The retinal locus of fixation in simulations of progressing central scotomas

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Patients with central scotoma use a preferred retinal locus (PRL) of fixation to perform visual tasks. Some of the conditions that cause central scotoma are progressive, and as a consequence, the PRL needs to be adjusted throughout the progression. The present study investigates the peripheral locus of fixation in subjects under a simulation of progressive central scotoma. Five normally sighted subjects participated in the study. A foveally centered mask of varying size was presented to simulate the scotoma. Initially, subjects developed a peripheral locus of fixation under simulation of a 6° scotoma, which was used as a baseline. The progression was simulated in two separate conditions: a gradual progression and an abrupt progression. In the gradual progression, the diameter of the scotoma increased by a fixed amount of either 1° or 2° of visual angle, thus scotomas of 8°, 10°, and 11° of visual angle were simulated. In the abrupt progression, the diameter was adjusted individually to span the area of the visual field used by the current peripheral locus of fixation. Subjects located the peripheral locus of fixation along the same meridian under simulation of scotoma progression. Furthermore, no differences between the fixation stability of the baseline locus of fixation and the incremental progression locus of fixation were found whereas, in abrupt progression, the fixation stability decreased significantly. These results provide first insight into fixation behavior in a progressive scotoma and may contribute to the development of training tools for patients with progressive central maculopathies.

Introduction

Patients with central vision loss use eccentric fixation during the performance of visual tasks. They repeatedly use one or more circumscribed locations of the functional retina for the performance of a specific visual task. This retinal location is referred to as the preferred retinal locus (PRL) of fixation and was widely studied in terms of location and stability (Cummings, Whittaker, Watson, & Budd, 1985; Timberlake, Peli, Essock, & Augliere, 1987; Whittaker, Budd, & Cummings, 1988; Guez, Le Gargasson, Rigaudiere, & O’Regan, 1993; Sunness, Applegate, Haselwood, & Rubin, 1996; Fletcher & Schuchard, 1997; Nilsson, Frennesson, & Nilsson, 2003; Crossland, Sims, Galbraith, & Rubin, 2004; Crossland, Culham, Kabarnow, & Rubin, 2005). The PRL was defined to be one or more regions of the functioning retina that is repeatedly aligned with a visual target for a specific task and may also be used for attentional deployment and as the oculomotor reference (Crossland, Engel, & Legge, 2011).

However, there is little known about the changes in the PRL position along the progression of the disease. For clinical and basic science, it is important to understand the effects that the evolving scotoma has on the PRL. The use of central scotoma simulations can be a model system for learning about how parafoveal fixations and oculomotor learning develops.

Some maculopathies that lead to central vision loss tend to progress gradually over time (e.g., Stargardt’s disease and geographic atrophy). In the case of Stargardt’s disease, studies with patients have demonstrated gradual as well as abrupt progression of the retinal damage and, furthermore, showed progressions beyond the macula (Francois & De Rouck, 1965; Klien, 1967; Irvine & Wergeland, 1972). To study the changes of PRL along the progression of a central scotoma can be a lengthy process that might include years of follow-up measurements; therefore, little is known about the
changes of PRL on cases of progression. Simulations of central vision loss have shown that individuals with normal vision can adopt a peripheral locus of fixation in a rapid and consistent manner (Pidcoe & Wetzel, 2006; Kwon, Nandy, & Tjan, 2013; Liu & Kwon, 2016; Barraza-Bernal, Rifai, & Wahl, 2017). Such simulations can be used to study oculomotor mechanisms underlying the development of an eccentric fixation under blocked foveal vision, and may contribute to clarification of visual and motor contributions to PRL development in retinal pathologies.

