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Simulation of a central scotoma using contact lenses with an opaque centre

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Abstract

Purpose—This study evaluated the feasibility of using soft contact lenses (CLs) with an opaque centre to induce absolute central scotomas that move with the eye. We examined the geometrical optics prediction that scotoma size will vary with the size of the CL's opaque centre and with ocular pupil size. We also tested the hypothesis that high environmental light levels will ensure that the ocular pupil will remain small enough, even with opaque centre CLs, to generate absolute scotomas representative of those experienced by patients with age-related macular disease.

Methods—Using an Octopus 900 Perimeter (Haag-Streit, Ohio, USA), kinetic visual fields (VFs) were measured in five normally-sighted subjects using a V4e Goldmann target with CLs that had central opaque areas with diameters of 2.8 mm, 3.0 mm, and 3.2 mm. To control pupil size, VFs were measured with background perimeter bowl luminances of 10 cd/m², 585 cd/m², and 1155 cd/m². Subjects attempted to (i) fixate the bowl centre; and (ii) place the scotoma edge at the bowl fixation target (eccentric viewing).

Results—As predicted, there was a direct relationship between scotoma size and both luminance level and diameter of the opacity. Mean scotoma diameters were 0°, 17.6° and 22°, for the low, medium and high bowl luminances, respectively. Scotoma size was determined primarily by the difference between the diameters of CL opacity and the entrance pupil of the eye and the axial separation between them, and between-subject differences in pupil diameters contributed most to the between-subject variability in scotoma diameter at each light level (SD: 6.01°). Scotoma displacement during eccentric fixation confirmed the gaze-contingent characteristics of this experimental model.

Conclusion—It is possible to induce a gaze-contingent absolute scotoma and hence mimic central vision loss using centrally-opaque CLs provided that the CL opacity is larger than the entrance pupil of the eye. This simulation tool will, therefore, be ineffective at low environmental light levels (as shown previously) if the entrance pupil of the eye is larger than the CL opacity.

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Keywords

low vision; age-related macular degeneration (AMD); simulated central scotoma; contact lenses; experimental AMD model

Introduction

In 2015, there were 901 million people aged 60 years and older in the world, and this number is projected to increase by 56% to be 1.4 billion by 2030.(1) With an ageing population, the number of people who will have major eye disease(s) will also increase given the association between the prevalence of ocular disease with increasing age.(2, 3) This association is particularly strong for age-related macular degeneration (AMD), and it is estimated that 196 million people will have AMD worldwide in 2020.(4) As we age, the outer retina can accumulate drusen and develop neovascularisation, both of which are characteristic of AMD and can cause a loss of central vision (scotoma) manifesting as reduced visual acuity and contrast sensitivity.(5, 6) Vision loss can be either complete, where affected retinal areas fail to detect light (absolute scotoma), or partial, where the sensitivity of the affected retinal areas is present but reduced (relative scotoma). These visual losses often lead to the disruption of many activities of daily living (e.g. reading (7–9) and driving (10, 11)) as well as overall decreased quality of life.(12, 13) Depression is also associated with AMD,(14) since the prevalence and level of depression has been shown to increase as the severity of vision loss from AMD increases.(15)

Investigating the functional impact of a central scotoma is difficult, in part because of reduced fixation stability (16–19) and re-fixation precision.(20–22) Furthermore, due to large variations in scotoma size and shape across subjects,(5, 23, 24) the impact of scotoma size can only be assessed with between-subject comparisons, and a large number of AMD subjects are often required to obtain statistically significant differences between AMD and control subjects.(25) An experimental model of AMD will therefore allow a within-subject experimental design with decreased variability, and allow for the systematic evaluation of the behavioural consequences of central scotomas of variable size.

