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Impact of Simulated Central Scotomas on Visual Search in Natural Scenes

Lee McIlreavy*, Jozsef Fiser†, and Peter J. Bex†

ABSTRACT

Purpose. In performing search tasks, the visual system encodes information across the visual field at a resolution inversely related to eccentricity and deploys saccades to place visually interesting targets upon the fovea, where resolution is highest. The serial process of fixation, punctuated by saccadic eye movements, continues until the desired target has been located. Loss of central vision restricts the ability to resolve the high spatial information of a target, interfering with this visual search process. We investigate oculomotor adaptations to central visual field loss with gaze-contingent artificial scotomas.

Methods. Spatial distortions were placed at random locations in 25° square natural scenes. Gaze-contingent artificial central scotomas were updated at the screen rate (75 Hz) based on a 250 Hz eye tracker. Eight subjects searched the natural scene for the spatial distortion and indicated its location using a mouse-controlled cursor.

Results. As the central scotoma size increased, the mean search time increased [$F(3,28) = 5.27$, $p = 0.05$], and the spatial distribution of gaze points during fixation increased significantly along the x [$F(3,28) = 6.33$, $p = 0.002$] and y [$F(3,28) = 3.32$, $p = 0.034$] axes. Oculomotor patterns of fixation duration, saccade size, and saccade duration did not change significantly, regardless of scotoma size.

Conclusions. There is limited automatic adaptation of the oculomotor system after simulated central vision loss.
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Key Words: scotoma, saccade, fixation, eye tracking, visual search

Saccades deliver the retinal image of an object to a specific retinal locus for detailed inspection during a period of fixation. The retinal locus for fixation in normally sighted subjects remains within 160 arc min[21] corresponding to a retinal diameter of approximately 35 foveal cone cells. Macular diseases, including age-related macular degeneration (AMD), may damage the fovea through various pathologic processes, resulting in a discrete region of blindness called a scotoma. Scotomas arising from macular disease are the leading cause of visual impairment in North America and Europe.[2] Attempting to fixate the former location of the fovea in an eye with central scotoma will image objects within the scotoma. Consequently, subjects may perceive objects as blurred, distorted, indistinct, or having vanished entirely from view.[3] To fixate without interference from foveal scotomas, many patients position their eyes so as to image objects upon a locus of relatively unimpaired eccentric retina,[4,5] called a preferred retinal locus (PRL). Failure to efficiently use existing unimpaired retina may preclude patients from achieving optimal performance in everyday visual tasks.

Bertera[6] highlighted that studying central retinal scotomas in patient populations is complicated by concurrent ocular disease as well as variation in the characteristics of scotomas between individuals. Artificial central scotomas provide a practical alternative because the experimenter can systematically manipulate individual scotoma characteristics in the same subject, thus eliminating confounding influences and greatly increasing statistical power.

Many techniques have been used to generate artificial central scotomas, including modified scleral contact lenses.[7] Currently, the preferred technique uses eye tracking[6,8–10] to achieve selective image stabilization. In brief, this involves producing an arbitrarily shaped mask that obscures part of a stimulus background or scene. The mask is moveable, controlled by means of signals from the eye tracker. When a subject moves his/her eyes, the obscuring mask is displaced in such a manner to remain fixed with respect to the subject's fovea, simulating the effects of central visual loss.

In the past few decades, researchers have investigated the effect of artificial central scotomas on the oculomotor behavior of normal

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subjects. To elicit eye movements, these studies used visual search tasks.[6,10] However, the visual search stimuli have been presented in highly regular matrices and lacked features and contrast variation normally present in natural scenes. Consequently, the location of candidate targets is highly predictable, and the oculomotor behavior for such search tasks may not correspond to that under more natural viewing conditions.

By introducing natural variation in luminance, contrast, textures, edge density, and object sizes into a visual search task, we aim to study the impact of artificial central vision loss under more realistic conditions than have been used previously. Natural variation in spatial structure can be implemented by requiring subjects to search for targets embedded within calibrated natural images. If the location of the target and non-target features were entirely random, previous knowledge of the stimulus layout could not influence oculomotor behavior.[11] Alternatively, replacing the search target with that of a random spatial distortion does not change natural image statistics (local spatial structure, luminance, contrast, edge density, or amplitude spectrum) in a predictable way.[12] This avoids the introduction of abrupt changes in luminance, chrominance, or contrast and prevents the use of a single filter to make the task unnaturally simple—e.g., the task of finding a "C" target among "O" distractors[10] or finding a Gabor target in pink noise.[13] Furthermore, distortion targets can be introduced anywhere in the natural scene, which prevents subjects from searching in expected locations (e.g., table tops only for cups) and avoids the introduction of obvious contextual anomalies (e.g., a cup in a tree).

Recent work suggests that training on a visual search task can improve mobility performance under low-light conditions for people with visual impairments, including AMD.[14,15] To develop novel rehabilitation interventions, a better understanding of the short-term oculomotor adaptations of the visual system after scotoma onset is needed. We, therefore, investigate short-term oculomotor adaptations to artificial central visual loss while normally sighted subjects conduct a visual search task within natural scenes.

METHODS

The research was conducted at the Schepens Eye Research Institute and followed the tenets of the Declaration of Helsinki. The research was approved by the departmental institutional review board. Subjects gave their informed consent before commencing the study.

Subjects

One of the authors (LM) and seven naive volunteers participated in the study (mean age: 23.1 years; range: 21–25 years). All subjects had normal or corrected-to-normal visual acuity of 0.0 logMAR or better, a Pelli–Robson contrast sensitivity of 1.80 log units or better, and no known ophthalmic disease. Subjects performed all trials under monocular conditions using their preferred eye, with their contralateral eye occluded with an eye patch. Other than one author (LM), no subject had previously participated in artificial scotoma or eye-movement experiments.

Stimuli were generated on a personal computer using MATLAB (Mathworks, Natick, MA), and routines from the PsychToolbox were used.[16,17] Stimuli were displayed on a gamma-corrected LaCie Electron 22 in CRT monitor (LaCie U.S., OR) with a mean luminance of 50 cd/m^2 at a frame rate of 75 Hz calibrated with a Minolta LS100 photometer. The display measured 36° horizontally (1152 pixels), 27° vertically (864 pixels), and was positioned 57 cm from the observer in an otherwise dark room.

Stimuli

A set of 4165 calibrated 16-bit grayscale natural images was downloaded from an online database (available at: http://hlab.phys.rug.nl/archive.html), the characteristics of which have been described in detail elsewhere.[18] On each trial, a 1536 × 1024 pixel source image was selected at random from all 4165 images, and an 800 × 800 pixel area (25° × 25° square) was then sampled at random from the source image and became the experimental image. No attempt was made to select images for particular content. The global root-mean-square contrast (the standard deviation of pixel values divided by the mean) of the experimental images was fixed at 0.2.

