Altered Eye Movements During Reading With Simulated Central and Peripheral Visual Field Defects

Haojue Yu and MiYoung Kwon

Department of Psychology, Northeastern University, Boston, Massachusetts, United States

Correspondence: MiYoung Kwon, Department of Psychology, Northeastern University, 125 Nightingale Hall, 360 Huntington Ave., Boston, MA 02115, USA; m.kwon@northeastern.edu.

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Purpose. Although foveal vision provides fine spatial information, parafoveal and peripheral vision are also known to be important for efficient reading behaviors. Here we systematically investigate how different types and sizes of visual field defects affect the way visual information is acquired via eye movements during reading.

Methods. Using gaze-contingent displays, simulated scotomas were induced in 24 adults with normal or corrected-to-normal vision during a reading task. The study design included peripheral and central scotomas of varying sizes (aperture or scotoma size of 2°, 4°, 6°, 8°, and 10°) and no-scotoma conditions. Eye movements (e.g., forward/backward saccades, fixations, microsaccades) were plotted as a function of either the aperture or scotoma size, and their relationships were characterized by the best fitting model.

Results. When the aperture size of the peripheral scotoma decreased below 6° (11 visible letters), there were significant decreases in saccade amplitude and velocity, as well as substantial increases in fixation duration and the number of fixations. Its dependency on the aperture size is best characterized by an exponential decay or growth function in log-linear coordinates. However, saccade amplitude and velocity, fixation duration, and forward/regressive saccades increased more or less linearly with increasing central scotoma size in log-linear coordinates.

Conclusions. Our results showed differential impacts of central and peripheral vision loss on reading behaviors while lending further support for the importance of foveal and parafoveal vision in reading. These apparently deviated oculomotor behaviors may in part reflect optimal reading strategies to compensate for the loss of visual information.

Keywords: eye movements, reading, visual field defects, simulated scotoma, gaze contingent display

Contrary to our perceptual impression, human vision is not homogenous across the visual field. Different regions of the visual field serve different purposes. For resolving fine spatial detail, humans rely exclusively on the fovea, the small center-most region of the retina, where light-sensitive cells are densely packed because this central 1° to 2° region of the visual field provides high-acuity vision (e.g., ≥20/20 Snellen acuity).[1–3] For this reason, we make a series of ballistic eye movements, *saccades* interleaved with *fixations,* to continuously bring a target of interest onto the fovea for processing fine-scale information.[4] Outside the fovea, visibility progressively declines with eccentricity. The parafoveal region covers the central 5° visual field (>20/40)[2] and offers coarse yet crucial spatial information for previewing that helps guide spatial attention and upcoming eye movements.[5–11] Besides these regions, the perifovea covering the central 8° visual field (>20/60)[2] and peripheral vision (<20/60)[2] helps in exploring a broader context in our surroundings.[12] This nonuniform visibility is mostly attributable to variations across the retina in the sampling density of the cone photoreceptor mosaic/ganglion cells[1,13] and in other optical/cortical properties[14] such as chromatic aberrations,[15,16] cortical magnification,[17,18] or visual crowding (i.e., inability to recognize targets in clutter).[19–21]

Several decades of research on the fundamental visual requirements for reading[22–29] have made clear that different patterns of visual field loss bring about different degrees of impairment in reading ability. Although most visual information for reading (e.g., detailed visual features such as line segments and curvatures that mediate letter or word recognition)[25,30–32] is obtained through foveal vision,[33] the parafovea is also known to play a vital role in efficient reading behaviors including previewing upcoming words, optimal saccade planning, and facilitating subsequent foveal processing.[5,6,8,9,11,34] In particular, parafoveal processing has been shown to be closely coupled with covert attention, which is oriented as a function of reading direction.[9,35] For instance, a vast collection of literature on reading performance has shown that skilled readers of alphabetic writing systems obtain letter information extending three to four letters to the left of fixation and 14 to 15 letters to the right of fixation,[29,33,36,37] supporting the role of attention in parafoveal processing.[11,35] On the other hand, peripheral vision has also been shown to be important for navigating text (e.g., changing to the next line of text during reading).[38] Thus it is not surprising that when the required field of view is not met, reading performance declines, and reading speed becomes noticeably slower.[39] Clinical research

with low-vision populations provides further support that reading rate is modulated by the location and shape of the visual field defect as shown in patients with AMD,[40–43] glaucoma,[44–47] and RP.[48]

When we read, our eyes make a series of distinctive eye movements such as forward saccades, regressive (backward) saccades, and fixations.[4,49] Although much less studied, the role of microsaccades during reading has also been reported in previous work.[50] Microsaccades (i.e., small involuntary eye movements produced during fixation) are known to prevent visual fading[4,51,52] and to sample high spatial frequency information, thereby enhancing the processing of fine spatial detail.[53] This fine oculomotor behavior appears to be beneficial in reading by enhancing visibility of nearby words.[50]

Empirical evidence from both clinical populations and healthy individuals with simulated vision loss have shown that decreased reading performance often covaries with noticeable changes in the pattern of eye movements.[54–56] For example, AMD patients with central vision loss have a saccade amplitude that tends to be much smaller than that of normal controls during reading (i.e.,1.62 vs. 6.0 letters per forward saccade)[57] in addition to more frequent regressive saccades.[40] Furthermore, patients with advanced bilateral glaucomatous visual field defects tend to make more saccades during reading compared to normal cohorts.[58] Thus it is apparent that perceptual visibility and eye movements are closely intertwined,[59,60] especially for fine scale visual function like reading. Previous research on clinical, as well as normal, populations have demonstrated the significant role of visual information obtained from different parts of the visual field in reading.[39–41,44,48] However, none has systematically studied how different types and sizes of visual field defects affect the way visual information is acquired via eye movements during reading. In particular, it remains unclear how much visual field loss (and which part of the field) should occur before the pattern of eye movements substantially deviate from that of the full-field (intact) viewing. Moreover, it is largely unknown whether different regions of the visual field (i.e., the foveal, parafoveal, perifoveal and peripheral regions) would have differential impacts on the key reading eye movements shown to be important for the reading process.[50,61] Perhaps we can speculate that depriving the perifoveal and peripheral regions of the visual field may not lead to any significant change in the pattern of saccade amplitude or fixation duration mostly involved in foveal processing and parafoveal previewing. On the other hand, they may be more affected by the loss of the parafoveal region. Thus we still have important questions to be answered to better understand the visual field requirement for reading behaviors.

The current study aims to investigate how visual field defects, one of the most common symptoms that accompanies AMD, glaucoma, or retinitis pigmentosa, would alter the aforementioned key eye movement patterns. To this end, gaze-contingent visual displays were used to simulate central and peripheral scotomas with varying sizes (i.e., scotoma or aperture size of 2°, 4°, 6°, 8°, and 10° visual angle corresponding to foveal, parafoveal, perifoveal, and peripheral regions of the visual field, respectively) in neurotypical adults with normal vision. A person's reading speed was assessed binocularly while his/her eye movements were continuously tracked using a high-speed eye tracker (i.e., monocular tracking with the dominant eye, see Methods for details). By using simulated scotomas in a cohort of young healthy adults with normal vision, we hope to minimize potential confounders such as age-related oculomotor deficits or other pathological factors. It should also be noted that our study design did not allow for any long-term adaptation (a period of several hours to years) to simulated scotomas that could induce changes in oculomotor behavior.[62,63] Therefore the current study is designed to examine the human oculomotor system's immediate and spontaneous response to simulated scotoma as it maximizes the information uptake required for reading. The outcome from the current study is expected to help us better characterize the relationship between reading eye movements and visual field loss, as well as provide insights into designing effective reading rehabilitations and aids for individuals with visual field loss.

