The invisibility of Scotomas: Scotoma Carving with Loss of High Level cells

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Purpose

Most simulations of vision with scotomas wrongly represent the scotoma as black patches. Patients with central scotomas report 'blurred' vision, not a black patch. Paracentral scotomas are not explicitly observable by the patients, though spatial distortions are sometimes reported. The 'field loss' is not apparent; the scotoma itself and anything in it is just missing (elided) from the patient's consciousness. Simulating such perception is difficult but is needed to be correctly understood for training professionals, educating family members, caretakers, as a research tool for vision rehabilitation, and for public advocacy. We developed new ways to explain and simulate the invisibility of the scotomas.

Methods

We applied a content-aware removal image processing algorithm (based on Seam Carving) to remove the area in images corresponding to instantaneous placements of the scotomas. This results in the area elided under the scotoma having seamless continuation with the remainder of the scene, but with spatial distortions around the missing content. In representing the effects of saccadic eye movements, we shift the scotomas positions in the image abruptly and incorporate saccadic blur. The reduced resolution with eccentricity is added to the simulations using pyramidal contrast modeling. In an additional "flipbook" representation we recenter the newly fixated object without saccadic blur.

Results

We obtained simulated perceived images (and videos) with no visible scotoma that are consistent with the description provided by patients with central and peripheral field losses caused by the loss of retinal ganglion or higher cells. The paracentral simulations successfully hide the content loss and the dynamic distortions caused by eye movement with saccadic blur/suppression.

Conclusions

These simulations provide insight into the lack of awareness of the scotoma as elided contents in the scene with and without eye movements. The lack of awareness may cause the poor compliance of patients with glaucoma that may be improved with better simulations and explanation.

Simulations of views with partial arcuate scotama carved at 2 different fixations causing distortions

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The invisibility of Scotomas: Scotoma Replacement with Loss of photoreceptors

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Purpose
Most simulations of vision with scotomas wrongly represent the scotoma as black patches. Patients with central scotomas report 'blurred' vision, not a black patch. Paracentral scotomas are not explicitly observable by patients. We presented (in another abstract) new way to simulate the view with scotomas caused by loss of retinal ganglion cells and higher. In that process the elided content under the scotoma is carved out from the perceived image. Here we show that with scotomas caused by loss of photoreceptors (i.e., Dry macular degeneration or retinitis pigmentosa) the perception is different from scotomas caused by loss of higher neurons, and the elided content is replaced by content surrounding the scotoma, rather than being carved out.

Methods
Some ganglion cells' receptive fields are centered inside the scotoma but are large enough to extend outside the scotoma. These cells contribute to perception inside the scotoma based on the image content outside the scotoma. This situation is simulated. The pixel values in scotomatous areas of an image are replaced with "not a number" (NaN). A pyramidal contrast model (Peli, JOSA 1991, 2001) is applied in the spatial domain. The interaction of the NaN convolution with the pyramidal structure results in a simulated percept with the scotoma consisting of low passed content from just outside the scotoma. Reduced resolution with eccentricity is applied by using eccentricity dependent contrast thresholds in the pyramidal structure.

Results
The images simulating perception with scotoma are consistent with descriptions of patients with either central or peripheral field losses due to photoreceptor loss, i.e. a "blurred" appearance inside and outside the scotoma area. The replacement operation constitutes an actual "filling-in", which is effective for natural images, unlike the misnamed "filling-in" described for the physiological scotoma. The effect is of retinal origin and thus does not require postulating cortical or other adaptation.

Conclusions
These simulations provide insight into the effect of lesion location on the invisibility of the scotoma. The high spatial correlation in images facilitated the acceptance of the replacement content as appropriate. These simulations need to be verified with patients.

There is no conflict of interest

Replacement of central scotoma with content from outside the photoreceptor scotoma

Replacement of peripheral ring photoreceptor scotoma, as in retinitis pigmentosa. Fixation on right

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Title: Detection of glaucoma progression based on changes of the circumpapillary RNFL thickness between two OCT scans