An area within the central 23° × 23°, thus avoiding the edge of the test image, was selected at random to contain the distortion. Fig. 1 shows a representative image in which a distortion has been introduced at a random location indicated by the red dashed circle (that was not present in the experiment). Spatial distortions were introduced by remapping the pixels from the source image to the distorted image. The remapping was controlled with band-pass filtered noise, newly randomly generated for each trial, using log exponential filters:

$$A(\omega)\alpha\ \exp\left(-\frac{|\ln(\omega/\omega_{peak})|^3 \ln 2}{(b_{0.5} \ln 3)^3}\right)$$

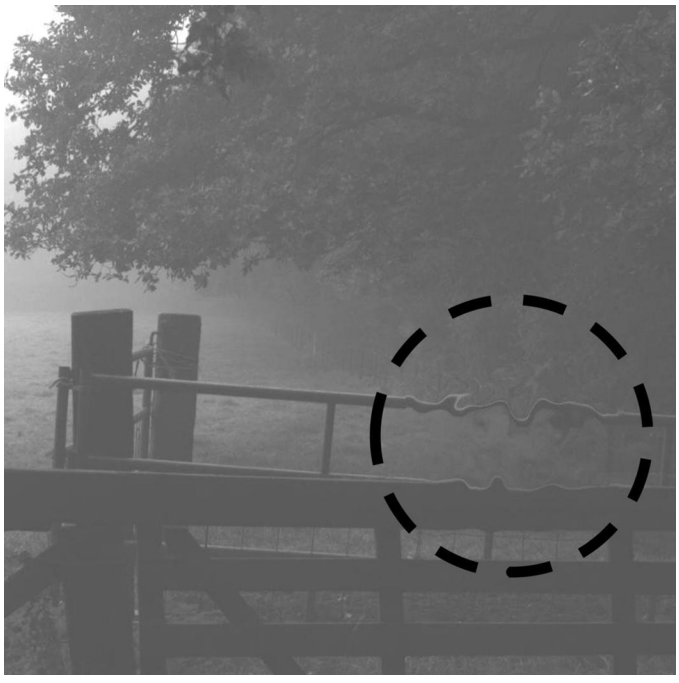

FIGURE 1.

Illustration of spatial distortion in natural image. Distortions were smoothly blended into a random location with a Gaussian window of $\sigma_{x,y} = 1°$. Although these images are probably unfamiliar and the first-order statistics of the distorted region do not differ from the rest of the image, the distortion (ringed with a red dashed circle that was not present in the experiments) can be detected with minimum effort.

where ω is spatial frequency, ω_{peak} specifies the peak frequency, which was fixed at 0.5 cycles per degree, and $b_{0.5}$ is the half bandwidth of the filter in octaves, which was fixed at 0.5 octaves (full width = 1 octave). The mean value of the band-pass filtered noise was fixed at 0, and its amplitude ($-\alpha$ to $+\alpha$), which controlled the magnitude of distortion, was fixed at 0.5°. One band-pass filtered random noise sample controlled the horizontal displacement of each pixel, and a separate band-pass filtered noise sample (with the same peak spatial frequency and amplitude) controlled the vertical displacement of each pixel. Bilinear interpolation was used to support subpixel precision. Spatial distortions were smoothly blended into the image with a Gaussian window with a standard deviation of 1°, thus the magnitude of distortion beyond a 2° radius ($\pm 2\sigma$) approached 0, which ensured that there were no abrupt transitions between distorted and undistorted areas of the image. The magnitude of the spatial distortion and the size of the Gaussian window were based on data from a recent study[12] in which we measured detection thresholds for spatial distortions in natural scenes across the visual field. The magnitude (0.5°) and spatial period (2°) of spatial distortion used in the present study were based on those data that were above detection threshold at all eccentricities and should therefore be detectable by peripheral vision with artificial central vision loss.

Central field loss was simulated with a circular Gaussian mask that occluded the background natural image on the CRT display. The Gaussian mask was continuously centered on fixation and progressively reduced the contrast to zero at the center of fixation. Three Gaussian central scotoma sizes were used: $\sigma_{x,y} = 1$, 2, or 4°. The x and y coordinates of the artificial visual loss were defined by the gaze-position data of the subject's preferred eye, sampled at 250 Hz with a video eye tracker (High Speed Video Eyetracker Toolbox; Cambridge Research Systems, Cambridge, UK). To determine the latency of the system, the delay between a drawing command to the graphics card and its corresponding update on the screen was measured with a photodiode and a USB-attached digital I/O acquisition board. The mean latency was 20.35 ms ($\sigma_{latency}$ = 3.75 ms) at the top of the screen. At 75 Hz, one refresh takes 13.33 ms, so the gaze-contingent updates nearly always occurred on the next video frame, but the gaze-contingent scotoma was potentially misaligned for 20 ms. The results are unlikely to be compromised by such a small temporal lag, as any information present for 13 ms or less will be unlikely to reach detection threshold.[19] To maintain the correct viewing distance and accurate eye tracking, a chin and forehead rest were used to restrict head movements. Many patients with central scotomas <6° do not readily perceive their scotoma on clinical testing,[20] and the scotoma can appear filled-in with the surrounding spatial structure.[3,21–24] The reported time course for the filling-in of scotomas depends on whether it is real or simulated, and if simulated, the method used to generate them. For real scotomas, perceptual completion took place instantaneously.[22] Scotomas simulated as an eccentrically viewed homogenous gray square filled in within 2 to 3 s,[25] whereas those simulated by retinal stabilization filled in after several seconds.[26] Subjective reports from all subjects in this current study indicated that filling-in of artificial scotomas did not occur. Nevertheless, whether the observer experienced filling-in over the artificial scotoma is irrelevant for the present task. The central gaze point of the search image was occluded by the simulated scotoma, and whether it appeared filled-in or randomly textured did not alter the absence of a foveal view of the stimulus.

Procedure

A minimum of two runs of visual search trials were performed in succession. Each run consisted of 70 search trials, 10 trials for each of the six artificial visual loss conditions: three central-simulated scotomas, three peripheral-simulated scotomas (not discussed here) and a control condition with no simulated visual field loss. Before each run, subjects performed an eye-tracker calibration for a set of 20 known target positions, evenly spaced over an area subtending 20° × 16°, using the calibration routines supplied with the eye tracker, accessed from MATLAB. Each run was relatively short, and it was not necessary to recalibrate the eye tracker between trials. Furthermore, the gaze-contingent scotoma was continuously visible during the experiment, and observers were instructed to inform the experimenter if the center of the scotoma drifted from the center of fixation. No subject reported any drift.

The search trials commenced with subjects fixating an isoluminant green central dot subtending 11 arc min in the center of an otherwise uniform field of mean luminance. To initiate a trial, the subject pressed a response button on a computer mouse, which caused the uniform background and fixation target to be immediately replaced with the experimental image and gaze-contingent artificial visual field loss superimposed. It was then the task of the subject to search the natural scene for the spatial distortion and, once located, to indicate its position by moving a mouse-controlled cursor to the center of the distortion and pressing a response button on the mouse. The standard Microsoft Windows XP operating system arrow was used in this experiment. It was composed of a white center (100 cd/m^2) and a black outline (0.1 cd/m^2) and was easily visible to subjects owing to its relatively high contrast against 0.2 root-mean-square contrast natural images. A correct feedback signal was provided if the cursor fell within a radius of 2° from the center of the distortion, outside which an incorrect response was recorded. Visual feedback was provided at the fixation dot, which was isoluminant green after a correct response or isoluminant red after an incorrect response. Finally, the subject returned their fixation to the central dot ready to begin the next trial. At the commencement of each trial, the cursor remained at, and was continuous with, the last cursor position. The order of the 10 trials for each of seven conditions was randomly interleaved.

