricity effect in visual search. Vis Res. 1997;37(1):63–82.
26. He PY, Kowler E. Saccadic localization of eccentric forms. J Opt Soc Am A 1991;8(2):440–9.
27. Kowler E, Blaser E. The accuracy and precision of saccades to small and large targets. Vis Res. 1995;35(12):1741–54.
28. Dick S, Ostendorf F, Kraft A, Ploner CJ. Saccades to spatially extended targets: the role of eccentricity. Neuroreport. 2004;15(3):453–6.
29. Walker R, Deubel H, Schneider WX, Findlay JM. Effect of remote distractors on saccade programming: evidence for an extended fixation zone. J Neurophysiol. 1997;78(2):1108–19.
30. Tatler BW, Vincent BT. The prominence of behavioural biases in eye guidance. Vis Cogn. 2009;17(6):1029–54.
31. Bahill AT, Adler D, Stark L. Most naturally occurring human saccades have magnitudes of 15 degrees or less. Invest Ophthalmol Vis Sci. 1975;14(6):468–9.
32. Heywood S, Churcher J. Structure of the visual array and saccadic latency: implications for oculomotor control. Q J Exp Psychol. 1980;32(2):335–41.
33. Hackman RB. An experimental study of variability in ocular latency. J Exp Psychol. 1940;27(5):546–58.