In the present study, we investigated changes in the location and fixation stability of a peripheral locus of fixation under simulation of progressive central scotoma. The locus of fixation was defined to be at the highest density of the fixations (Kwon et al., 2013), and fixation stability was quantified using the bivariate contour ellipse area (BCEA) of the fixations (Steinman, 1965; Crossland, Sims, Galbraith, & Rubin, 2004). We simulated the progression of a central scotoma in two separate conditions: a gradual progression and an abrupt progression. In the gradual progression, the diameter of the scotoma increased by steps of either 1° or 2° of visual angle. In the abrupt progression, the diameter of the scotoma was increased in such a way that the area of the visual field used by the current locus of fixation was completely blocked. This allowed addressing whether potential relocation of the locus of fixation on the same meridian was influenced by the degree of the scotoma progression. Specifically, from an oculomotor point of view, the situation in which the scotoma progresses gradually was represented by the gradual and partial overlap of the PRL with the scotoma, in which parts of the PRL area were still available for fixation. In contrast, in the abrupt progression, the PRL area was completely covered by the progressed scotoma; thus a new PRL had to be developed.

Subjects reselected the identical meridian for the new locus of fixation, independent from the simulated progression type (gradual or abrupt). Furthermore, fixation stability decreased significantly when the size of the scotoma was abruptly increased.

**Methods**

**Apparatus**

The stimuli were presented on a ViewPixx/3-D display with a vertical refresh rate of 100 Hz and a spatial resolution of 1920 × 1080 pixels.

Eye-positional data were collected using a gaze-contingent setup based on MATLAB (MathWorks, Inc., Natick, MA) and the Eyelink 1000 Plus eye tracker (SR Research, Ltd., Ontario, Canada). The setup combined the Psychtoolbox (Brainard, 1997) and the Eyelink toolbox (Cornelissen, Peters, & Palmer, 2002) to present a set of gaze-dependent and gaze-independent stimuli. The gaze-dependent stimulus was a foveally centered circular mask. The gaze-independent stimulus was a target that evoked a saccadic eye movement. The target consisted of multiple components that were used in combination with a discrimination task to increase the fixation time of the subjects. Vertical and horizontal positions of the right eye were recorded at 1 kHz while the left eye was patched. The accuracy of the gaze-contingency system depended on two main factors: presentation delay of the stimuli, which was less than 20 ms, and the calibration inaccuracies of the eye tracker, which were monitored and did not exceed 0.1°. Thus we expect a fast retinal slip (driven by temporal delays) of a few arcmin and a slow retinal slip (induced by, for example, head movements) of approximately 0.1°. The developed PRLs will probably be impacted by the slow slip of 0.1°, which is a maximum of 10% of the width of the PRLs.

A chin rest was used to minimize movements of the head and to hold the eyes at a distance of 62 cm from the display.

**Participants**

Five participants took part in the study: three males and two females aged between 26 and 34 years (mean 28.8 years). The subjects had healthy eyes and normal or corrected-to-normal vision. Every participant had a peripheral locus of fixation, acquired under simulation of central scotoma after four training sessions (Barraza-Mernal, Rifai, & Wahl, 2017). A baseline measurement was used to control that the fixation behavior of the subjects was still present and that fixations were still consistent at one consistent location of the visual field. Only if the participants presented this fixation behavior were they qualified to participate in the study.

The study was performed in accordance with the declaration of Helsinki, and subjects signed an informed consent before their participation.

**Study design**

Figure 1 shows the sequence of experimental blocks performed in this study. In the first block, subjects performed a visual task without the scotoma to assess the variance of the fixations when the fovea is used as locus of fixation. In this block, the location and variance of the fixations were determined. The location and variance were used as a baseline to compare the
foveal fixation with the eccentric fixation. Afterward, a 6° central scotoma was simulated, which corresponded to the diameter of scotoma under which the peripheral locus of fixation was developed. In this block, the location and variance of the subject’s locus of fixation were determined. The location and variance were used as a baseline to compare the peripheral locus of fixations under simulation of progression. The progression of the central scotoma was studied in a series of three separate measurements. In each measurement, the diameter of the central scotoma was increased. The first progression type studied was a gradual progression. In this progression, the increase in scotoma size was small between the sessions, such that the already developed PRL was not completely covered by the increased scotoma. The diameters of scotoma increased between sessions by small steps to 8°, 10°, and 11° of visual angle. Subsequently, an abrupt progression of scotoma was simulated. The diameter of this scotoma was adjusted in one large step individually to cover the space in the visual field used by the peripheral locus of fixation. The timing between the experimental blocks depended on the availability of subjects but was always at least 24 hr between. For example, between baseline and gradual progression, subjects had to wait at least 24 hr. The time duration from start to end of simulations varied between subjects according to their availability to participate. The shortest time needed for the performance of the complete experiment was 4 days whereas the longest was 7 days.