Methods

Participants

A total of 24 normally-sighted subjects (age range 18–23 years, mean age 19.62 ± 0.22 years, nine males) participated in the study. All subjects were recruited from Northeastern University. They were native English speakers without any known visual disorders or any known cognitive or neurological impairments. They had normal ocular health (i.e., no AMD, glaucoma, or diabetic eye disease in either eye) and had normal or corrected-to-normal vision. Normal vision was defined as having better than or equal to 0.1 logMAR (equivalent to 20/25 Snellen acuity) visual acuity, normal contrast sensitivity (better than 1.9 log units), and normal stereoacuity (40–45 arcsec). Mean visual acuity (Early Treatment Diabetic Retinopathy Study Chart) was −0.19 ± 0.06 logMAR (i.e., better than 20/15). Mean contrast sensitivity (Pelli-Robson Contrast Sensitivity Chart) was 1.96 ± 0.02 log units. Mean stereoacuity (Titmus Fly SO-001 StereoTest) was 45″ ± 3.01″ (arcsec). All measurements including the main experiment were performed binocularly. The experimental protocols followed the tenets of the Declaration of Helsinki and were approved by the Institutional Review Board at Northeastern University. Written informed consent was obtained from all subjects before the experiment and after an explanation of the nature of the study.

Stimulus and Apparatus

The 26 lowercase Courier New font letters of the English alphabet—a serif font with fixed width and normal spacing—were used for the reading task. The letters were black on a uniform gray background with a contrast of 100%. Letter size was defined as the font's x-height of 0.5° at the 57 cm viewing distance. As shown in Figure 1C, a paragraph of text consisting of five sentences was presented on a display screen at a time (hereafter referred to as a *text page*). All the sentences were 56 characters (including spacing) in length and formatted into one line. The horizontal end-to-end distance of each sentence spanned an approximate 32° visual angle. For each subject, a total of 28 text pages were used for the reading task. The presented sentence had a reading difficulty ranging between second- and fourth-grade levels. These simple and standardized sentences were chosen to minimize the influences of higher-level cognitive and linguistic factors, thereby assessing the front-end visual aspects of reading.[25,64–66] The same set of sentences was also used in our previously published work.[56]

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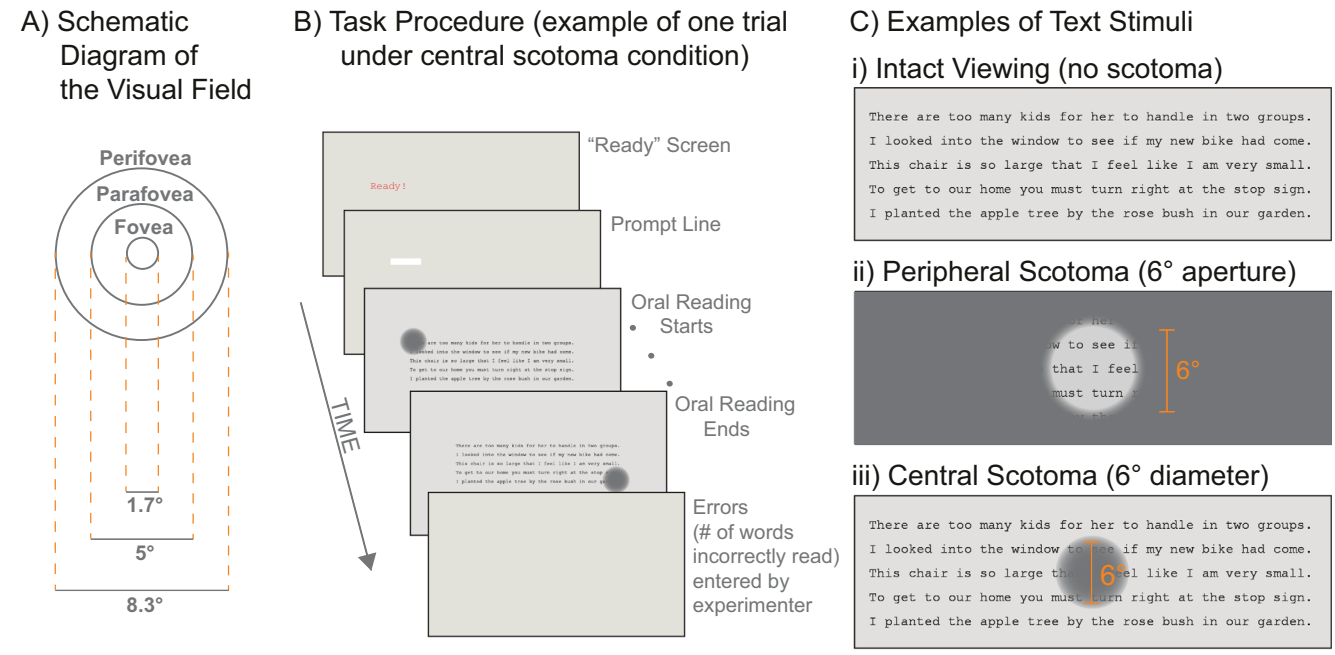

Figure 1. **(A)** Schematic diagram of the visual field. The approximate size of each sub-region of the visual field (fovea, parafovea, and perifovea) in degree units was given.[75] **(B)** Task procedure. The sequence of one trial under a central scotoma condition was shown as an example. **(C)** Examples of text stimuli. **(i)** Five sentences within one text page under intact viewing (no scotoma) condition. **(ii)** Text page with simulated peripheral scotoma with a 6° aperture. **(iii)** Text page with simulated central scotoma with a 6° diameter. **(D)** Peripheral scotoma with five aperture sizes in degree units and the corresponding number of letters visible. **(E)** Central scotoma with five scotoma sizes in degree units and the corresponding number of letters masked. The orange bars in each example indicate the diameter of the central scotoma or the aperture of the peripheral scotoma (in degree units). For ease of visibility in the figure, the luminance of the scotoma is rendered two times darker than the original luminance.

Eye Movement Recording and Simulated Scotoma

Subjects' gaze positions were monitored (binocular tracking) using an infrared video-based eye-tracker with a sampling rate of 500 Hz (EyeLink 1000 Plus/Desktop Mount, SR Research Ltd., Ontario, Canada) and a maximum spatial resolution of 0.01°. A nine-point calibration/validation sequence was performed at the beginning of every experimental condition. Calibration and validation were repeated until the validation errors for all points were smaller than 0.5°. The gaze position error (i.e., the difference between the target position and the computed gaze position) was estimated during the nine-point validation process. The average gaze position error for the current study was 0.2° (± 0.1°).