There are several methods to simulate a central scotoma. Mounting a central opacity on a spectacle frame can generate a scotoma that moves with the head,(26) but subjects still can foveate by changing their gaze angle. Computer-based eye-tracking systems can also be used to induce a gaze-dependent scotoma that remains at a fixed retinal location as the eye rotates,(27–33) but this approach is currently limited to the laboratory environment and visual field (VF) size is restricted by monitor size. Other methods that have been used to induce a central scotoma include using an occluder mounted to a scleral contact lens (CL) affixed with suction(34) or embedding a small mirror plane within a scleral lens.(35) However, these methods require precise positioning of the scotoma.(34, 35). More recently, opacities tinted into soft CLs have been developed as a convenient method to induce fixed peripheral and central scotomas that move across the visual world as the eye moves (gaze-dependent).(36–39). However, Butt et al.(40) failed to create an absolute central scotoma using CLs with a tinted central opacity, most likely because pupil dilation allowed light to

enter the eye around the edge of the CL opacity. In order to generate an absolute scotoma with the CL opacity method, the angular subtense of the opacity must be greater than the angular subtense of the eye's entrance pupil when both angles are measured from the stimulus. Previous successful attempts with CL opacities have met this requirement by either employing large (8 mm) corneal plane opacities,(37) or reducing entrance pupil size with a miotic drug.(36)

In this study, we investigated the feasibility of using centrally-opaque CLs to generate gaze-dependent absolute central scotomas without the use of miotic drugs. We employed high environmental light levels sufficient to make the entrance pupil smaller than the CL opacity, and compared the induced scotoma sizes with optical ray tracing predictions.

Methods

Ray tracing

Scotoma size was calculated using a single surface ray tracing model with a circular opacity (radius h) located on its anterior surface with sag (s). The ray tracing model contained a spherical refracting surface (cornea) with radius $r = 7.71$ mm, anterior chamber depth $w = 3.52$ mm, and a refractive index $n' = 1.376$. Pupil radius (p) was allowed to vary from 1.0 mm to 2.3 mm, and opacity diameters varied from 2.8 mm to 3.2 mm (the size of actual opacities in our custom CLs). Rays were traced from distant objects at various field angles (θ). In order to compare with experimentally measured VFs, the same model was used to calculate VFs for individual eyes using eye-specific values for corneal radius (r), anterior chamber depth (w) and pupil radius (p). If the CL opacity is larger than and centred relative to the entrance pupil (see Figure 1), no light from the axis ($\theta = 0^\circ$) will enter the eye (since the CL opacity completely covers the pupil when viewed from the fixation point, Figure 2A). However, for eccentric light sources beyond θ_1 (the maximum angle for which full pupil occlusion occurs) some rays will pass through the pupil, but the pupil will not be fully illuminated, creating a relative scotoma (Figure 1 and Figure 2B). As the angle of incidence increases beyond θ_T , 100% pupil illumination is achieved, and thus the presence of the central opacity will have no effect on retinal illuminance (Figure 1 and Figure 2C). Therefore, we can define the absolute scotoma extent as $2*\theta_1$ for a well-centred CL.

From Figure 1 we can state that:

$$\varphi = \tan^{-1} \frac{h}{(r-s)} \quad (1)$$

$$\theta_N = \tan^{-1} \frac{(h-p)}{(w-s)} \quad (2)$$

and

$$\theta_r = \varphi - \theta_N \quad (3)$$

Substituting θ_r into Snell's law, we obtain θ_i :

$$\theta_i = \sin^{-1} \frac{n' \sin \theta_r}{n} \quad (4)$$

From Figure 1 we can state that:

$$\theta_1 = \varphi - \theta_i \quad (5)$$

The proportion of rays passing through the eye's pupil for light sources between θ_1 and θ_T is defined by the geometry of vignetting by two axially separated overlapping ellipses, which depends on the size of the corneal plane opacity, size of the ocular pupil, and field angle θ . For small field angles, similar predictions can be made with a paraxial approximate equation ($\theta_1 = \tan^{-1} (h-EP)/(ACD-s)$), where EP is entrance pupil radius, and ACD is the axial separation of the corneal apex and EP).