The time interval between the experimental blocks also depended on the subjects. Some subjects were able to continue shortly after the end of one experimental block whereas others needed longer breaks. The maximum break time given was 20 min.

Stimuli and procedure

All experiments were performed in a darkened room. The central scotoma simulation consisted of a foveally presented circular scotoma, which blocked 100% of the image on the background. The diameter of the scotoma varied according to the experimental block. In addition, a gaze-independent compound stimulus was presented. A visual task was assigned to the compound stimulus, requiring fixation of a prolonged duration. The subjects had to report specific properties of the compound stimulus with key presses. After response, a new compound target appeared at a different location, requiring relocation of gaze, again followed by fixation. The number of components of the compound stimulus as well as its appearance and associated task varied according to the block of simulation. The size of the components and of the stimulus were selected to be above the perceptual threshold; thus perception was not limited at different eccentricities from the fovea. The arrangement of the components was designed to increase the fixation time. Overall, the distribution of the components spanned 1.5°. The main task during the simulation was to discriminate either the color or the shape of the components of the stimulus. The variation of task (discrimination of color or shape) was introduced to avoid monotonous single tasks and, thus, to keep the subjects challenged and motivated.

The background luminance of the screen and the circular scotoma was 64 cd/m², and their color was identical (dark gray). The outline of the scotoma was drawn to help subjects to orient their saccades. The main task was to discriminate the components of a stimulus that was presented at varying screen positions.

A 13-point calibration was performed at the beginning of each training block. This calibration collected fixation samples from 13 known target points in order to map raw eye-position data to gaze position. Subsequently, a validation with 13 points was performed to provide information about the calibration accuracy. The experiments continued only if the validation was confirmed to be good by the eye tracker. The calibration of the Eyelink eye tracker is categorized as good when minimal nonlinearity exists when fixating different target positions (maximum ratio of gains = 1.5:1 horizontally, 3:1 vertically).

Figure 2 shows examples of the procedure followed in the study. The experiment consisted of a Baseline 1, a Baseline 2, a gradual progression of scotoma, and an abrupt progression of scotoma. Baseline 1 and Baseline 2 were performed to assess the foveal and peripheral fixation of the subjects. Each gray box represents an experimental block. Each experimental block corresponded to a 10-min collection of eye-movement data.
with a constant scotoma size. All changes of scotoma size were performed in between experimental blocks, not during the data acquisition. For example, the simulation of the 8° scotoma was performed in two 10-min blocks; subsequently, the simulation of the 10° scotoma was performed in two 10-min measurement blocks.

**Baseline 1: Foveal fixation**

Subjects performed a discrimination task without scotoma. To increase the fixation time, the stimulus consisted of four components that were assigned randomly to be either lines or squares. Subjects used the space key to mark the squares red and used the up and down arrow keys to report whether the components were two lines and two squares or whether the components had a different arrangement, for example, only one line and three squares.

**Baseline 2: Fixation under developed peripheral locus of fixation**

Subjects performed two visual tasks under simulation of central scotoma. The diameter of the scotoma was 6° of visual angle, which corresponded to the diameter of the scotoma used on the locus of fixation training. The tasks were separated into two measurements. In the first measurement, the stimulus consisted of three components that were assigned randomly to be either two red discs and a blue disc or two blue discs and a red disc. Subjects used the up and down arrow keys to report whether there were more red or blue discs. In the second measurement, the stimulus consisted of four components. Three of the components were red or blue discs and the fourth was a cyan disc. Subjects used the up and down arrow keys to report whether there were more red or blue discs.