Forehead rests were used throughout the experiment to minimize head movements and trial-to-trial variability in the estimation of gaze position. A real-time gaze position was sent to the display computer through a high-speed Ethernet link. Continuous gaze information was used to draw an artificial scotoma on the display screen at a refresh rate of 60 Hz where the gaze position corresponded to the center of the scotoma. Although the reading task was binocular, scotomas were induced using the gaze position of each subject's dominant eye. It has been shown that during reading, the eyes

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move more or less in synchrony with the movement of each eye beginning/ending in close temporal approximation of each other.[4,67,68] Thus tracking with the dominant eye has been shown to effectively induce scotomas in normal vision.[63,69–72]

As illustrated in Figures 1D and 1E, our study design included two scotoma types: (i) peripheral scotoma (aperture size of 2°, 4°, 6°, 8°, and 10°); (ii) central scotoma (diameter size of 2°, 4°, 6°, 8°, and 10°). The simulated peripheral scotoma with a luminance of 32 cd/m^2 masked the rest of the visual field except for a small circular aperture. The simulated central scotoma was a circular disc and was rendered as a uniform gray patch (luminance 32 cd/m^2) on the screen. The edges of the scotomas were smoothed with a Gaussian filter ($\sigma = 10$ pixels, corresponding to 0.3°). The luminance of the text background was set at 45 cd/m^2.

It is noteworthy that given the letter size of 0.5° used in the current study, the aperture or central scotoma size of 2°, 4°, 6°, 8°, and 10° corresponded to approximately 3.5, 7, 11, 14, and 18 letters. The number of visible (or masked) letters was computed by dividing the size of aperture (or central scotoma) by 0.565° i.e., a sum of the letter size (0.5°) and interletter spacing (0.065°) used for our reading text.

Central and peripheral scotoma conditions were tested independently in separate sessions. Thus each session contained six scotoma conditions: five sizes of either central or peripheral scotoma and one no-scotoma as a baseline intact viewing condition. Each scotoma condition was tested twice within a session in a random order. However, the order of sessions—central versus peripheral scotoma—were counterbalanced across subjects to minimize any potential confounding effects (if any) such as practice, learning, or fatigue effects.

All stimuli were generated and controlled using MATLAB (version 9.10.0; MathWorks, Inc., Natick, MA, USA) and Psychophysics Toolbox extensions[73,74] for Windows 10 Pro, running on a PC desktop computer (Dell Precision Tower 5810 X-Series; Dell, Inc., Round Rock, TX, USA). Stimuli were presented on a liquid crystal display monitor (Asus VG278Q; ASUS Computer International, Fremont, CA, USA) with a refresh rate of 60 Hz and resolution of 1920 × 1080, subtending 60° × 34° visual angle at a viewing distance of 57 cm. Luminance of the display monitor was made linear using an 8-bit look-up table in conjunction with photometric readings from a luminance meter (Minolta LS-110 Luminance Meter; Konica Minolta, Inc., Japan).

Task Procedure

Subjects' reading speed was measured with static text consisting of five sentences with the same length on one page (see Fig. 1) while their gaze position was continuously recorded by a high-speed eye tracker (See the section above for details on eye movement recordings). As shown in Figure 1B, subjects were first shown the screen where a short white line was presented in the upper left corner of the screen prompting the subject where the first sentence would appear. They were asked to fixate on this white line while preparing for the reading task. Whenever subjects were ready, they pressed the space bar to initiate the task. Subjects were instructed to read the sentences out loud as quickly and accurately as possible (i.e., oral reading task) as soon as the text page appeared on the display screen. When they completed reading each text page, the experimenter pressed a key on the keyboard to record the end of the reading time and entered the number of words read incorrectly. Subjects were allowed to self-correct errors among previously read words in the same trial, and these self-corrected words were not counted as errors. Thus, for each text page, the time taken to read, as well as reading accuracy (i.e., number of words read correctly), were recorded, and corresponding reading speed (i.e., number of words read correctly per minute [wpm]) was computed.

The study design consisted of two experimental sessions with one short break in between. Each subject was tested for all three different viewing conditions: peripheral scotoma, central scotoma, and intact viewing (no scotoma). For each session, two text pages were used for each scotoma size and four text pages for the intact viewing condition (no scotoma). Therefore, for each subject, a total of 28 (2 scotoma types: central vs. peripheral × 5 scotoma sizes × 2 text pages + 1 no-scotoma type × 4 text pages × 2 sessions) unique text pages (28 reading speed measurements) were used. No subject saw the same text twice. For each subject, the final reading speed for scotoma viewing conditions was the average across the two measurements (i.e., two text pages) and for the intact viewing condition was the average across the four measurements. Subjects performed all tasks in a dimly lit room while they were seated in a comfortable position with a forehead rest. The forehead rest was used to maintain the desired viewing distance, as well as to minimize head motion while allowing subjects to read out loud freely without compromising eye tracking accuracy.

Data Analysis of Eye Movement Measurements

Gaze data were analyzed using the EyeLink parsing algorithm, which robustly classified fixations and saccades, excluding blinks. The saccadic velocity threshold of 30°/s, saccadic acceleration threshold of 8000°/s^2, and saccadic motion threshold of 0.1° were used to distinguish saccades from fixations.[28,46,75–77] Microsaccades were defined as saccades with an amplitude of less than 1° and its velocity exceeding 30°/s.

Data Analysis

We performed a separate one-way repeated measures ANOVA for each eye movement parameter (i.e., saccade amplitude and velocity, fixation duration, number of fixations, microsaccade rate and amplitude, and proportion of regressive saccades). For the peripheral scotoma condition, the aperture size (2°, 4°, 6°, 8°, 10°, and no scotoma) was entered as a within-subject factor. For the central scotoma condition, the diameter size (2°, 4°, 6°, 8°, 10°, and no scotoma) was entered as a within-subject factor. Data from three subjects in the central scotoma condition was excluded because of poor eye tracking performance. Statistical analyses were performed using SPSS software (version 28.0.0.0). Under both scotoma conditions, the relationship between each eye movement parameter was modeled with either exponential, linear, or constant function in log-linear coordinates. The best fitted model was determined as follows: Based on visual inspections of the data, a few candidate models were selected. Then we performed model comparisons to further identify the model that best accounts for the given data with the fewest parameters. These fits were achieved using a simplex search method[78] to search for the optimal fit producing the least squares error. As shown

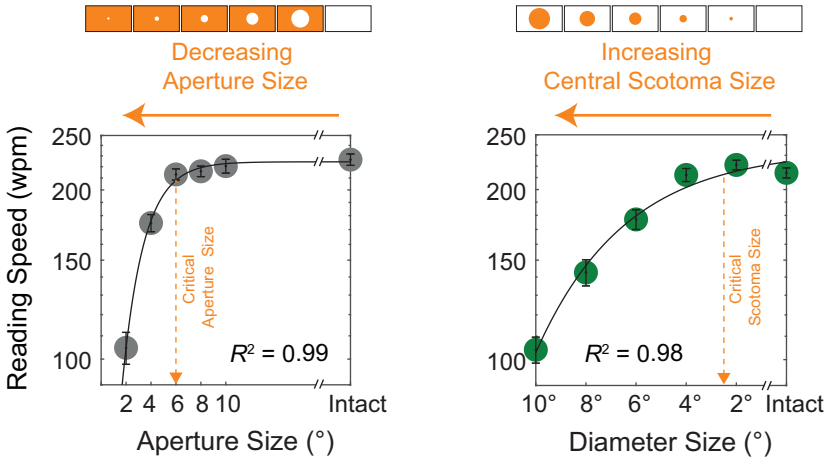

Figure 2. The relationship between reading speed and simulated visual field defects. The left and right panels plot reading speed (wpm) as a function of the size of the peripheral scotoma (i.e., aperture size of 2°, 4°, 6°, 8° and 10°), and the size of the central scotoma (i.e., diameter size of 10°, 8°, 6°, 4° and 2°) in log-linear coordinates, respectively. Each data point is the average value across all subjects in each condition (n = 24 for the peripheral scotoma condition, and n = 21 for the central scotoma condition). The critical aperture (or scotoma) size was shown with *orange dashed arrows*. Note that the sixth level (i.e., the rightmost datapoint on the x-axis) in both plots represents no scotoma (intact viewing) condition. For the peripheral scotoma condition, a visual angle of 33° sufficiently covers the text passage displayed on the screen, and was substituted for the aperture size of no scotoma condition for the sake of model fitting.

in Figures 2 and 4, these fits pass through nearly all data points, demonstrating that our models capture the data well. Furthermore, we performed lack-of-fit tests[79] (see more details in the Results section) to further confirm the fitness of the models indicated by the R^2 (see more details in the Results section). Probability density maps of the landing positions of microsaccades and saccades were derived via density estimation with a bivariate Gaussian kernel,[80] which was also used in our previous work.[56,63,71] Each density map was constructed based on the data from all subjects for each viewing condition.