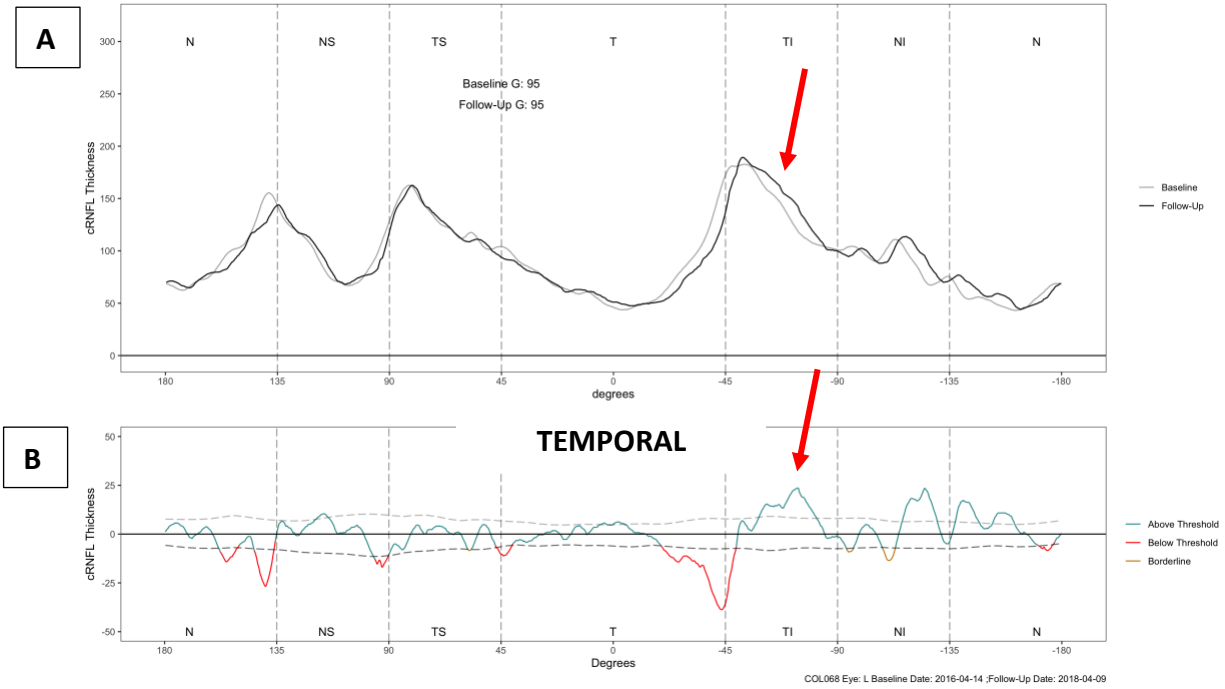
Purpose: To develop and evaluate a new method that identifies progression based on changes on the circumpapillary retinal nerve fiber layer profile (cpRNFL)

Methods: 121 eyes/individuals (29 HCs, 92 patients) had baseline and follow-up OCT scans (Heidelberg, Spectralis) and 24-2 and 10-2 visual fields (VFs) from a longitudinal, observational study (MAPS, PI: CG De Moraes). The difference in thickness between the 1st baseline (gray line, Fig. 1A) and the last follow-up test (black line, Fig. 1A) is shown in Fig. 1B. Short-term variability was calculated from scans within a period of 4 months. Regions of the difference curve in Fig. 1B that fell below the short-term variability threshold (5th percentile, lower dashed line Fig. 1B) and were wider than 5 degrees were considered significant and are shown in red. The amount of 'red' was calculated by a custom R-program. C_{crit} was defined as the amount of change (red) in degrees based on the 95th percentile of the distribution of change in the 29 HCs (bootstrapping: 1000 iterations). In addition, cpRNFL summary metrics were extracted and 'statistical progressors' were identified based on an event analysis.[1,2] For a reference standard, 3 experts rated each of the 92 patient eyes based on the whole series of OCT and VF tests and identified 17 eyes as Definitely Progressors (DP), based on topographical agreement of changes on both the OCT and VFs.

Results: A C_{crit} of ~20 degrees identified 2 HCs as progressing – these were false positives (FP) (specificity 93%). A post-hoc analysis suggested that segmentation errors and misplaced follow-up circle scans were the reason behind these FPs. These FPs could be identified by the abnormal positive change (i.e., thickening) seen on the difference plots (red arrows, Fig. 1B). In addition, all but one of the DP eyes were identified by the automated method. The commonly used averaged global (G) summary metric showed similar levels of specificity (97%, 1 FP), but detected only 11 of the 17 DP eyes. The best performing summary metric was the temporal-superior (TS) with perfect specificity and 13 out of the 17 DP eyes correctly identified.[2]

Conclusions: The proposed method successfully detected 94% of the eyes with progressing damage and performed better than commonly used summary metrics. However, segmentation errors and misplaced follow-up circle scans can cause FPs. Future improvements can take into account thicker-than-before regions which are indicative of these scan artifacts.

1. Tsamis et al. TVST 2022; 2. Tsamis et al. [under review] 2022



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Remaining Authors: No Conflict of Interest.

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Title: Characterization of retinal nerve fiber layer and ganglion cell layer inner plexiform thickness in the Ocular Hypertension Treatment Study (OHTS 3) 20-year follow-up.

Purpose: OHTS 3 is unique in that the date of primary open angle glaucoma (POAG) onset is precisely determined. With OCT scans acquired, OHTS 3 presents a rare opportunity to characterize the magnitude of structural damage after the onset of POAG. The objective of this study is to characterize retinal nerve fiber layer thickness (RNFLT) and ganglion cell inner plexiform layer thickness (GCIPLT) in eyes that did and did not develop POAG and determine the rate of structural change before and after the onset of primary open angle glaucoma.