**Gradual progression of scotoma**

Subjects performed six visual tasks under simulation of central scotoma. In first and second tasks, the diameter of the scotoma was 8° of visual angle, and the stimulus consisted of five and six components, respectively. The stimulus with five components consisted of a combination of discs colored red, blue, cyan, and magenta. The stimulus with six components consisted of a combination of discs colored red, blue, cyan, magenta, and green. Subjects used the up and down arrow keys to report whether there were more red or blue discs.

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**Figure 2.** Example of procedure and stimuli, ordered from left to right according to their occurrence in the measurement series. In Baseline 1, subjects performed a visual task in the absence of the scotoma. In Baseline 2, subjects performed two visual tasks with the scotoma in which the peripheral locus of fixation was developed. The simulation of gradual progression was performed in three different steps. In each step, the diameter of the scotoma was increased. The three scotoma diameters were 8°, 10°, and 11° of visual angle. Finally, an abrupt progression of scotoma was simulated. In this simulation, the diameter of scotoma was adjusted individually and was determined by the span of the peripheral locus of fixation over the visual field. The diameter selected covered the area used for fixation completely. Eye-position data were collected for 10 min.
In the third and fourth tasks, the diameter of the scotoma was 10° of visual angle, and the stimulus consisted of two and three components, respectively. The stimulus was a combination of squares and lines. In the visual task with a stimulus of two components, the components were a combination of two lines, two squares, or a square and a line. Subjects reported whether the components were equal or different. In case they were equal (e.g., two squares), subjects used the space key to mark both components red and reported that they were equal using the up arrow key. In case they were different (a square and a line), subjects used the space key to mark only the square red and reported that they were different using the down arrow key. In the visual task with a stimulus of three components, the components were a combination of squares and lines. Subjects used the space key to mark all squares red and to report whether there were more squares or lines. They used the up arrow key to report more squares and the down arrow key to report more lines.

In the fifth and sixth tasks, the diameter of the scotoma was 11° of visual angle, and the stimulus consisted of four and five components, respectively. In both tasks, the stimulus was a combination of squares and lines. In the visual task with a stimulus of four components, subjects used the space key to mark all squares red and reported whether the components were two lines and two squares or the components had a different arrangement, for example, only one line and three squares. Finally, in the sixth visual task, subjects used the space key to mark all squares red and reported whether there were more squares or lines. They used the up arrow key to report more squares and the down arrow key to report more lines.

Data analysis

Eye-movement data

The fixations were separated from other events (blinks and saccades) using the Eyelink parsing algorithm. The algorithm classified fixations, saccades, and blinks using a saccadic velocity threshold of 30°/s, a saccadic acceleration threshold of 8000°/s², and a saccadic motion threshold of 0.1° (Lingnau, Schwarzbach, & Vorberg, 2008; Van der Stigchel et al., 2013; Bethlehem et al., 2014; Smith, Glen, Monter, & Crabb, 2014; Liu & Kwon, 2016). If the eye movement was below the velocity and acceleration thresholds criteria, then it was categorized as a fixation eye movement; otherwise, it was categorized as a saccade eye movement. In case it was considered a saccade, only the eye-movement data before and after the thresholds criteria were considered for the locus of fixation analysis.

Location of the retinal locus of fixation

The location of the peripheral locus of fixation was obtained from the kernel density estimation (Botev,
Figure 4. Fixation maps and locations of the peripheral retinal loci for gradual and abrupt progression of the scotoma. The colors of each fixation map represent the fixation density, where red colors represent high fixation densities and blue colors represent lower fixation densities. Baseline 1 shows the central fixations that subjects performed when they underwent a visual task without scotoma.
Baseline 2 shows the fixation maps and retinal loci of each subject. The diameter of the scotoma was 6° of visual angle and corresponded to the diameter used for the retinal locus training. The gradual progression shows the retinal loci of each subject when 8°, 10°, and 11° diameter scotomas were simulated. All subjects maintained the location in which the baseline retinal locus was located. The same is observed in the abrupt progression. The angular locations for the baseline (BL) and progression (P) retinal loci are summarized in the location block.