Results

Changes in Reading Speed Under Simulated Visual Field Defects

Figure 2 summarizes how reading speed (wpm) is modulated by types of visual field defects. Data were best fitted with an exponential decay function in log-linear coordinate as follows:

$$y = ae^{-bx} + c \quad (1)$$

where y is log reading speed, x is the size of scotoma in degree of visual angle (°), a is the scaling factor, b is the decay rate, and c *is* a constant. Solid lines are the best-fitted model. The R^2 values of the best-fitted models are reported in the corresponding plots. The lack-of-fit test[79] uses F statistics to fit the magnitude of the residual error resulting from model fitting against the magnitude of the intrinsic error (or pure error) of a dependent variable resulting from measurements and/or responses. Its null hypothesis ($P > 0.05$) states that the proposed model fits the data well. Therefore $P > 0.05$ observed in the current study supports that our models offer satisfactory descriptions of the data. In Figure 2, the critical aperture (or scotoma) size shown with orange dashed arrows was estimated by finding the x-value (aperture or scotoma size) at which the y-value (reading speed or eye movement metrics) starts to significantly deviate from its asymptotic value. Consistent with previous findings,[40–42,81] we found that reading speed is significantly reduced under both peripheral and central scotoma conditions, which was further confirmed by our statistical analyses.

Peripheral Scotoma Conditions. Our ANOVA analysis showed a significant effect of peripheral scotoma size on reading speed ($F_{(5, 115)} = 117.24$, $P < 0.001$). Post hoc analysis with a Bonferroni correction (hereafter we call it *post hoc analysis* for simplicity) further showed that reading speed with peripheral scotoma with the aperture size of 2° (3.5 visible letters) and 4° (7 visible letters) were significantly different from that of the rest of scotoma sizes, as well as each other ($P < 0.001$). Recall that the number of visible (or masked) letters was computed by dividing the size of aperture (or scotoma) by 0.565° (i.e., a sum of the letter size [0.5°] and interletter spacing [0.065°] used for our reading text).

This relation between reading speed and peripheral scotoma size was also well captured by the best-fitted exponential model (Fig. 2, left panel). Overall, reading speed decreased by 54% from intact viewing (no scotoma) to the most severe peripheral scotoma (2° aperture) (226.44 ± 5.35 wpm vs. 104.73 ± 6.70 wpm, $P < 0.001$). Thus it is apparent that when the peripheral visual processing is disrupted, a visual field of at least 6° (i.e., approximately 11 visible letters) at one fixation needs to be met for a person to achieve his/her maximum reading speed.

Central Scotoma Conditions. Our ANOVA analysis showed a significant effect of central scotoma size on reading speed ($F_{(5, 100)} = 123.77$, $P < 0.001$). Post hoc analysis further showed that reading speed with central scotoma of 6°, 8°, and 10° were significantly different from that of the rest of scotoma sizes and each other ($P < 0.001$). Overall, reading speed decreased by 51% from intact viewing (no scotoma) to the most severe central scotoma condition (10° diameter) (214.08 ± 4.48 wpm vs. 104.09 ± 5.64 wpm, $P < 0.001$). For central visual field defects, a scotoma size exceeding 2.5° (i.e., approximately 4.4 letters) was detrimental to a person's reading speed.

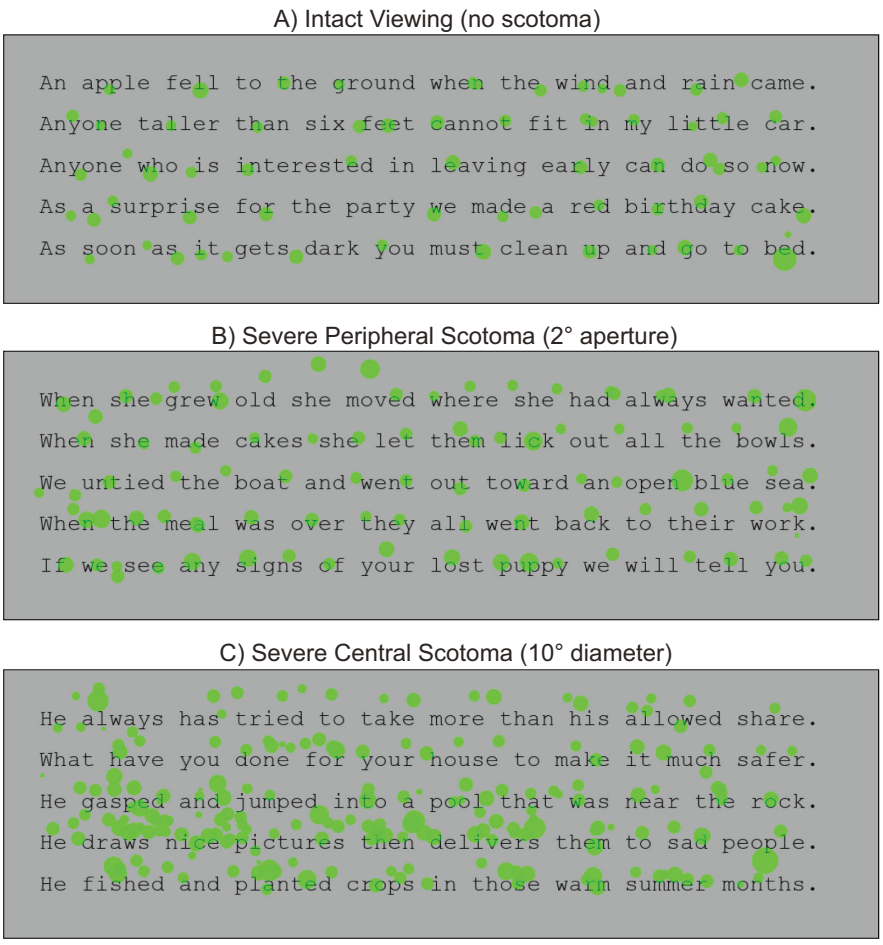

Figure 3. Examples of eye movements during reading under (**A**) Intact viewing (no scotoma), (**B**) severe peripheral scotoma viewing (2° aperture), and (**C**) severe central scotoma viewing (10° diameter). *Green circles* represent a person's fixational eye movements. The radius of the circles indicates fixation duration with bigger circles corresponding to longer fixations. Note that each fixation position of the central scotoma condition represents the center point of the central scotoma as there is no method to accurately define which part of the visual field (or the retina) our subjects used for reading under this condition. Note that for ease of the visibility, the luminance of the text background was made darker than the original one.