No time limit was imposed on any trial. Subjects were not provided with any instruction on adopting a specific search strategy but were encouraged to search freely within the experimental image. Subjects were instructed not to guess, but to be confident of, the location of the spatial distortion before responding, if possible. Subjects were given sufficient practice trials to demonstrate all conditions of artificial visual loss and the spatial distortion target.

Data Analysis

Eye-movement data were analyzed offline using MATLAB software with routines written specifically for this purpose. A time-stamped data file of the x and y coordinates of gaze position, in millimeters from the screen center, was recorded at 250 Hz during all search trials. Gaze positions were not tracked between search

trials. Before the statistical analysis of eye-movement data, lost data samples arising from blink movements and eye-tracking failures were identified. Such lost gaze points amounted to <9.5% of the total data recorded. Gaze positions during lost intervals were linearly interpolated between the adjacent gaze positions, evenly distributing missing data points between recorded data points. Similar results were obtained when lost data points were removed or substituted with the last successfully recorded gaze position, which were alternative methods we considered. Once the interpolated data was substituted, all eye-position data were analyzed in the same way. A saccade was defined statistically as any change in eye position $>1.96\sigma$ above the mean of all changes in eye position made within a particular trial. For a comprehensive discussion on the use of our fixation-saccade classification technique, the reader is directed to Appendix 1 (available at http://links.lww.com/OPX/A103).

Saccade duration was defined as the time elapsed between saccade onset and saccade offset, and saccade amplitude was defined as the absolute distance between the last eye position of one fixation epoch and the first eye position of the next fixation epoch. Saccade frequency was expressed as the number of saccades per second. Fixations were defined as the collection of gaze points that were separated by eye movements $<1.96\sigma$ above the mean made in a particular trial. The small eye movements that occur during fixation were defined as microsaccades. Fixation duration was defined as the time elapsed between two successive saccades. Fixation frequency was expressed as the number of fixations per second. Fixation stability was defined as the spatial distribution of eye positions during a period of fixation. We calculated the horizontal and vertical standard deviation of fixation stability and the bivariate contour ellipse area (BCEA), with a probability area of 68%,[27] as an estimate of the area of a given fixation.

Search time was defined as the time elapsed between start of a trial and the mouse button press ending the trial. Before statistical analysis of search times, data were examined to determine the proportion of trials in which subjects had failed to locate the spatial distortion, that is, the position of the mouse-controlled cursor exceeded a 2° radius from the center of the target spatial distortion at the time of the mouse button press. Unfortunately, data on the accuracy of mouse placement were collected for only two of the eight subjects tested. Each of the two subjects completed 80 trials each, 20 trials for each of the four conditions. The combined data for both subjects are presented later in the article.

Given that subjects were permitted unlimited search time, no significant correlation was found between search time and failure to identify the spatial distortion. This suggests that despite the difficulty posed in searching with artificial visual field loss, subjects did not forfeit trials but rather made a persistent attempt to locate the spatial distortion. In addition, search time data exceeded 1 s in duration in >99% of search trials, indicating that subjects made a reasonable attempt to locate the spatial distortion during each trial (Table 2).

To determine whether search time, fixation duration, number of saccades, saccade duration, and saccade size were normally distributed, the raw data were analyzed with a two-sample Kolmogorov-Smirnov test, accessed with MATLAB's "ktest2" function. This analysis confirmed that the data were not significantly different from normally distributed and parametric statistics could be applied. Uncorrected raw data were then analyzed by within-subjects ANOVA statistical analysis using SPSS (SPSS, IBM Corporation).

For group analyses, the data for each subject were normalized to their performance in the control condition (no artificial visual field loss). This corrected for variations in baseline performance between subjects and for differences in the total number of trials completed by each subject. Search times and eye-movement parameters for each condition reported are the normalized mean and standard deviations of a minimum of 20 searches per condition per subject. Pearson linear correlation coefficient, r, was computed using the "corr" function accessed through MATLAB. The corresponding p value, an output of the "corr" function, indicates whether the correlation is significantly different from zero based on a Student's t-test.

RESULTS

A summary of the raw search time data for each condition is provided in Table 1. Fig. 2 illustrates mean normalized search time as a function of scotoma size. Mean normalized search time increased significantly with scotoma size [$F(3,28) = 5.27$, $p = 0.05$], and the two factors were significantly correlated [$r(30) = 0.569$, $p = <0.001$, with degrees of freedom based on eight subjects and four conditions]. The largest central scotoma condition, $\sigma_{x,y} = 4°$, resulted in a doubling of the mean search time. Tukey post hoc comparisons show that only this scotoma condition differed significantly from the control condition ($p = 0.003$).

There were strong correlations of the mean normalized number of saccades with both search time [$r(30) = 0.8337$, $p = <0.001$] and scotoma size [$r(30) = 0.538$, $p = 0.002$]. Saccade frequency remained constant across the control and artificial scotoma conditions ($\mu_{freq} = 2.57$ saccades per second, $\sigma_{freq} = 0.11$ saccades per second). No statistical difference was found in saccade duration between the control condition and the scotoma condition.

Of the saccades made in the control condition and in each of the scotoma conditions, 99% were ≤15° in amplitude. Fig. 3 shows

TABLE 1.
Summary of data for two subjects on the proportion incorrect trials

Scotoma size ($\sigma_{x,y}$)	μ; ($\pm\sigma$) search time (s)	μ; ($\pm\sigma$) error distance (°)	μ; ($\pm\sigma$) proportion of incorrect trials (%)
Control	4.54 (±4.69)	1.85 (±2.52)	25.0 (±10)
1°	4.57 (±3.07)	2.46 (±3.81)	32.5 (±13)
2°	6.32 (±5.74)	3.21 (±4.60)	40.0 (±16)
4°	8.92 (±8.84)	4.32 (±5.07)	42.5 (±17)

TABLE 2.
Summary of raw search time data for the control and each of the scotoma conditions

Scotoma size ($\sigma_{x,y}$)	μ; ($\pm\sigma$) raw search time (s)	Range (s)	
		Minimum	Maximum
Control	3.41 (±1.24)	1.46	5.30
1°	4.41 (±2.27)	2.81	9.76
2°	4.70 (±1.77)	1.89	7.31
4°	6.84 (±1.68)	4.82	9.94

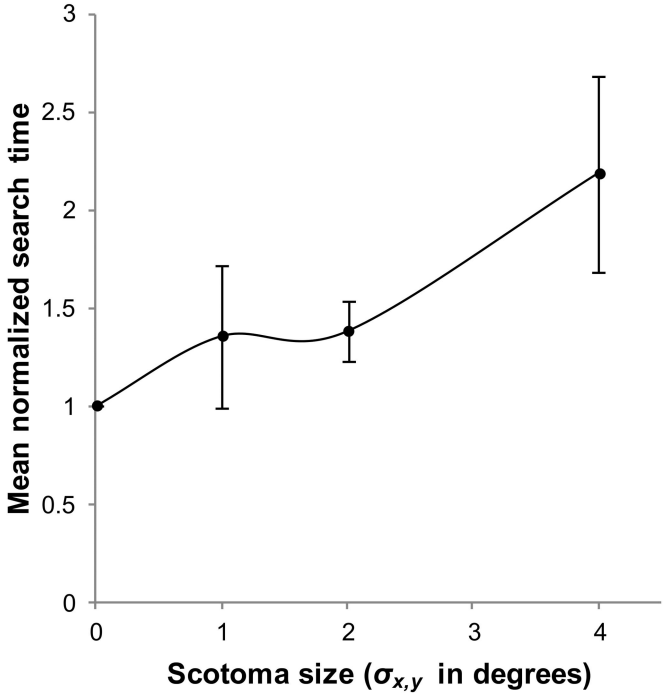

FIGURE 2.