Grotowski, & Kroese, 2010) of the fixations and was defined to be at the peak density (Kwon et al., 2012), which corresponded to the retinal position with the highest probability at which to locate the center of a stimulus.

The variance of the retinal locus of fixation

The variance of the fixations was used as a measure of fixation stability and as a consequence of retinal locus of fixation quality. It was obtained by calculating the BCEA of the fixations (Steinman, 1965; Crossland, Sims, Galbraith, & Rubin, 2004) that encompassed 68% of fixations around the mean (Castet & Crossland, 2012; Kwon et al., 2012; Liu & Kwon, 2016). Small BCEAs corresponded to smaller fixation areas and, therefore, higher fixation stability.

Results

The location of the peripheral retinal locus on gradual and abrupt progression of the scotoma

Figure 4 shows the fixation maps of the five subjects at every experimental block and a summary of the location of their peripheral retinal locus for baseline PRL, gradual progression, and abrupt progression. From top to bottom, Baseline 1 shows the fixations collected when subjects performed a visual task without the scotoma. Baseline 2 shows the baseline retinal locus of every subject after the performance of the two visual tasks. The 6° disc represents the simulated central scotoma. The fixation maps show how all subjects had a peripheral retinal locus of fixation. The location and variance of these retinal loci were used as a baseline to compare the retinal loci showed on the simulation of progression. The gradual progression block shows the fixation maps for the subjects when a central scotoma of 8°, 10°, and 11° of visual angle was simulated. Every subject located the retinal locus on the same meridian as the baseline retinal locus along the simulation of gradual progression. In the abrupt progression block, when the diameter of the scotoma was increased to values larger than 20° of visual angle, subjects again located the retinal locus on the same meridian. The location block summarizes the locations used during the performance of the experiment. Each diagram includes a series of rings that represent every scotoma diameter tested. A red line connects all locations collected for each stage of the experiment. BL corresponds to the location used on Baseline 2. The points P1, P2, and P3 show the locations used on the simulation of the gradual progression of the scotoma. P1 represents the location when the 8° scotoma was simulated, P2 when the 10° scotoma was simulated, and P3 when the 11° scotoma was simulated. AP corresponds to the location used during the abrupt progression simulation. In addition, the angular location of the baseline retinal locus (BL) together with the angular mean and standard deviation of the simulation of progressive scotoma (P) are given for each subject. Subjects located the retinal locus on the same meridian, independently of the progression simulated (gradual or abrupt). These results suggest that once retinal locus has developed, the fixation technique used to perform a visual task is likely to be used over time if the scotoma progresses.

Variance of the retinal locus

The variance was calculated using the BCEA that encompassed 68% of fixations around the mean. Figure 5 shows the mean BCEA of all subjects as a function of the experimental stage. NS refers to the stage in which subjects performed the visual task without scotoma. At this stage, the mean and standard deviation of the BCEA was 23.6 ± 4.9°². This relatively large value may be attributed to the size of the stimulus, which was 1.5° × 1.5°. BL refers to the baseline retinal locus measurement. Mean and standard deviation of the baseline BCEA was 52.3 ± 10.0°². P1, P2, and P3 correspond to the gradual simulation of progression, and the means and standard deviations of the BCEAs obtained at these stages were 46.4 ± 12.6°², 39.5 ± 11.3°², and 34.5 ± 11.7°², respectively. Finally, AP corresponds to the abrupt scotoma progression; mean and standard deviation of the BCEA obtained was 111.7 ± 17.6°².