Changes in Patterns of Eye Movements Under Simulated Visual Field Defects

Figure 3A visualizes one subject's patterns of eye movements obtained while he/she was engaged in the reading task. It is evident that the number, duration, and location of fixations vary under different viewing conditions, and visual field defects were associated with long and more frequent fixations. Figure 4 shows the relationships between the pattern of eye movements and simulated visual field defects in log-linear coordinates. The Table summarizes ANOVA statistics.

Saccade Amplitude and Velocity

Peripheral Scotoma Conditions.

Saccade Amplitude. Saccade amplitude decreased sharply ($P < 0.001$) in log-linear coordinates as the aperture size decreased below 8° (14 visible letters) (Fig. 4A, left panel). There was a 30% reduction in saccade amplitude (i.e., from 3.27° to 2.29°) from intact viewing to the most severe peripheral scotoma, 2° aperture ($P < 0.001$).

Saccade Velocity. The dependency of saccade velocity on types of visual field defects was similar to that of saccade amplitude (Fig. 4A, left panel). Saccade velocity reached its maximum, 108 ± 2.71°/sec, under intact viewing, but it dropped substantially when the number of letters visible in the central visual field decreased below 8° (14 visible letters).

Central Scotoma Conditions.

Saccade Amplitude. Saccade amplitude increased linearly with increasing central scotoma size in log-linear coordinates (Fig. 4A, right panel). For example, it increased by 98% from intact viewing to the most severe central scotoma (10° diameter) ($P < 0.001$).

Saccade Velocity. Its dependency of scotoma size was similar to that of saccade amplitude (Fig. 4A, right panel). Saccade velocity increased by 22% from intact viewing to the most severe central scotoma ($P < 0.001$).

Fixation Duration and Number of Fixations Per Line

Peripheral Scotoma Conditions.

Fixation Duration. Fixation duration increased sharply when the aperture size decreased below 6° (11 visible letters) ($P < 0.001$) (Fig. 4C, left panel). Fixation duration

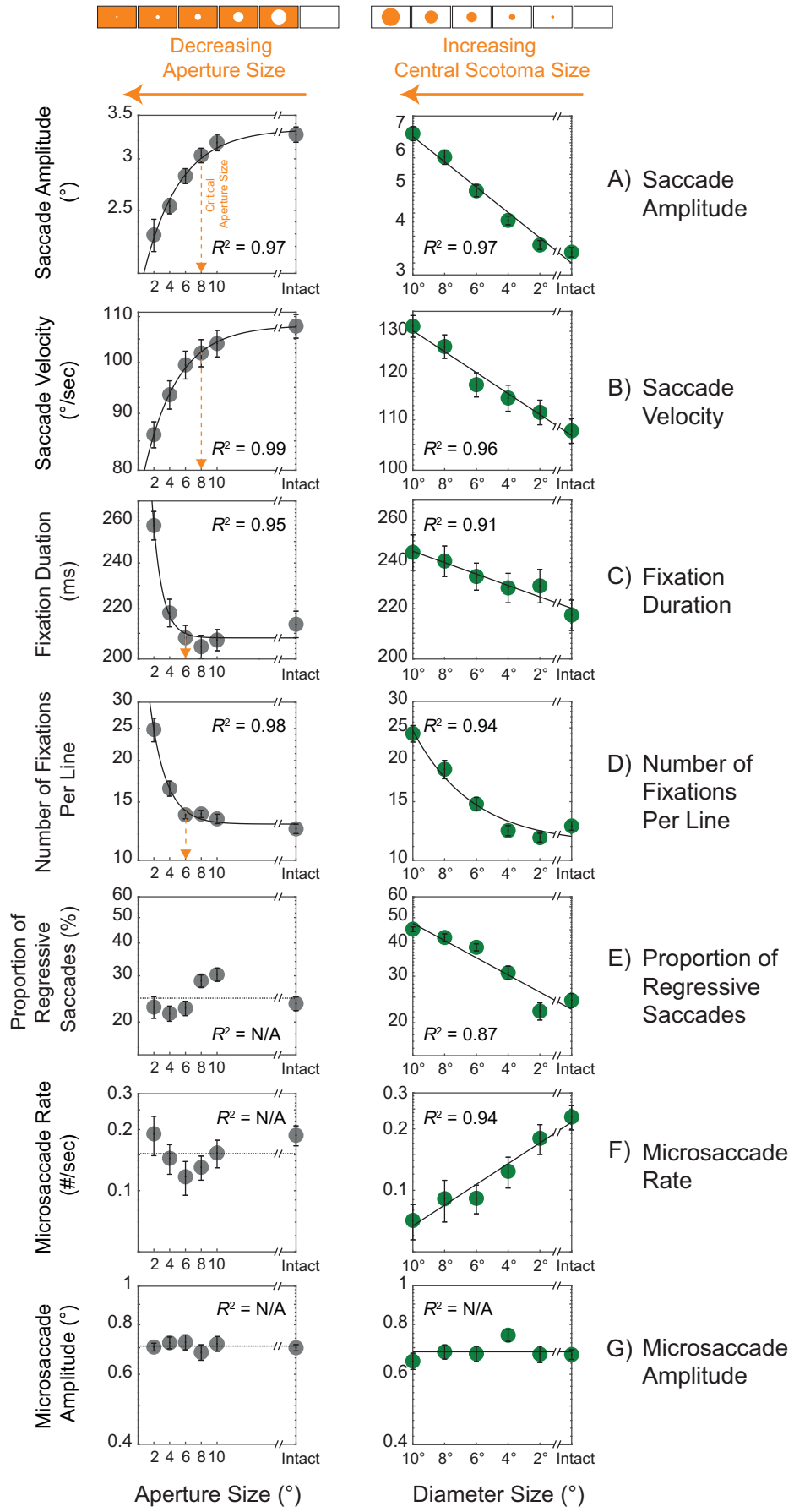

Figure 4. The relationships between the pattern of eye movements and different types of simulated visual field defects. The left and right panels plot each eye movement parameter as a function of the peripheral scotoma size (i.e., aperture size of 2°, 4°, 6°, 8° and 10°), and the

central scotoma size (i.e., diameter size of 10°, 8°, 6°, 4° and 2°) in log-linear coordinates, respectively. Each data point is the average value across all subjects in each condition (n = 24 for the peripheral scotoma condition and n = 21 for the central scotoma condition). Note that the sixth level (i.e., the rightmost datapoint on the x-axis) in both plots represents no scotoma (intact viewing) condition. For the peripheral scotoma condition, a visual angle of 33° that sufficiently covers the text passage displayed on the screen, was substituted for the aperture size of no scotoma condition for the sake of model fitting. (**A**) Saccade amplitude (°). (**B**) Saccade velocity (°/sec). (**C**) Fixation duration (ms). (**D**) Number of fixations per line. (**E**) Proportion of regressive saccades (%). (**F**) Microsaccade rate (no./sec). (**G**) Microsaccade amplitude (°). The *orange panels* on the bottom represent a schematic diagram of changes in aperture or scotoma size. The critical aperture (or scotoma) size was shown with *orange dashed arrows*.