Mean normalized search time as a function of scotoma size. Search time was defined as the time elapsed between the start of a trial and the mouse button press ending the trial. Error bars show ±95% confidence intervals.

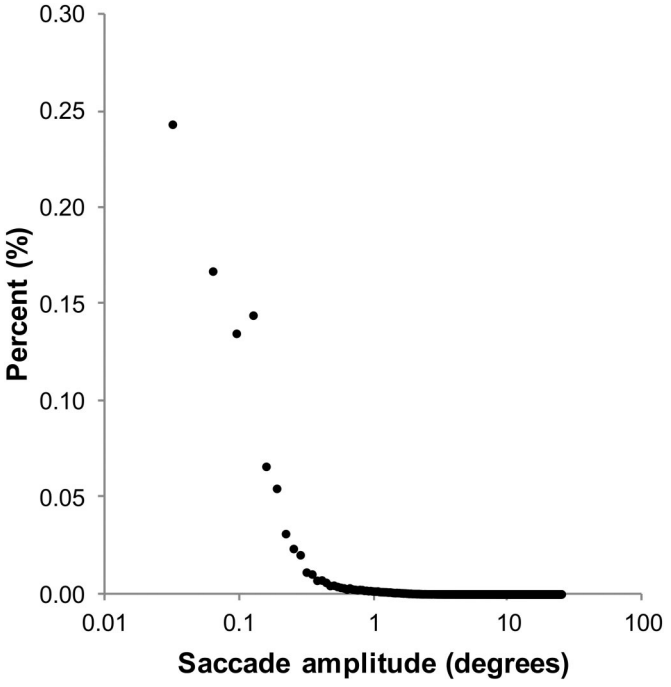

FIGURE 3.

Frequency distribution of saccade amplitude (degrees) for the control condition.

the frequency distribution of saccade amplitude for 24,010 saccades made in the control condition. This distribution was fitted with an exponential curve, of the form y = a*exp(−1/b*x), using the curve-fitting toolbox within MATLAB. The decay constant for the control condition exponential was $0.108°^{-1}$ ($R^2 = 0.982$). Similar decay constants of $0.100°^{-1}$ ($R^2 = 0.984$), $0.101°^{-1}$ ($R^2 = 0.982$), and $0.115°^{-1}$ ($R^2 = 0.977$) were obtained for central scotoma conditions of 1, 2, and 4°, respectively. This suggests no systematic relationship between saccade amplitude and scotoma size.

Figs. 4a to d illustrate the distributions of saccade amplitudes, plotted as histograms for each subject, for the control condition and scotoma conditions. In each case, saccade amplitudes have been $\log_{10}$ scaled to reveal the underlying distribution because the linear distributions were excessively skewed. The general distribution of saccade amplitudes in the control condition (Fig. 4a) is negatively skewed. Although none of the mean normalized saccade amplitudes for the scotoma conditions differs significantly from the control condition, Figs. 4b to d show that as scotoma size is increased, the distribution becomes less skewed and appears more bimodal, with the first peak for saccades at 1 to 1.5°and the second peak at approximately 3.5°. The decrease in negative skew indicates that subjects made fewer very small saccades when the scotoma was present (see discussion of fixation stability later in the text).

The statistical significance of bimodality was tested using the Hartigan dip test of unimodality. However, although there was a trend toward bimodality, none of the distributions in Fig. 4a to d were significantly different from unimodal, Fig. 4a (dip = 0.08, p = 0.196), b (dip = 0.0875, p = 0.094), c (dip = 0.0626, p = 0.607), and d (dip = 0.081, p = 0.189).

Given that the artificial scotoma was centered on fixation, if oculomotor behavior remained referenced on the fovea, there should be no saccades smaller than the radius of the scotoma because any peripheral target would therefore remain within the scotoma. We, therefore, determined the proportions of saccades that were smaller than the scotoma (i.e., saccades $\leq \sigma_{x,y}$). These were 0.119, 0.390, and 0.630 for $\sigma_{x,y}$ of 1, 2, and 4°, respectively. This means that for the smallest scotoma ($\sigma_{x,y} = 1°$), approximately 12% of eye movements failed to move the (masked) fovea out of the scotoma; this number increased to 63% for the largest scotoma ($\sigma_{x,y} = 4°$).

Because each saccade is followed by a fixation and vice versa, the data for fixation number and frequency are identical to that for saccades. As with saccade frequency, because the fixation frequency remained constant across the control and scotoma conditions, it must follow that fixation duration remained constant across the control and scotoma conditions. No statistical difference was found in fixation duration between the control condition and the scotoma conditions; however, it did vary a mean factor of 1.25 ($\sigma = 0.28$), 1.14 ($\sigma = 0.22$), and 1.23 ($\sigma = 0.21$) from the control condition for scotomas of $\sigma_{x,y} = 1$, 2, and 4°, respectively.

Fig. 5a plots the standard deviation of microsaccades during periods of fixation as a function of scotoma size, and Fig. 5b plots the BCEA as a function of scotoma size. The spatial distribution of microsaccades during fixation and the BCEA are both indications of fixation instability, that is, when eye position is distributed over a larger area during a fixation, then fixation is more unstable. The mean BCEA was 947 min arc^2 for the control condition, which increased to 3398 min arc^2 for the 4° scotoma. We next examined horizontal and vertical standard deviations to determine the principal source of this additional variation. The standard deviation of x axis [$F(3,28) = 6.33$, $p = 0.002$] and y axis [$F(3,28) = 3.32$, $p = 0.034$] microsaccades during fixation increased significantly with scotoma size. The x and y axis standard deviations were significantly correlated with scotoma size [$r(30) = 0.600$, $p = <0.001$;