We found a significant increase between the NS BCEAs and the BL BCEAs (paired-sample t test), t(4)
This showed that fixation is less stable under a scotoma simulation. No significant differences were found between the BL BCEAs and the P1, P2, and P3 BCEAs (paired-sample t test), \( t(4) = 2.2, p = 0.09 \); between baseline and P2, \( t(4) = 1.7, p = 0.15 \); and between baseline and P3, \( t(4) = 2.7, p = 0.052 \); however, significant differences were found between the BL BCEAs and the AP BCEAs (paired-sample t test), \( t(4) = 9.9, p = 0.0005 \). This suggests that the fixation stability of the PRL may be a factor that depends on the progression type as well as the size of the scotoma.

Discussion

Location of the peripheral locus of fixation

The retinal loci of every subject were located along the same meridian, independently of the progression simulated (gradual or abrupt). These results provide a first analysis of retinal locus reselection under a simulated scotoma paradigm and suggest that once peripheral retinal loci are developed, the same meridian of the retinal locus may be kept when the scotoma progresses. Similar maintenance of PRL location along the progression of the scotoma was also shown by Sunness & Applegate (2005). Sunness used patients with different types of low vision to show that they consistently located the PRL on the same meridian over a follow-up period longer than 4 years.

The selection and consistent relocation of the retinal locus on the same meridian may be attributed to a subject’s oculomotor predisposition to perform the saccades to a specific position under occlusion of foveal vision. Such kind of predisposition may be refined by means of training and may lead to the discouragement of otherwise directed saccades. Although there is no evidence for such kind of predisposition, the results showed that once a location was preferred, the saccades were mainly directed to the specific position. Alternatively, this reselection of retinal location on the same meridian may also be a consequence of a subject’s ability to deploy their attention on some locations of the visual field rather than others. Barraza-Bernal et al., 2016 and Barraza-Bernal, Ivanov, Nill, Rifai, Trauzettel Klosinski, & Wahl, 2017 showed that subjects under simulation of central scotoma tend to develop a peripheral retinal locus of fixation on locations with good attentional performance and that the attentional performance patterns are preserved across the retina.
The location of the locus of fixation was, in most of the cases, adjacent to the edge of the scotoma, independently of the stage of simulated progression. This kind of proximity to scotoma edge was shown in several studies with patients and subjects. Fletcher & Schuchard (1997) showed that in 88.7% of patients with different kind of maculopathies, the PRLs were within 2.5° from the border of the scotoma, and Sunness et al. (1996) showed that the PRLs of 27 eyes with dry age-related macular degeneration were always within 2° from the scotoma. Moreover, Kwon et al. (2013) showed that subjects under simulation of central scotoma developed a peripheral retinal locus of fixation at the proximity of the simulated scotoma. Retinal loci at the border of the scotoma can be explained by the retinotopy-driven hypothesis for the selection of the PRL (Cheung & Legge, 2005). The hypothesis suggests that the PRL selection might be a consequence of retinotopic reorganizations, where deafferented V1 neurons spontaneously remap to the inputs from retinal locations near the scotoma. As a consequence, the location of the PRL is predicted at the edge of the scotoma.

**Fixation stability**

When the retinal damage is absolute, PRLs will develop on retinal areas that are still functioning and that are outside of the absolute damaged retina. As a consequence, the larger the absolute damage, the higher may be the distance between PRL and fovea. In normally sighted individuals, the fixation stability worsens as the distance between targets and fovea increases (Sansbury, Skavenski, Haddad, & Steinman, 1973). In cases of dense central scotomas, patients bring the PRL to retinal areas free of damage located at eccentric distances from the fovea; accordingly fixation stability is expected to be decreased. Thus it can be assumed that the fixation stability of the PRL may also worsen with the progression of the scotoma. However, previous studies showed that the fixation stability is significantly impaired in patients with maculopathies, but the impairment was not associated to visual acuity, contrast sensitivity, or size of the scotoma (Culham, Fitzke, Timberlake, & Marshall, 1993; Schuchard & Fletcher, 1994; Rohrschneider, Becker, Kruste, Fendrich, & Völcker, 1995). Nevertheless, in this study, the variance (BCEA) of the fixations for subjects under simulation of abrupt scotoma progression increased significantly. This drop in fixation stability might originate in the large distance of the stimulus relative from the fovea or to the fact that the subjects cannot use former fixation locations and suggests that the changes in fixational stability may be associated with the stage of the progression.