Experimental Methods

Monocular, right eye semi-automated kinetic perimetry was performed with the Octopus 900 perimeter (www.Haag-Streit.com) on five healthy, normally-sighted subjects aged between 23 to 31 years. The V4e Goldmann stimulus (1.7° diameter, 0 dB intensity) was presented along 12 meridians separated by 30° intervals. The test stimulus was always moved at 5° per second in random order from non-seeing to seeing. Subjects were instructed to fixate the centre of a 5° diameter fixation circle located centrally within the perimeter bowl. If subjects were unable to see the fixation circle due to a central absolute scotoma, they were asked to fixate their best estimate of the centre of the fixation circle. The presence of a central scotoma >5° in diameter, therefore, gave subjects the challenging task of maintaining central fixation.

To test that the induced scotoma was gaze-contingent, subjects were instructed to direct their fixation in order to place one edge of the scotoma inferior, superior, nasal and temporal to the perimeter bowl fixation circle, making the edge of the 5° fixation circle coincident with the near edge of the scotoma. This is analogous to a patient using a preferred retinal locus (PRL) at each of the four (principal) edges of the scotoma.

Initial estimates of scotoma location and size were obtained with a single pass through the full visual field documenting a total of three points along each meridian; the VF extent and then the two edges of the induced scotoma. To obtain VF extent, the light stimulus was moved from the extreme periphery towards the perimeter bowl centre. Subjects were instructed to press a button whenever they first saw the light stimulus moving in their peripheral VF. To obtain the borders of the induced scotoma, subjects were instructed to

press the button if the light disappeared as it moved from the VF extent towards the centre of the bowl and press the button again if the light reappeared. The test stimulus was then placed in the centre of the estimated scotoma location, and we confirmed that the stimulus was invisible. The scotoma was then mapped along at least the eight principal meridians by moving the light stimulus out from the centre of the scotoma (non-seeing) at a rate of 5°/second, until the subject reported when they first saw the stimulus (seeing).

We employed supplemental lighting within the perimeter bowl to achieve significant pupil miosis. Two LED lighting strips (LUXEON XF-3535L, www.Lumileds.com) with a specified luminous flux of 1584 lumens and forward voltage of 18 volts were installed to the interior sides of the perimeter bowl (to the right and left of the bowl opening through which the patient views the stimulus), which elevated bowl luminance levels from 10 cd/m² without supplemental lighting, to 585 cd/m² or 1155 cd/m². Bowl background and stimulus luminance were measured with a CS-100A (www.KonicaMinolta.com) luminance meter by taking a minimum of three repeat luminance measurements at four locations (perimeter bowl centre and 20° to the inferior, nasal and temporal side of perimeter bowl centre).

Scotoma and VF extent data were extracted from the VF plots by using a web-based plot digitiser program. After scanning all VF plots for all subjects, the eccentricities and corresponding meridians of the measured VF extents, blind spots and absolute scotoma boundaries were manually determined.

Each subject's right eye was fitted with a prism ballast, plano, centrally-opaque soft CL (www.Kontur55.com, Methafilcon A, 55% water, daily wear) with central opacity diameters of 2.8 mm, 3.0 mm, and 3.2 mm (www.handpaintediris.com and www.techcolors.com). All CLs had a diameter of 15 mm and a base curve of 8.3 mm or 8.6 mm. To minimise lens rotation, a prism of 1.5 dioptre (D) was included in all lenses. To generate a scotoma centred at the fixation point, we had to compensate for the typical temporal decentration of soft CLs. (41, 42) To identify the CL location centred over the pupil, all CLs were fitted to a subject with a typical cornea (7.7 mm), and a small dot was made at the CL position that appeared centred on the pupil from along the subject's primary line of sight. The circular opacities were then painted concentric with these dots. For most subjects, these decentred (on the lens) opacities were centred over the subjects' pupils and produced well-centred scotomas, but some subjects were rejected because the CL opacity was decentred to such an extent that the resulting displacement of the scotoma enabled foveal fixation.