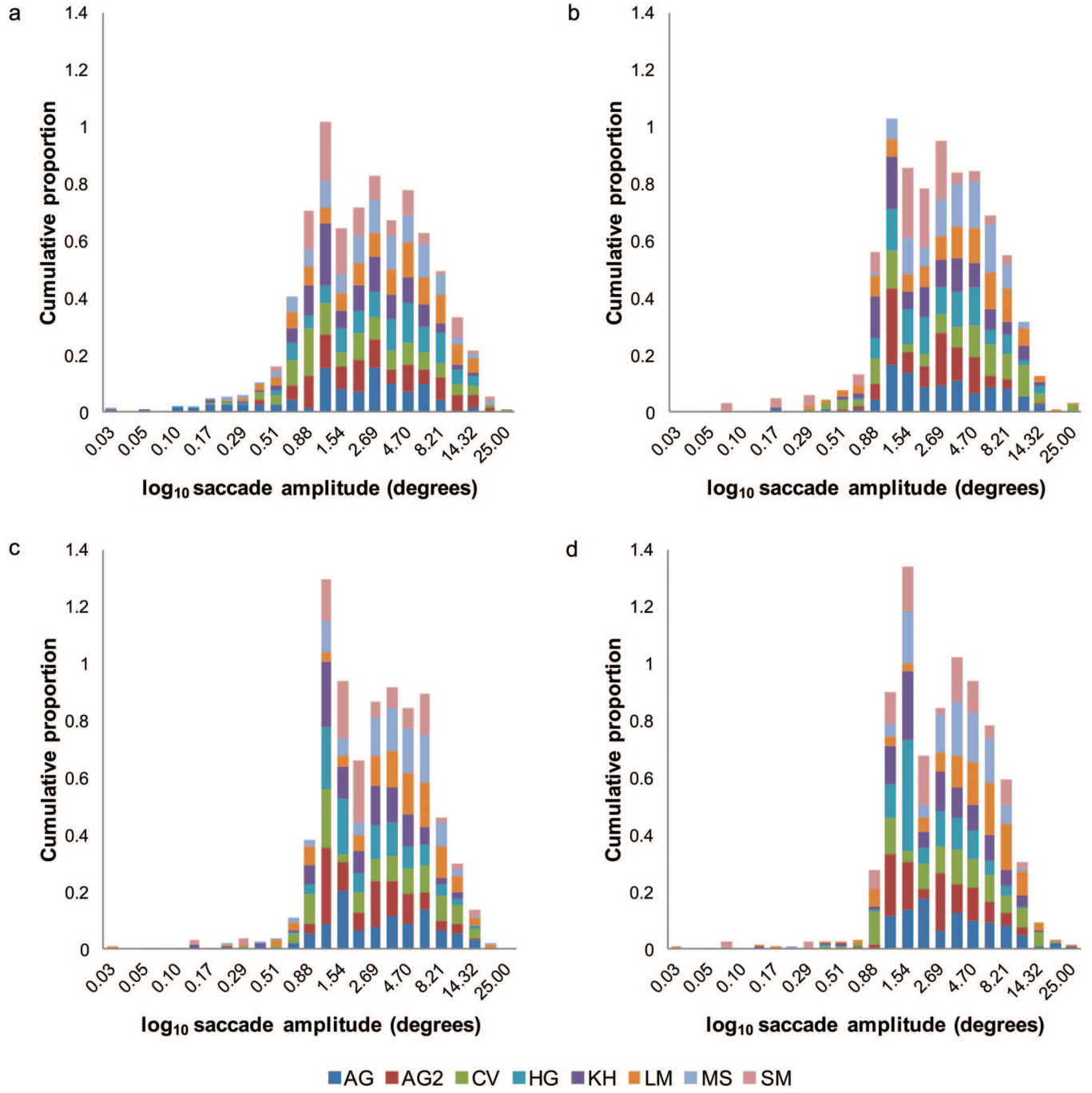

FIGURE 4.

Distribution of $\log_{10}$ mean saccade amplitudes for eight subjects, indicated by the caption. Cumulative proportion of saccade amplitudes for (a) control—no scotoma condition, (b) scotoma $\sigma_{x,y} = 1°$, (c) scotoma $\sigma_{x,y} = 2°$, and (d) scotoma $\sigma_{x,y} = 4°$. It is important to note that the cumulative proportions for each subject, represented by a different color, sum to unity.

and r(30) = 0.493, p = 0.004, respectively]. Tukey post hoc comparisons showed that only the largest scotoma condition ($\sigma_{x,y} = 4°$) differed significantly from the control condition on both the x axis (p = 0.001) and the y axis (p = 0.021) microsaccade distributions. These data show that fixation became progressively more unstable as the size of the artificial scotoma increased, with slightly larger increases in horizontal microsaccadic eye movements.

Although the magnitude of the horizontal fixation distribution exceeds that of the vertical ($\mu_{difference}$ = 1.426 pixels/0.0445°, $\sigma_{difference}$ = 1.983 pixels/0.062°), there was no significant difference between the x and y axis distributions for the control condition [F(1,14) = 0.018, p = 0.896], $\sigma_{x,y} = 1°$ [F(1,14) = 0.399, p = 0.538], $\sigma_{x,y} = 2°$ [F(1,14) = 0.005, p = 0.946], or $\sigma_{x,y} = 4°$ [F(1,14) = 1.281, p = 0.227].

DISCUSSION

Search time increased with scotoma size. Individual variation in search time was observed, suggesting that some subjects were more

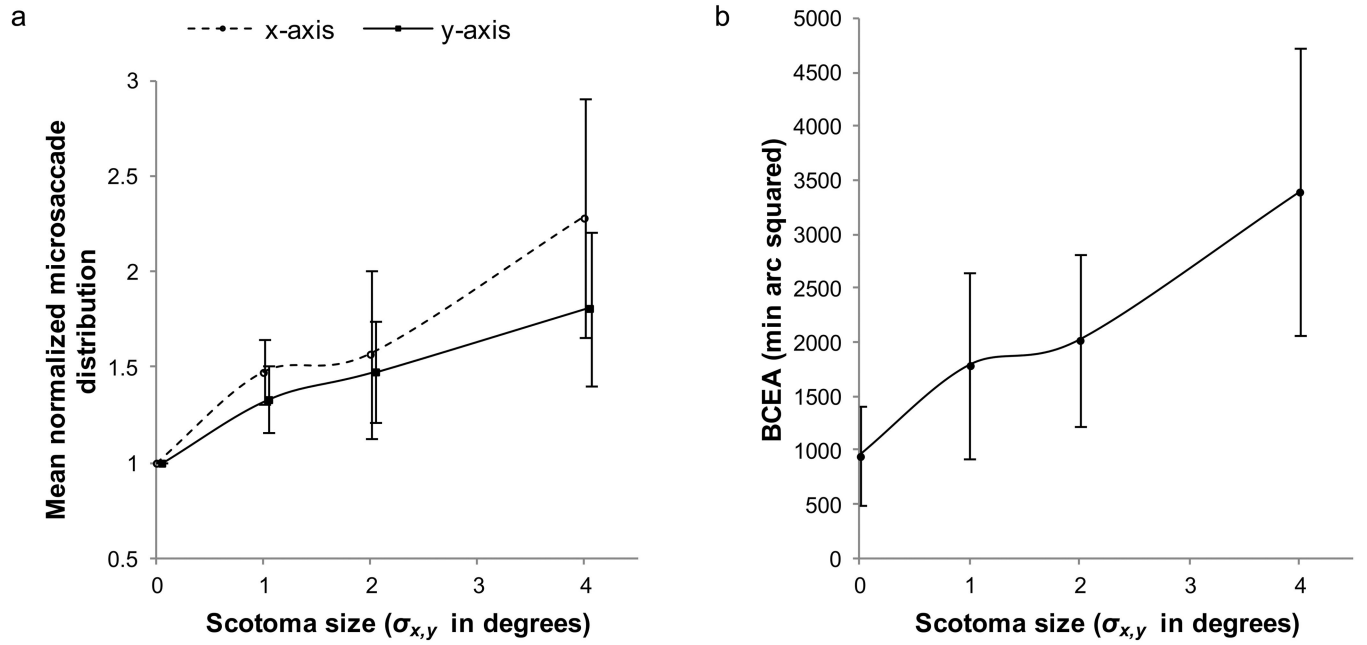

FIGURE 5.