Table. ANOVA Statistics and the Mean Value of Intact Viewing for Each Key Eye Movement

	Peripheral Scotoma		Central Scotoma	
Types of Eye Movements	**ANOVA**	**Mean Value of Intact Viewing***	**ANOVA**	**Mean Value of Intact Viewing***
Saccade Amplitude	$F_{(5, 115)} = 27.08$ ($P < 0.001$)	3.27 ± 0.13 (°)	$F_{(5, 100)} = 98.07$ ($P < 0.001$)	3.19 ± 0.09 (°)
Saccade Velocity	$F_{(5, 115)} = 71.72$ ($P < 0.001$)	107.84 ± 2.71 (°/sec)	$F_{(5, 100)} = 56.34$ ($P < 0.001$)	107.73 ± 2.57 (°/sec)
Fixation Duration	$F_{(5, 115)} = 32.89$ ($P < 0.001$)	213.64 ± 5.49 (ms)	$F_{(5, 100)} = 6.58$ ($P < 0.001$)	217.33 ± 6.41 (ms)
Number of Fixations Per Line	$F_{(5, 115)} = 31.45$ ($P < 0.001$)	12.45 ± 0.42	$F_{(5, 100)} = 61.88$ ($P < 0.001$)	12.68 ± 0.38
Proportion of Regressive Saccades	$F_{(5, 115)} = 8.12$ ($P < 0.001$)	23.51 ± 0.07 (%)	$F_{(5, 100)} = 119.64$ ($P < 0.001$)	24.31 ± 0.15 (%)
Microsaccade Rate	$F_{(5, 115)} = 2.98$ ($P < 0.05$)	0.19 ± 0.02 (#/sec)	$F_{(5, 100)} = 13.14$ ($P < 0.001$)	0.23 ± 0.03 (#/sec)
Microsaccade Amplitude	$F_{(5, 105)} = 0.57$ ($P = 0.78$)	0.70 ± 0.01 (°)	$F_{(5, 80)} = 1.21$ ($P = 0.31$)	0.66 ± 0.02 (°)

* Note that the intact viewing condition refers to the condition in which text passages were presented without any simulated visual field defects. The intact viewing condition was tested twice: once in the central scotoma session and the other in the peripheral scotoma session.

increased by 21% (i.e., from 214 to 256 ms) from intact to the most severe peripheral scotoma (2° aperture).

Number of Fixations Per Line. The number of fixations also increased abruptly when the aperture size decreased below 6° (11 visible letters) ($P < 0.001$) (Fig. 4D, left panel). The number of fixations increased by a factor of 2 (i.e., from 12.45 to 24.83 per line) from intact viewing to the most severe peripheral scotoma.

Central Scotoma Conditions.

Fixation Duration. Fixation duration increased linearly with increasing central scotoma size in log-linear coordinates (Fig. 4C, right panel). For example, it increased by 13% (i.e., from 217 to 245 ms) from intact viewing to the most severe central scotoma ($P < 0.001$). The slope of the regression line indicated that an increase in the size of central scotoma by two masked letters led to an increase in fixation duration by 0.005 log units.

Number of Fixations Per Line. The number of fixations increased significantly when the central scotoma size increased beyond 4° (7 letters) ($P < 0.001$) (Fig. 4D, right panel). It increased by 89% from intact viewing to the most severe central scotoma.

Proportion of Regressive Saccades

To calculate the proportion of regressive saccades, the total number of regressive saccades was divided by the total number of (forward and regressive) saccades per sentence.

Peripheral Scotoma Conditions. The proportion of regressive saccades remained more or less constant across different aperture sizes except for a noticeable departure at the aperture size of 8° and 10° (Fig. 4E, left panel).

Central Scotoma Conditions. The proportion of regressive saccades increased linearly with increasing central scotoma size in log-linear coordinates ($P < 0.001$). It increased by 86% (i.e., from 24% to 45%) from intact viewing to the most severe central scotoma (Fig. 4E, right panel). The slope of the regression line suggested that for every two extra letters masked by the central scotoma, there was a corresponding 0.03 log units increase in the proportion of regressive saccades.

Microsaccade Rate, Amplitude, and Distribution

Microsaccade Rate. As shown in Figure 4F (left panel), microsaccade rate of the peripheral scotoma conditions remained relatively constant across different peripheral scotoma sizes with the exception of an aperture size of 6°. In contrast, microsaccade rate of the central scotomas decreased linearly with increasing central scotoma size in log-linear coordinates (Fig. 4F, right panel). More specifically, microsaccade rate decreased by nearly 70% (i.e., 0.23 ± 0.03 to 0.07 ± 0.02 no./sec) from intact viewing to the most severe central scotoma ($P < 0.001$). The slope of the regression line indicated that an increase in the size of central scotoma by two masked letters led to a reduction in microsaccade rate by 0.05 log units.

Microsaccade Amplitude. Consistent with our previous findings,[56] we found that microsaccade amplitude remained relatively stable across viewing conditions with an average amplitude of 0.70° under intact viewing (Fig. 4G).

Microsaccade Distribution in Comparison With Saccade Distribution. Figures 5A and 5B shows the distribution of microsaccades in comparison with all saccades (including microsaccades). In each row, from left to

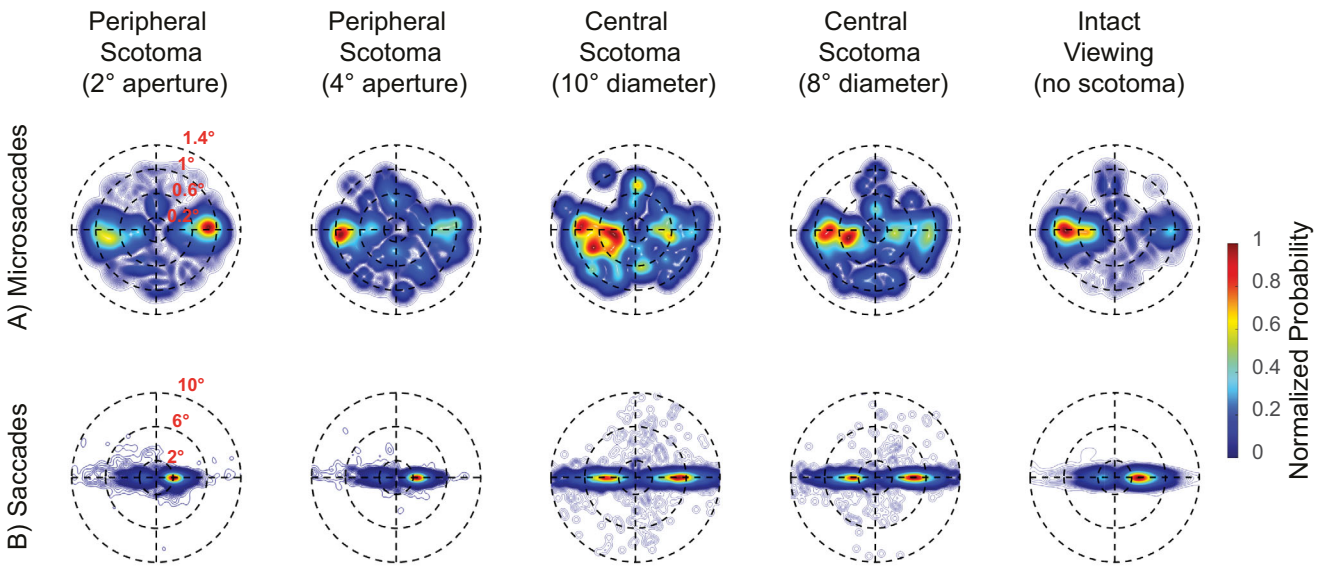

Figure 5. Distribution of microsaccades and saccades. Probability density maps of (**A**) microsaccades and (**B**) saccades are shown in two-dimensional polar maps representing the visual field. Each density map shows the data from all subjects in each condition. Note that saccade maps are based on the data from both regular saccades and microsaccades. Density maps are compared for severe peripheral scotomas (2° and 4° aperture), severe central scotomas (10° and 8 ° diameter), and intact viewing (no scotoma). The color bar indicates the colors corresponding to different probability density values. The radii of the polar plots and the numbers in *red color* indicate retinal eccentricity in degree units.

right, the five two-dimensional polar maps plot the probability density maps under two of the most severe peripheral (2° and 4° aperture) and central scotomas (10° and 8° diameter) for all subjects. The rightmost column shows intact viewing conditions. The radii of the polar plots and numbers in red color indicate retinal eccentricity in degree units. Note that as we were interested in the relative probability densities rather than the absolute values, we normalized the probability densities in each plot.