Entrance pupil diameters for each subject were measured at each of the three luminance levels by imaging the subject's untested left eye which was covered by an infrared transmitting and visible blocking filter while the tested right eye fixated the bowl. This approach was necessitated because entrance pupil size could not be measured directly in the eye wearing the CLs because the central opacity in the CL obscured the subject's pupil (see Figure 2).

Each of the five subjects was tested with the 3.2 mm diameter opacity, and two subjects were tested with each of the 2.8 mm and 3.0 mm opacities for a total of seven eye-CL pairings. Visual fields were measured at all three background light levels, for a total of 21 sets of VF

plots. The present study adhered to the tenets of the Declaration of Helsinki and informed consent was obtained from all subjects.

Data Analysis

Using the x- and y- coordinates from the scanned scotoma borders, the mean geometric centre of each subject's absolute scotoma was calculated. The average distance from the scotoma borders to the mean geometric centre of each scotoma along each meridian represented the average radius of an equivalent circle of the absolute scotoma in degrees. The predicted scotoma diameter was determined for each subject using the ray tracing model along with their measured pupil size, measured CL opacity size, and measured values for corneal radius and anterior chamber depth which were obtained with a Lenstar LS 900 (www.Haag-Streit.com).

A Kolmogorov-Smirnov test confirmed that scotoma size distributions obtained at each luminance level were not significantly different from a normal distribution and thus parametric statistics were subsequently performed.

Pearson correlation coefficients were computed to assess the relationship between the predicted and measured scotoma size. A repeated measures Analysis of Variance (ANOVA) was performed to assess for the main effects of luminance (10, 585, and 1155 cd/m²) and fixation pattern (central, superior, inferior, nasal and temporal) and for any interactions on measured scotoma diameters. Given an inflated Type I error rate associated with multiple pair-wise comparisons on the same data set, we applied a Bonferroni adjustment which yielded a new significance level of 0.01.

Results

Ray tracing

There was a direct relationship between predicted scotoma size and both luminance level and CL opacity size. As luminance level increased, ocular pupil size decreased and consequently the scotoma size increased. Figure 3 shows the impact of the CL opacity on light entering the pupil as a function of field location for two sample cases.

With pupil size smaller than the CL opacity diameter, all light was prevented from entering the eye on the visual axis (Figure 3), but with increasing eccentricity (8.5° radius and 11.5° radius for the 2.8 mm and 3.2 mm CL opacities, respectively) light started to enter the pupil. At 26.5° radius for the 2.8 mm CL opacity and 29.6° radius for the 3.2 mm CL opacity (Figure 3), the pupil was fully illuminated and not occluded by the CL opacity (see slit lamp images in Figure 2). Comparison of the 2.8 mm and 3.2 mm diameter CL opacities revealed a direct relationship between predicted scotoma size and CL opacity size. The visual field angles between the 0% and 100% pupil illumination represent a relative scotoma. Absolute scotoma diameter was larger as CL opacity increased in size and as pupil decreased in size (Figure 4).

Experimental Results

Perimetry was first assessed at the three different bowl luminance levels using a V4e test stimulus without any CLs. As background luminance was increased from 10 cd/m² to 585 (SD 23.8) cd/m² and 1155 (SD 45.5) cd/m², test stimulus contrast (L/L) was reduced from 944% to 16.2% and 8.2%. In spite of the increased retinal illuminance, this reduction in test stimulus contrast reduced stimulus visibility and hence visual field size, but visibility of the test stimulus was retained over the majority of the visual field, including all central field locations (compare Figures 5A and 5B). In Figure 5, we show examples of perimetry data from one subject tested with two different pupil diameters (i.e. 2.7, and 2.4 mm) that were obtained when the bowl luminance levels were 585 and 1155 cd/m², respectively. The outer edge of the visual field is shown for the eye without the centrally opaque CL (blue dashed line) and with the centrally opaque lens on the eye (green line). The increased VF size observed with the CL may reflect the small pupil dilation that accompanies the loss of light centrally caused by the centrally opaque CLs. This control experiment was run on three subjects, and the mean (SD) increase in VF diameter was 3.7° (0.7°).