(a) Mean normalized microsaccade standard deviation as a function of scotoma size for x axis (dashed line) and y axis (solid line). (b) Mean BCEA as a function of scotoma size.

impaired than others by the presence of a central scotoma. Although there was a trend at all sizes, only the largest scotoma condition, $\sigma_{x,y} = 4°$, was significantly different from the control condition. From this observation, we can conclude that either the visual system is able to immediately compensate for small to moderate amounts of central visual deprivation or the resolution of parafoveal vision with the two smaller scotomas ($\sigma_{x,y} \leq 2°$) was adequate for the present task. The latter suggests that the parafoveal vision can be sufficient for visual search within natural scenes, as long as the target object subtends an area that is larger than the scotoma. If so, the expected impairment in visual search after central vision loss will be highly dependent on the extent of loss and the size of the target to be detected.

No relationship was found between saccade frequency and scotoma size; trials in which search times were longer simply had a greater number of saccades. The amplitude of most saccades does not exceed 15° when viewing images or walking outdoors,[28] consistent with 99% of saccades in all conditions of the present study. However, the decay constants reported for the saccade frequency distributions are, in general, half of that measured in normal subjects for an outdoor walking task.[28] This indicates that saccades made during our visual search task were smaller than those made in full-field outdoor natural scenes.

No relationship was found between the mean saccade amplitude and scotoma size. In the present task, subjects were required to search within a natural scene and to identify and locate a spatial distortion. The position of the distortion was completely random, thereby precluding knowledge or priors about the likely location of particular objects in natural scenes[11] and thereby forcing the task to be entirely visually based. The magnitude and period of the spatial distortion was chosen to be identifiable across the retina so that any retinal location could, in principle, be used to complete the task. Therefore, the task does not provide any implicit stimulus for PRL development, and choosing a PRL closest to the scotoma boundary will improve the resolution of local spatial information. Although subjects were naive to this fact, the results seem to suggest that in the presence of a central scotoma, subjects continued to make saccades as normal, possibly using a temporary PRL.

The observation that many saccades (63% for the 4° scotoma) were smaller than the radius of the scotoma indicates that a nonfoveal retinal locus drove eye movements. This is because if eye movements remained referenced on the former fovea, all saccades smaller than the radius of the scotoma would retain the point of regard within the scotoma. This suggests that a temporary nonfoveal location may have been referenced for eye movements. We attempted to identify whether subject had spontaneously developed a PRL in two ways. First, we examined the direction of the center of fixation relative to the end point of each preceding saccade. We hypothesized that each saccade would initially bring the target into the location of the masked fovea. Next, a small corrective saccade would be deployed to bring the target out of the scotoma and into a PRL if one existed. According to this hypothesis, the direction of such a corrective saccade should be relatively constant. The presence of a second peak in the distribution of saccade sizes in scotoma conditions (Fig. 4) was consistent with this hypothesis. However, there was no consistent relationship between the end point of the saccade and the center of the following fixation. Second, we examined the relative location of gaze with respect to the target location at the end of the trial. At this point, we hypothesized that subjects would attend to the target with any PRL, had they developed one, and this would produce a fixed directional or angular relationship with the target and the calibrated foveal location. However, there was no consistent relationship between the final gaze point and the location of the target (Figure 6). Therefore, the non-foveal reference varied across saccades and therefore failed to meet the definition of a PRL. Despite

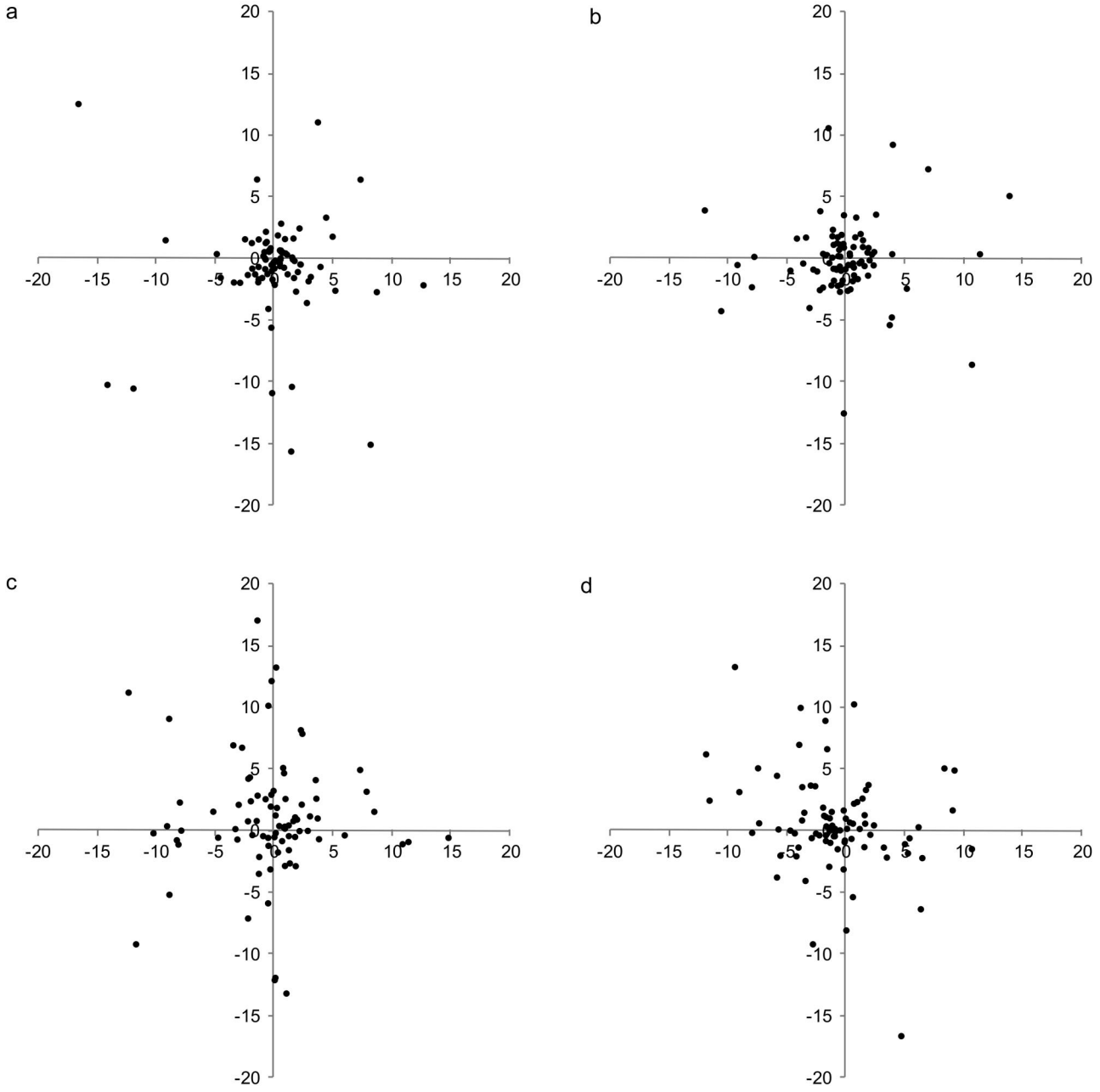

FIGURE 6.