Methods: 659 (40.3%) of the original 1636 OHTS participants completed the OHTS 3 visit. The OHTS Endpoint Committee determined whether an eye developed POAG based on clinically significant photograph-based disc and/or repeatable visual field changes. Participants with good quality Spectralis (n=241) and Cirrus (n=478) optic nerve head (ONH) and macula imaging were included (68 participants had both). The rate of RNFLT and GCIPLT change over time was calculated for the participants that provided longitudinal Spectralis (n=88 eyes) and Cirrus (n=261 eyes) images. Linear mixed effects models were used to compare RNFLT and GCIPLT thickness and change over time in eyes that developed POAG compared to eyes that did not.

Results: At ~20 years of follow-up, 260 (54.4%) of the 478 participants with Cirrus scans did not develop POAG and 218 (45.6%) developed POAG. The mean (95% CI) RNFLT and GCIPLT of the eyes that did not develop POAG (84.7 (83.7, 85.7) um and 74.1 (73.4, 74.9) um, respectively) was significantly thicker than in the eyes that developed POAG (71.4 (70.1, 72.7) um and 66.7 (65.5, 67.9) um, respectively) (both $p<0.001$). Spectralis OCT results were similar. The mean (95% CI) Cirrus RNFLT of the POAG eyes that were imaged 0-10 years before POAG (n=177 eyes), 0-10 years (n=56 eyes) and 10+ years (n=88 eyes) after the development of POAG was 76.7 (75.0, 78.3) um, 69.3 (66.0, 72.5) um and 70.0 (67.7, 72.2)) um, respectively ($p<0.001$). The mean rate of Cirrus RNFL thinning was 5.3x faster in eyes that developed POAG (-1.48 um/yr) compared to eyes that did not develop POAG (-0.28 um/yr) ($p<0.001$). The mean rate of Cirrus GCIPL thinning was 3.6x faster in eyes that developed POAG (-0.94 um/yr) compared to eyes that did not develop POAG (-0.26 um/yr) ($p=0.048$).

Conclusion: Among eyes that developed POAG, the RNFL and GCIPL was ~10 um thinner and the rate of RNFL and GCIPL thinning was much faster than in eyes without POAG.

Poster Only

Table 1: Summary of Demographic Characteristics and OCT Results						
	Spectralis OCT			Cirrus OCT		
	No POAG	POAG	p-value	No POAG	POAG	p-value
By Subject	n = 125	n = 116		n = 260	n = 218	
Age	52.9 (51.5, 54.3)	52.5 (51.04, 54.02)	0.706	52.1 (51.2, 53.0)	53.5 (52.5, 54.6)	0.041
Sex % Female	80 (64.0%)	59 (50.9%)	0.050	160 (61.5%)	121 (55.5%)	0.091
Race						
Black, Non-Hispanic	11 (8.8%)	29 (25.0%)	0.004	61 (23.5%)	66 (30.3%)	0.155
White, Non-Hispanic	108 (86.4%)	83 (71.6%)		185 (71.2%)	137 (62.8%)	
Other	6 (4.8%)	4 (3.4%)		14 (5.4%)	15 (6.9%)	
By Eye	n = 246	n = 184		n = 497	n = 321	
Global RNFL Thickness (um)	n= 246 90.5 (89.1, 92.0)	n = 184 76.2 (73.7, 78.7)	0.001	n = 462 84.7 (83.7, 85.7)	n = 295 71.4 (70.1, 72.7)	0.001
Global GCIPL Thickness (um)	n = 152 71.1 (69.8, 72.4)	n = 135 63.9 (62.1, 65.7)	0.001	n = 472 74.1 (73.4, 74.9)	n = 275 66.7 (65.5, 67.9)	0.001
Rate of RNFL thinning (um/yr)	n = 50 -0.75 (-1.23, -0.26)	n = 38 -1.10 (-1.73, -0.48)	0.679	n = 158 -0.28 (-0.73, 0.07)	n = 103 -1.48 (-2.08, -0.88)	0.001
Rate of GCIPL thinning (um/yr)	n = 51 -0.42 (-0.73, -0.11)	n = 37 -0.84 (-1.20, -0.47)	0.150	n = 156 -0.17 (-0.68, 0.11)	n = 85 -0.99 (-1.55, -0.43)	0.048
VF MD (dB)	n = 246 0.36 (0.20, 0.51)	n = 184 0.27 (0.10, 0.43)	0.596	n = 497 0.36 (0.22, 0.50)	n = 321 0.01 (-0.15, 0.17)	0.001
Median OCT Follow-Up (Years)	n = 148 4.5 (4.1, 4.9)	n = 94 4.9 (4.4, 5.4)	0.162	n = 266 3.4 (3.2, 3.6)	n = 159 3.6 (3.3, 3.9)	0.277

Categorical variables displayed as percentages, and continuous variables displayed as means with 95% confidence intervals.