Testing at a bowl luminance of 10 cd/m² without supplemental lighting, no absolute scotomas were generated using centrally-opaque CLs for all subjects and CL opacity diameters. This is because under these conditions pupil diameters were always larger than the CL opacities and the high contrast perimeter stimuli remained visible within the relative scotomas. However, when the luminance level increased to 585 and 1155 cd/m², the average ocular pupil diameter constricted to 2.4 mm and 2.3 mm, respectively which meant that the pupil diameters were smaller than any of the central opaque CL diameters. In the example shown in Figure 5A and Figure 5B, pupil diameters were 2.7 mm and 2.4 mm, which generated scotoma diameters of 13.8°, and 17.4° with the 3.2 mm diameter central opaque CL.

For each CL opacity size, absolute scotoma diameters differed from one subject to another because pupil diameter and anterior chamber depths were not the same (four sample data sets are shown in Figure 6). For our sample of five eyes, the monocular measured scotoma diameters for the three CL opacities at the different perimeter background luminance levels revealed that scotoma size depended on background luminance and pupil diameter. For the low (10 cd/m²), medium (585 cd/m²) and high (1155 cd/m²) luminance conditions, the average pupil diameters were 4.2 mm, 2.4 mm and 2.3 mm and for the 3.2 mm CL opacity, there were only central scotomas at the two brighter conditions, and their average diameters were 17.6° and 22°. As predicted, the luminance modulated pupil sizes had a very significant effect on measured scotoma size ($F_{(1, 6)} = 50.8$, $p < 0.001$). Ray tracing predicted no scotoma when the pupil was larger than the CL opacity and a linear increase in scotoma size as pupil size decreased below the CL opacity size (Figure 4). Our experimental data confirmed this prediction (Figure 7), showing that there was no absolute scotoma (0°) until the opacity diameter was larger than the ocular pupil diameter. It should be noted that our model assumed a distant light source, while our experimental data are collected with stimuli at 30 cm, which should increase measured scotoma diameter by about 1% relative to the model predictions.

Using a criterion of 0% pupil illumination to define the size of the absolute scotomas, our model underestimated the measured centrally fixated scotomas (see Figure 8A, black circles) with a root mean square error (RMSE) = 5.3° . On average, observed scotoma diameters were larger than predicted scotoma diameters as shown by the slope being significantly different from 1 ($t_{(19)} = 3.7$, $p < 0.05$) (see Figure 8A, black circles). Multiple reasons might explain why the observed scotoma size was larger than the predicted absolute scotoma size. One main factor is the expected false expansion due to the dynamic perimetric methodology we used.⁽⁴³⁾ For example, for a typical reaction time of 370 msec,^(44, 45) and a target speed of 5° per second we anticipate a small increase in apparent scotoma diameter of 3.7° . After removing this artificial expansion of the observed scotoma, the measured centrally fixated scotomas and the predicted scotomas were highly correlated with a RMSE = 3.4° (see Figure 8A, purple triangles). This was also confirmed by the slope being insignificantly different from 1 ($t_{(19)} = 1.3$, $p = 0.6$). Another possible explanation as to why our model underestimated the measured scotomas may relate to the stimulus visibility threshold. It is possible that subjects reported perimeter light stimulus to disappear although the pupil was partially illuminate. The disappearance of targets just beyond the edge of the absolute scotoma reflects the combined lowered target contrasts in the perimeter bowl (e.g. lowered to 8% with the highest background levels) and the low retinal illuminances produced at the edge of the absolute scotoma. Both these factors reduce visibility.⁽⁴⁶⁾ When employing a 10% pupil illumination threshold criterion (target not seen if $<10\%$ pupil transmission), the measured centrally fixated scotomas and the predicted scotomas were highly correlated and approximately the same size (see Figure 8A, red circles) with the slope being insignificantly different from 1 ($t_{(19)} = 0.4$, $p = 0.2$) (RMSE = 3.10°).