From these plots, it is apparent that under all conditions, both saccades and microsaccades exhibited a horizontal bias. However, saccades exhibited a noticeable horizontal rightward bias (in both intact viewing and peripheral scotoma conditions), whereas microsaccades appeared to be more spread out across the visual field. This pattern of results is in line with previous findings.[50,56] On the other hand, saccades in severe central scotoma conditions appeared to exhibit more regressive saccades as compared to the intact viewing or peripheral scotoma conditions. Taken together, we found that the direction and amplitude of microsaccades were comparable across different viewing conditions.

Discussion

Key Take-Home Messages of Our Findings

(1) Reading speed is closely coupled with the pattern of eye movements such as fixation duration and the number of fixations.
(2) The central 2.5° visual field (i.e., foveal vision or 4.4 visible letters) is fundamental to human reading behaviors. When it is deprived, noticeable changes in all the key eye movements and reading speed begin to emerge. In particular, a lack of foveal vision appears to lead to more regressive saccades but lesser microsaccades.
(3) The central 6° visual field (i.e., both foveal and parafoveal vision or 11 visible letters) appears to help maintain maximum reading speed as well as stable fixational eye movements. When the central 6° visual field is not met, a person's reading speed begins to decline sharply along with an abrupt increase in fixational eye movements (fixation duration and the number of fixations). It is, thus, evident that optimal reading performance requires both foveal and parafoveal vision.
(4) On the other hand, the perifoveal region (i.e., between the central 6° and 8° visual field) appears to influence saccade amplitude and velocity. Yet, it seems to be less critical to achieving a person's maximum reading speed as compared to foveal and parafoveal vision. Thus the central 6° to 8° (11–14 visible letters) visual field appears to be the upper limit of the visual field that may influence overall reading eye movements.
(5) The visual field requirement of 6° (11 visible letters) for reading speed is well aligned with the visual span hypothesis that posits the size of the visual span (approximately 9–14 letters for normal healthy adults)[39,64,66] plays a determining role in reading speed.[82]
(6) Our findings collectively lend support for a close linkage between human oculomotor behaviors and visual perception.[59,60]

Changes in Eye Movements and Reading Speed Under Peripheral Scotomas

We found that human observers tend to make more frequent and longer fixations as the visible portion of the visual field

diminished (Figs. 4C, 4D, left panel) while making faster and shorter saccades (Figs. 4A, 4B, left panel). For example, fixation duration and the number of fixations increased by 21% and 98%, respectively, whereas saccade amplitude decreased by 30% from intact viewing to the most severe peripheral scotoma condition (2° aperture). However, the aperture size had modest impact on the proportion of regressive saccades and microsaccades. Importantly, the dependency of each eye movement on the aperture size was best described by an exponential function in log-linear coordinates, that has a critical point beyond which the pattern of eye movements changes sharply.

As shown in the left panels of Figures 4A through 4D, we found that the critical aperture size for reading eye movements occurs somewhere between the aperture size of 6° to 8° (11–14 visible letters). There was a slight yet noticeable difference between the pattern of saccade amplitude/velocity and the pattern of fixational eye movements (i.e., fixation duration and the number of fixations). More specifically, once the central 6° visual field (i.e., foveal and parafoveal vision) is available, fixational eye movements stayed unchanged with respect to intact viewing. However, the perifoveal region (i.e., between the central 6° and 8° visual field) continued to remain relevant to saccade amplitude and velocity.

Considering that the average word length of English is about five characters,[83,84] we can infer that at least two or three words should be visible at fixation before any significant changes in reading eye movements occur. For example, from Figure 4C (left panel), we can infer that with the aperture size of 3° (five visible letters or one word length), a person's fixation duration is expected to significantly increase by 21 ms. This prediction is well aligned with a previous study reporting that fixation duration increases by 18 ms when allowing one word to be visible.[85] Furthermore, the pattern of more frequent and longer fixational eye movements was also observed in reading or face/letter recognition under degraded viewing conditions such as blur or low text contrast.[56,86] Perhaps such behaviors reflect the system's compensatory mechanism used to enhance information uptake in response to deprived visual sensory input.

How, then, is reading performance related to eye movements when peripheral visual processing is disrupted? As shown in Figure 2A, reading speed remained quite stable up to an aperture size of 6° (11 visible letters) before making a sharp decline, further confirming the critical role of both foveal and parafoveal vision in maintaining a person's maximum reading speed. The role of parafoveal processing in reading has been well documented in the literature: It has been shown that readers can process the upcoming word parafoveally and parafoveal information (e.g., word length or boundaries, lexical and semantic information) influence where readers' eyes move next and also facilitate subsequent foveal processing.[11,33,34,87–90]

The dependency of reading speed on the visible portion of the visual field is well aligned with the findings of previous studies on the visual span and reading.[25,39,41,42,66,91,92] The visual span (i.e., the number of letters that can be reliably recognized in one glance) can be thought of as a window in the visual field within which letters can be reliably recognized.[39,82] The visual span size is typically measured with a trigram letter-recognition task while participants fixate centrally without moving their eyes. It is estimated to extend about nine to 14 letters for adults with normal vision,[39,64,66] although neurotypical children[91] or adults with visual impairment[66] were shown to exhibit a much shorter visual span. Because the size of the visual span is largely limited by visual crowding, it is also called the "uncrowded window."[11] Ample evidence has demonstrated a close linkage between reading speed and the size of the visual span in both normal and clinical populations.[8,9,11,22,32,34,35] For example, slow reading speed in patients with either central or peripheral visual field defects was closely related to the shrinkage of the visual span.[8,66]

On the other hand, the perceptual span refers to the region of the visual field that influences eye movements and fixation times in reading.[36] Similar to the current study, the size of the perceptual span is typically measured dynamically using either the moving window technique[29] or the moving mask technique[93] in which the extent of the central visual field that disrupts a person's reading performance is estimated. The perceptual span is estimated to extend about 14 to 15 (skilled readers) or 11 (less-skilled readers) characters to the right of fixation and 3-4 characters to the left of fixation.[29,33,36,37] Thus the perceptual span of 14 to 19 letters appears to be slightly larger than what we have observed in the current study and the size of the visual span (i.e., the central 6°-8° visual field corresponding to approximately 11–14 visible letters). This could be due to various factors known to affect the size of the visual or perceptual span. For instance, it has been shown that the perceptual span reflects readers' linguistic processing[9,34] or overall cognitive processing[35] rather than visual sensory processing.[37] On the other hand, the visual span is assumed to be relatively immune to oculomotor and top-down contextual influences and is primarily determined by the characteristics of front-end visual sensory processing[39,94] (e.g., text properties such as letter contrast and size,[37] letter spacing,[38] and spatial resolution of letters[26]).