Absolute distance, in degrees, of final fixation locations relative to the geometric center of the spatial distortion. Each plot is a representative sample for (a) control condition for subject LM, (b) $\sigma_{x,y} = 1°$ for subject CV, (c) $\sigma_{x,y} = 2°$ for subject SM, and (d) $\sigma_{x,y} = 4°$ for subject MS.

this finding, we attempted to determine whether subjects maximized the resolution of visual information, based on distance from former fovea. This is possible only by analyzing the final fixation relative to the spatial distortion, as it is impossible to know what subjects were attending to as they searched within the natural scenes. Because the spatial distortion was smoothly blended into the image with a Gaussian window of $\sigma_{x,y} = 1°$, the magnitude of distortion beyond a 2° radius (representing $\pm 2\sigma_{x,y}$) approached 0. To maximize the resolution of visual information in the control condition, subjects would have to have placed their fovea as close as possible to the spatial distortion. Thus, the distance between the geometric center of the distortion and the fovea would be less than the radius of the distortion (2°). In scotoma conditions, subjects would have to place the edge of their scotoma (defined as $1\sigma_{x,y} =$ 61% amplitude) within the radius of the spatial distortion (corresponding to a former foveal distance of $<2° + \sigma_{x,y}$). Using these definitions, subjects maximized the resolution of visual information of the target on 48.2, 67.1, 55.3, and 71.7% of trials for the control and $\sigma_{x,y} = 1$, 2, and 4° scotoma conditions, respectively.

Is it plausible for subjects to intuitively search natural scenes without the need for a PRL or to maximize the resolution of potential targets? The fovea is typically considered to subtend the central 5° of a monocular visual field that subtends approximately 130° vertically and 150° horizontally. At any one time, most of the visual field is imaged not on the high-resolution fovea but on lower resolution non-foveal regions. In addition, the world we inhabit is

not one composed solely of high-contrast or high-spatial frequency. The benefit expected to be gained through the use of a PRL for visual search tasks is questionable. This idea is supported in a case report by Crossland et al.[29] detailing "An unusual strategy for fixation" in a patient with advanced bilateral AMD. The patient's fixation behavior suggested a preference for a larger area of lower resolution retina for distance tasks and navigation but a smaller area of higher-resolution retina for reading.

We observed no significant relationship between fixation duration and scotoma size. Geisler et al.[13] found that fixation duration increased, by a factor of up to 1.5, when subjects took longer to search for a Gabor in pink noise. In this study, we found a comparable increase in fixation duration, but this failed to reach significance. Our results for fixation duration conflict with other studies that have used artificial scotomas with different tasks, including reading[30] and visual search tasks in highly regular search arrays.[6,10] These tasks did not use natural images but instead resolution-dependent stimuli. For the highly regular search arrays, this took the form of a gap subtending either 0.05 to 0.10°[6] or 0. to 0.93°,[10] whereas for reading, text subtending 0.52 to 2°. The variation in spatial structure within our experimental images did not require an explicit resolution threshold to achieve the task—we intentionally selected a distortion that could be detected equally well at all eccentricities. Given that the temporal integration period increases[31] with retinal eccentricity, if subjects had attempted to use nonfoveal vision to maximize resolution, we would have expected an increase in fixation duration with scotoma size. However, subjects did not spontaneously make this adaptation, and this suggests that rehabilitation efforts might concentrate on training longer more efficient fixations.

A strong relationship was found between fixation stability and scotoma size. The BCEA for normal subjects without macular disease who fixate a single isolated target for several seconds, often within a microperimetry system, is generally $<$1200 min arc^2.[27,32–35] The present results show comparable BCEA values for brief periods of fixation during visual search within a natural scene. Previous studies failed to find a clear relationship between scotoma size and BCEA in patients with macular disease, both long-standing[33,36] and of recent onset.[27] Although a larger scotoma may be expected to force a subject fixate with more eccentric retina, this is based on the assumption of a symmetrical and foveally centered scotoma, as in the present study. Unlike our simulation, this is rarely, if ever, the case for pathological central scotomas. A more accurate prediction of fixation impairment in pathological vision loss may be achieved by considering the minimum distance between the scotoma boundary and the former fovea, that is, the minimum eccentricity of the scotoma boundary. We found an increase in fixation instability with scotoma size, but this reached statistical significance only for the largest scotoma. This implies a critical scotoma eccentricity, beyond which fixation becomes significantly unstable. This idea is consistent with previous research that found unstable fixation for artificial scotomas of $>$10° diameter (for comparison, a $\sigma_{x,y}$ of approximately 5°), but found little reduction in fixation stability with scotomas of 5° diameter (for comparison, a $\sigma_{x,y}$ of approximately 2.5°).[37] It is likely that this impairment in fixation stability contributes to the impairment of visual search performance observed by reducing identification performance during periods of fixation. Although fixation instability is likely to reduce contrast sensitivity at a given eccentricity relative to an eye with stable fixation, it has been previously shown that acuity for high-contrast targets is relatively unaffected, unless the fixation instability moves the target to more eccentric locations.[38] Crowding of features within the natural scenes is also likely to have occurred as subjects viewed eccentrically.[39] This may have led to difficulty in disambiguating features when viewing with peripheral retina. Note that in previous studies, targets were presented at relatively isolated locations, surrounded by blank backgrounds of local mean luminance, thus minimizing the effects of crowding, but limiting the relevance to visual search in natural scenes.

Both horizontal and vertical components of fixation became increasingly unstable with an increase in scotoma size. Despite a symmetrical central scotoma, this was more pronounced along the horizontal axis than vertical axis. Given the asymmetry between the two meridia, tasks requiring eye movements parallel to the meridian of greatest instability (e.g., horizontal for reading) are likely to be more detrimentally affected than tasks requiring perpendicular eye movements.

Several recent studies have reported benefits of visual search training for mobility in patients with low vision, mostly under conditions of low luminance or contrast.[14,15] The present results suggest that the main oculomotor change with the use of peripheral vision is an increase in fixation in stability. It is therefore possible that visual search training in these studies aided either the stability or the duration of fixation. Measures to counteract fixation instability, either by means of meridian specific training or manipulation of stimuli along the axis of greatest fixation instability (magnification, contrast enhancement etc), may facilitate the development of viewing strategies beneficial to patients with central vision loss.

We acknowledge that a temporary simulated scotoma may only partially reproduce the effects of a chronic pathological scotoma, which cannot be simulated reliably with current technology. We speculate that for stable pathological scotomas, search efficiency and eye-movement accuracy may improve with a trained PRL. However, owing to the progressive nature of central vision loss from AMD, it is likely that this performance gain may require ongoing rehabilitation. A limitation of this study is that a spatial distortion is an ideal search target only in subjects with normal vision, no ophthalmic disease, and no metamorphopsia. We have devised alternative visual search tasks for ongoing work in clinical subjects with ophthalmic disease with and without metamorphopsia.[40]

CONCLUSIONS

This study found impairment in visual search of natural scenes in the presence of central scotomas. This impairment is primarily due to a reduction in fixation stability associated with the use of peripheral vision for fixation in the presence of a central scotoma. The results indicate a minimum scotoma extent beyond which fixation becomes significantly unstable. For absolute central scotomas, the eccentricity of the scotoma boundary from the former fovea is a better predictor than scotoma size for fixation impairment. No difference was found between the oculomotor system between normal subjects and those viewing with artificial central scotomas. This result suggests that subjects intuitively conduct visual search without any attempt to maximize resolution. This failure may be particularly problematic for reading, which is lim-

ited by high-spatial frequency resolution and may critically depend on PRL development. Therefore, the requirement for the detection of high-spatial frequency targets may facilitate PRL training and rehabilitation. Although the use of a PRL may facilitate detailed tasks, it remains unclear whether a non-central focus of optic flow impacts on navigation and mobility. Spatial resolution does not appear to be a factor in the present visual search task, in which targets were designed to be visible at all eccentricities. This property may be desirable for overcoming the fixation instability, but it may not promote the development of a stable PRL.