With eccentric viewing (EV) and using a criterion of 0% pupil illumination to define the size of the absolute scotomas, our model also underestimated the measured scotomas (see Figure 8B, black circles) with the slope being significantly different from 1 ($t_{(19)} = 3.5$, $p < 0.05$) (RMSE = 3.6°). Both factors mentioned earlier (i.e. false expansion due to the dynamic perimetric methodology and stimulus visibility threshold) might also explain why the observed scotoma size was larger than the predicted absolute scotoma size when EV was maintained. Removing the artificial expansion due to the dynamic perimetric methodology provided the best agreement between the predicted and measured scotomas with the slope being insignificantly different from 1 ($t_{(19)} = 0.002$, $p = 0.9$) (RMSE = 2.0°) (see Figure 8B, purple triangles). Also, when we fitted the EV scotoma data, a 5% pupil illumination provided the best agreement between the predicted and measured scotomas (see Figure 8B, red circles) with the slope being insignificantly different from 1 ($t_{(19)} = 0.3$, $p = 0.8$) (RMSE = 2.0°).

Perimetry data from one subject using four eccentric fixation locations (Figure 9) placed the scotoma to the inferior, superior, nasal or temporal edge of the fixation circle. When subjects were instructed to place the edge of their visible scotoma at the edge of the 5° fixation circle, the measured mean edge location of the scotoma along the eccentric fixation meridian was 2.5° , revealing the accuracy of this adaptive fixation strategy. We also found that scotoma size did not change significantly across the four different eccentric fixation locations (see Table 1, Repeated Measures ANOVA, $F_{(3,39)} = 0.99$, $p = 0.4$), being well fit by an equivalent circle whose radius was equal to the average distance in degrees from the

scotoma centre to its borders along each meridian (Figure 9). The average (SD) scotoma radius for all subjects across all conditions with central fixation was 10.4° (3°) while it was 9.6° (2.4°) with eccentric fixation. Repeated measures ANOVA with a Bonferroni correction revealed no significant difference between the scotomas sizes measured centrally with those measured at the four different eccentric fixation locations ($F_{(4, 24)} = 3.7$, $p = 0.02$). The trend of having larger scotomas with central fixation (Table 1) may reflect the fixational challenges faced by subjects when trying to fixate the invisible centre of the bowl.

Discussion

As predicted by a single surface ray tracing model, we were able to produce a central scotoma using soft CLs containing 2.8 mm, 3.0 mm and 3.2 mm diameter opacities as long as we employed high photopic light levels sufficient to reduce pupil diameters to less than 3 mm. It is likely that previous studies which failed to detect an absolute central scotoma with centrally located opacities on CLs(40) did not employ high enough light levels, and for rays originating from the fixation target, the CL opacity probably only occluded part of the pupil. It is not surprising, therefore, that a study employing pilocarpine miotics successfully generated absolute central scotomas with central CL opacities,(36) because a single drop of 2% pilocarpine has been shown to reduce pupil diameters to 2.8 (SD 0.5) mm.(47) It is important to note that it is possible to generate absolute central scotomas at low light levels by employing large corneal plane opacities. However, this will result in a very large relative scotoma that could cover the entire extent of the VF, which is not characteristic of the VF defect present in AMD.(48, 49) Because our model is designed to mimic scotoma characteristics of patients with AMD, we achieved a more localised transition between absolute scotoma and full sensitivity with smaller opacities and using high luminance levels to decrease pupil size (Figure 2 Figure 5).

We generated scotomas with diameters ranging from 10° to 32° , similar in size to those observed in moderate to advanced AMD, where scotoma diameters have been shown to vary between 1.4° and 30° .(5, 24, 48, 50, 51) Our eccentric gaze testing confirms that this experimental model is capable of generating stable central scotomas that are gaze-contingent; scanning across the visual world as the eye moves (Figure 9).