The differences in fixation stability between previous studies and our study may be originated from the differences between participants. Patients with a macular disease have a less defined edge between scotoma and healthy retina whereas our subjects had a clearly defined, visible scotoma border. Crossland, Culham, & Rubin (2004) measured patients with maculopathy acquired by either age-related or juvenile macular degeneration and found no relationship between the scotoma size and the fixation stability. However, they measured the size of the dense scotoma, leaving unknown the size of retinal areas that are partially damaged. Thus they suggested that the nature of the variation in fixation stability may be related to the extent of the relative scotoma.

We also found that the fixation stability between the baseline and the retinal loci obtained at the gradual simulation of scotoma progression did not vary significantly. This supported that the changes in fixation stability may be significant only under abrupt progression. In addition, we showed a slight decrease in BCEA during performance of the gradual progression simulation. Heinen & Skavenski (1992) studied oculomotor adaptation using the BCEA in three adult monkeys after appearance of a scotoma. They found a slide improvement in fixation stability to foveal loss after a sequence of measurements as well. However, the improvements presented in our study may also be attributed to training effects.

Furthermore, we compared the fixation stability of the foveal fixations with the fixation stability of the baseline retinal locus developed under a 6° scotoma simulation and found that the fixation stability decreased significantly. Similar differences in fixation stabilities between normally sighted subjects and patients were described in previous studies (White & Bedell, 1990; Bellmann, Feely, Crossland, Kabanarou, & Rubin, 2004). The only difference in our study was that the BCEAs were approximately 10 times higher in patients than in normal subjects whereas in our study the BCEA increased by a factor of roughly two. Kwon et al. (2013) simulated visible and invisible scotomas and found that participants showed an eccentric fixation variance that was significantly lower when the scotoma boundaries were presented. But, apart from differences in the variances, the behavior of the two groups was comparable. In our study, the relatively low difference between the BCEAs obtained in the foveal fixations and in the eccentric fixations might be explained by the previous training that the subjects underwent.

The conclusions made in this manuscript are from a data set of five normally sighted subjects that underwent simulations of scotoma progression. The use of these kind of simulations have shown that normally sighted subjects can adopt spontaneously and rapidly a locus for fixation (Kwon et al., 2013). However,
differences between these subjects and real patients must be acknowledged. Clinical observations of patients suffering from central scotomas suggest slow adaptations to eccentric fixation (Crossland et al., 2005) whereas in this simulation the adaptation occurs faster. Furthermore, the rate at which the scotoma normally progresses is much slower than the simulated one. Moreover, scotoma simulation reveals scotoma boundaries, which may be used as an oculomotor reference to redirect saccades. Thus perception is different from the perception of patients because only in some cases of central vision loss are the scotoma boundaries perceived. Thus the conclusions drawn from the data presented cannot be fully generalized to all clinical cases.

Conclusion

The location and fixation stability of the peripheral retinal loci were studied in five normally sighted subjects who underwent a simulation of progressive central scotoma. Subjects located the retinal locus along the same meridian under simulation of progression. This result suggests that the fixation technique used to perform a visual task is unlikely to be changed if the scotoma progresses. Given the importance of the PRL location on the performance of a visual task, the results may help in the development of training techniques for the visually impaired.

Furthermore, we found differences in fixation stabilities depending on the type of simulated progression. The fixation stabilities between the baseline and gradual progression were not significantly different whereas the fixation stabilities between the baseline and abrupt progression decreased significantly. These results suggest that the fixation stability of subjects decreases with the size of the scotoma. Low fixation stabilities may imply more eye movements, which may lead to a lower efficiency on the performance of the visual tasks. Thus these results may be of significance for the development of training techniques that aim to improve the efficiency in the performance of a visual task.

Keywords: preferred retinal locus of fixation, simulation of central scotoma, progressive central vision loss

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