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APPENDIX

The Appendix is available at http://links.lww.com/OPX/A103.

REFERENCES

1. Kosnik W, Kline D, Fikre J, Sekuler R. Ocular fixation control as a function of age and exposure duration. Psychol Aging 1987;2:302–5.
2. Klein R, Klein BE, Tomany SC, Meuer SM, Huang GH. Ten-year incidence and progression of age-related maculopathy: the Beaver Dam eye study. Ophthalmology 2002;109:1767–79.
3. Burke W. Psychophysical observations concerned with a foveal lesion (macular hole). Vision Res 1999;39:2421–7.
4. Von Noorden GK, Mackensen G. Phenomenology of eccentric fixation. Am J Ophthalmol 1962;53:642–60.
5. Whittaker SG, Cummings RW, Swieson LR. Saccade control without a fovea. Vision Res 1991;31:2209–18.
6. Bertera JH. The effect of simulated scotomas on visual search in normal subjects. Invest Ophthalmol Vis Sci 1988;29:470–5.
7. Yarbus AL. Eye movements during the examination of complicated objects. Biofizika 1961;6:52–6.
8. Crane HD, Kelly DH. Accurate simulation of visual scotomas in normal subjects. Appl Opt 1983;22:1802.
9. Crane HD, Steele CM. Generation-V dual-Purkinje-image eye tracker. Appl Opt 1985;24:527.
10. Cornelissen FW, Bruin KJ, Kooijman AC. The influence of artificial scotomas on eye movements during visual search. Optom Vis Sci 2005;82:27–35.
11. Ehinger KA, Hidalgo-Sotelo B, Torralba A, Oliva A. Modeling search for people in 900 Scenes: a combined source model of eye guidance. Vis Cogn 2009;17:945–78.
12. Bex PJ. (In) sensitivity to spatial distortion in natural scenes. J Vis 2010;10(2):23, 1–15.
13. Geisler WS, Perry JS, Najemnik J. Visual search: the role of peripheral information measured using gaze-contingent displays. J Vis 2006;6(9):858–73.
14. Fuhr PS, Liu L, Kuyk TK. Relationships between feature search and mobility performance in persons with severe visual impairment. Optom Vis Sci 2007;84:393–400.
15. Kuyk T, Liu L, Elliott J, Fuhr P. Visual search training and obstacle avoidance in adults with visual impairments. J Visual Impair Blind 2010;104:215–27.
16. Brainard DH. The psychophysics toolbox. Spat Vis 1997;10:433–6.
17. Pelli DG. The VideoToolbox software for visual psychophysics: transforming numbers into movies. Spat Vis 1997;10:437–42.
18. van Hateren JH, van der Schaaf A. Independent component filters of natural images compared with simple cells in primary visual cortex. Proc Biol Sci 1998;265:359–66.
19. Nachmias J. Effect of exposure duration on visual contrast sensitivity with square-wave gratings. J Opt Soc Am 1967;57:421–7.
20. Schuchard RA. Validity and interpretation of Amsler grid reports. Arch Ophthalmol 1993;111:776–80.
21. Cohen SY, Lamarque F, Saucet JC, Provent P, Langram C, LeGargasson JF. Filling-in phenomenon in patients with age-related macular degeneration: differences regarding uni- or bilaterality of central scotoma. Graefes Arch Clin Exp Ophthalmol 2003;241:785–91.
22. Gerrits HJ, Timmerman GJ. The filling-in process in patients with retinal scotomata. Vision Res 1969;9:439–42.
23. Sergent J. An investigation into perceptual completion in blind areas of the visual field. Brain 1988;111(Pt. 2):347–73.
24. Zur D, Ullman S. Filling-in of retinal scotomas. Vision Res 2003;43:971–82.
25. Ramachandran VS, Gregory RL. Perceptual filling in of artificially induced scotomas in human vision. Nature 1991;350:699–702.
26. Gerrits HJ, De Haan B, Vendrik AJ. Experiments with retinal stabilized images. Relations between the observations and neural data. Vision Res 1966;6:427–40.
27. Crossland MD, Culham LE, Rubin GS. Fixation stability and reading speed in patients with newly developed macular disease. Ophthalmic Physiol Opt 2004;24:327–33.
28. Bahill AT, Adler D, Stark L. Most naturally occurring human saccades have magnitudes of 15 degrees or less. Invest Ophthalmol 1975;14:468–9.
29. Crossland MD, Kabanarou SA, Rubin GS. An unusual strategy for fixation in a patient with bilateral advanced age related macular disease. Br J Ophthalmol 2004;88:1479–80.
30. Scherlen AC, Bernard JB, Calabrese A, Castet E. Page mode reading with simulated scotoma s: oculomotor patterns. Vision Res 2008;48:1870–8.
31. Cheong AM, Legge GE, Lawrence MG, Cheung SH, Ruff MA. Relationship between slow visual processing and reading speed in people with macular degeneration. Vision Res 2007;47:2943–55.
32. Rohrschneider K, Becker M, Kruse FE, Fendrich T, Volcker HE. Stability of fixation: results of fundus-controlled examination using the scanning laser ophthalmoscope. Ger J Ophthalmol 1995;4:197–202.
33. Timberlake GT, Mainster MA, Peli E, Augliere RA, Essock EA, Arend LE. Reading with a macular scotoma. I. Retinal location of scotoma and fixation area. Invest Ophthalmol Vis Sci 1986;27:1137–47.
34. Kosnik W, Fikre J, Sekuler R. Visual fixation stability in older adults. Invest Ophthalmol Vis Sci 1986;27:1720–5.
35. Steinman RM. Effect of target size, luminance, and color on monocular fixation. J Opt Soc Am 1965;55:1158–65.
36. White JM, Bedell HE. The oculomotor reference in humans with bilateral macular disease. Invest Ophthalmol Vis Sci 1990;31:1149–61.
37. Boman DK, Kertesz AE. Horizontal fusional responses to stimuli containing artificial scotomas. Invest Ophthalmol Vis Sci 1985;26:1051–6.
38. Falkenberg HK, Rubin GS, Bex PJ. Acuity, crowding, reading, and fixation stability. Vision Res 2007;47:126–35.
39. Bouma H. Interaction effects in parafoveal letter recognition. Nature 1970;226:177–8.
40. Wiecek EK, Jackson ML, Dakin S, Bex PJ. Visual search with image enhancements in age-related macular degeneration. Invest Ophthalmol Vis Sci 2012;53:E-abstract 4392.

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