Importantly, the way reading speed is modulated by the aperture size closely resembles that of both fixation duration and the number of fixations: Both reading speed and the two eye movement metrics exhibit substantial changes when the visual field requirement of 6° (11 visible letters) is not met. Although the dependency of saccade amplitude and velocity on the aperture size was found to be also exponential, their decline was more gradual, resulting in a larger critical aperture size of 8° (14 visible letters) as compared to that of reading speed or fixational eye movements. On the other hand, changes in regressive saccades and microsaccades do not seem to covary with changes in reading speed under peripheral scotoma conditions.

Together, our findings suggested that reading speed appears to be more influenced by fixational eye movements such as its duration and frequency as compared to other eye movements. Previous work done by our group indeed supports this view: Yu et al.[56] investigated changes in eye movement patterns during reading under degraded viewing conditions such as low contrast or blurred text. Stepwise linear regression was performed to determine the degree to which eye movement variables contribute to prediction of reading speed. It was found that fixation duration alone accounted for 78% of variance in reading speed ($R^2 = 0.78$; $P < 0.001$), indicating a major role of fixation duration in reading speed. On the other hand, adding the proportion of regressive saccades and saccade amplitude to the model increased the R^2 value only by 12% while the contributions of the other eye movements such as microsaccades or saccade velocity were negligible. Perhaps the high relative contribution of fixational eye movements explain the

apparent dissociation between reading performance and some eye movement metrics.

Changes in Eye Movements and Reading Speed Under Central Scotomas

We found that the overall reading speed decreased by 51% from intact viewing to the most severe central scotoma condition (10° diameter) (214 wpm vs. 104 wpm). As compared to peripheral scotoma conditions, the decline of reading speed as a function of central scotoma size is rather gradual. However, the detrimental impact of central scotomas emerged as soon as the scotoma covered the central visual field of 2.5° (4.4 letters). Our findings further underscore the critical role of the foveal vision in the reading process, consistent with previous work on patients with central vision loss [40–42,54] or individuals with simulated central scotomas/masks.[8,33,93]

We observed that saccade amplitude and velocity, fixation duration, the number of fixations, and the proportion of regressive saccades more or less linearly increased with increasing central scotoma size in log-linear coordinates. For example, with increasing central scotoma size, saccade amplitude and fixation duration increased by 98% and by 13%, respectively, from intact viewing to the most severe central scotomas (10° diameter). The number of fixations also nearly doubled under the most severe central scotoma conditions. On the other hand, microsaccade rate decreased with increasing scotoma size. Our findings are consistent with the view that sensory information from the fovea is critical for microsaccade generation.[95] Otero-Millan et al.[95] also found that when imposed with simulated central scotoma, microsaccade rate dropped from 0.50 to 0.15 per second during free viewing of natural scenes.

Scherlen et al.[96] observed that the number of saccades almost doubled when reading under simulated central scotomas. Rubin and Turano[97] also showed that patients with dense central scotomas exhibit slower reading even in rapid serial visual presentation where saccadic eye movements were minimized, supporting a longer fixation duration during reading with central vision loss. Thus more frequent and longer fixational eye movements appear to be an emerging reading behavior when either foveal or parafoveal vision is not available, as also shown by our peripheral scotoma conditions. On the other hand, increased saccade amplitude was also reported in previous studies using simulated scotomas or moving mask paradigm for either reading[93] or visual search tasks.[98] For example, saccade amplitude increased from six to nine letters in size when the moving mask obscuring the central vision increased from 1 to 17 letters in size during reading.[93]

On the other hand, it has been reported that patients with central vision loss (i.e., AMD) tend to exhibit noticeably smaller saccades compared to normal controls.[57] The increased saccade amplitude observed in our current study may be partially attributed to an oculomotor reflex of normally-sighted subjects in response to simulated central scotomas. As previously mentioned, our study design did not include any adaptation period in which normally-sighted subjects were given time to fully adapt to central scotomas. Thus our subjects might have coped with the unfamiliar viewing condition by consistently switching between the fovea and peripheral retina to read, thereby leading to larger and more volatile saccades. Such large and volatile saccades have been observed in both normally-sighted individuals with simulated central scotoma, as well as in AMD patients before they have fully adapted to a central scotoma.[71,99] However, it is known that once AMD patients fully adapt, they often adopt a relatively stable preferred retinal locus in the intact peripheral retina for fixational and saccadic eye movements while performing visual tasks.[100] The development of a preferred retinal locus has also been observed in normally-sighted individuals with simulated central scotoma after several hours of exposure to the simulated central scotoma.[63] Thus the discrepant findings in saccade amplitude between AMD patients and our subjects with simulated central scotomas call for a future study in which the progression of changes in saccades can be examined as subjects adapt to simulated central scotomas.

Differential Effects of Peripheral and Central Scotomas on Reading and Eye Movements

We conjecture that some of the noticeable differences in reading behaviors between the peripheral and central scotoma conditions may be attributed to intrinsic differences in oculomotor control. In healthy normal vision, the fovea naturally serves to guide fixational and saccadic eye movements. The accuracy and precision of oculomotor control (e.g., fixational stability or saccade accuracy) are significantly higher in foveal vision compared to peripheral vision.[71,101–104] For example, we observed that regressive saccades appeared to be relatively immune to changes in the size of the peripheral scotomas where the foveal vision remained available throughout the duration of testing. However, regressive saccades increased by 86% from intact viewing to the most severe scotoma condition. More frequent regressive saccades were also reported in patients with central vision loss.[40,57,105] When reading five letters from left to right, patients with AMD showed almost two times more regressive saccades compared to healthy controls.[40] Thus poor oculomotor control without foveal vision may in part explain the apparent discrepancy in the proportion of regressive saccades between central and peripheral scotoma conditions.

However, we cannot rule out the possible interactions between oculomotor control and perceptual processing. The central scotoma conditions were deprived of not only oculomotor control but also foveal and parafoveal processing known to optimize reading eye movements.[8,11,93] Thus insufficient foveal or parafoveal information might have indirectly led to the differences in the pattern of eye movements between central and peripheral scotoma conditions. Evidence from a computational model of reading eye movements further supports this view. Using Mr. Chips—an ideal observer computational model for reading—Legge et al.[106] demonstrated that a higher rate of regressive saccades and reduced saccade amplitude are shown by Mr. Chips in response to central scotomas. The task of Mr. Chips was to read the text in the minimum number of saccades by reducing uncertainty of currently viewed words (i.e., an entropy minimization principle) given a set of visual, oculomotor, and lexical constraints. As the oculomotor control of Mr. Chips was represented by Gaussian noise that remained constant across the visual field, the oculomotor factor could not account for Mr. Chips' reading behaviors that emerged under central scotomas. Therefore these assumed-to-be poor reading strategies appeared to be an optimal reading

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strategy that served to compensate for deprived foveal and parafoveal information rather than a result of poor oculomotor control.

In conclusion, our results show that when visual information is limited by visual field defects, the pattern of eye movements during reading is markedly deviated from that of intact viewing with differential impacts of central and peripheral vision loss. These apparently deviated oculomotor behaviors may in part reflect optimal reading strategies to compensate for the loss of visual information.

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