Our observed absolute scotoma diameter was larger than the predicted absolute scotoma by 33% (RMSE = 5.3°), which is anticipated when measuring scotomas using kinetic perimetry where subjects' reaction time might modulate the size of the measured scotoma. Another reason for the discrepancy between the observed and predicted scotoma sizes may relate to the near edge of the relative scotoma not providing adequate retinal illuminance or contrast to enable target visibility. When including corrections for either factor, we find excellent agreement between the psychophysically measured scotoma sizes and those predicted by our ray tracing model (slopes of observed vs predicted are close to 1.0, see Figure 8). Although both of the above factors must be present, the differences in the required pupil illumination threshold for central fixation and eccentric viewing (10% and 5%) suggest a possible contribution of fixation inaccuracy in expanding the measured scotoma size beyond that predicted by the geometrical model.

Imperfect retinal stabilization of the induced scotoma is a limitation of the present model as confirmed by the observation that subjects could, unlike actual AMD patients,(48)(52) generally see a dark central scotoma. However, anecdotal observations revealed that the scotoma sometimes disappeared and perceptual ‘filling-in’ was observed when viewing a spatially homogeneous field.(34) In spite of efforts to employ tight-fitting soft CLs and prism ballasting, subjects observed small movements in the scotomas immediately after blinking, due to CL movements during a blink.(53–55) The maximum observed 0.2 mm lens movement during a blink is predicted to result in less than 3.9° of scotoma movement. As noted by Nau,(37) hippus of the pupil also produced small fluctuations in scotoma size, which have been shown to be smallest at high light levels.(56) Our model (Figure 4) predicts that the observed 2.8° SD of scotoma diameter is consistent with a pupil diameter SD (hippus) of 0.16 mm. Previous experimental studies of hippus predicted pupil diameter SDs on this order of magnitude with small pupil diameters,(56) suggesting that fluctuations in pupil size likely contributed significantly to the measurement variability of scotoma size.

We also observed fluctuations in scotoma size most likely due to decreased fixation stability. With the CL opacity blocking the central fixation target, it was difficult for subjects to maintain fixation stability.(16–18, 20) This reduced fixation stability might explain why we found that measured scotomas were slightly larger and scotoma radii more variable when subjects attempted to fixate centrally than with eccentric fixation. The difficulty experienced in measuring scotoma size might also explain why the majority of previous scotoma simulation studies have not measured scotoma size.(34, 36, 37).

Despite these limitations, this model is still representative of AMD because subjects were prevented from using their central retina. As a result, subjects experienced the same functional consequences of central vision loss as seen in real AMD.(21, 23) Furthermore, our experimental model of AMD produced only a small amount of between-subject variability in scotoma diameter (SD = 6.01°). Significantly, this experimental model will allow for a within-subject experimental design with decreased variability (and hence a reduced need to recruit subjects) and the ability to systematically vary scotoma size.

The parallax effect seen in Figure 2 is generated by the axial separation of the central opacity in the CL and the eye’s pupil, creating a field loss that varies with the amount of pupil obstruction. A similar, but not desirable effect has been documented for the centrally transparent KAMRA (www.AcuFocus.com) corneal inlay, which provides a small pupil and increased depth of focus foveally, but an obstruction of the pupil in the near peripheral field.(57, 58) Also, the axial separation between the pupil and an implanted intraocular lens has been recently implicated in a parallax effect causing negative dysphotopsia in the far peripheral field.(59) As can be seen from our ray tracing schematic (Figure 1), small axial separations between the opacity and the eye’s pupil can result in a large region of the visual field that has a relative scotoma. The diameter of this relative scotoma can be reduced by increasing the axial separation between the pupil and opacity, as Foley, *et al* did by attaching an opaque occluder to an 8.5 cm extension arm attached to a scleral CL.(34) More detailed knowledge about retinal sensitivity along the borders of central scotomas in AMD will help guide future experimental models of gaze-contingent central scotomas.

Conclusion

The present study showed that it is possible to induce gaze-contingent absolute scotomas and hence mimic central vision loss in AMD using centrally-opaque CLs. This experimental model of AMD, however, works only if the ocular pupil is prevented from dilating and allowing light to enter the eye around the outside of the CL opacity. This simulation tool will, therefore, be ineffective at low environmental light levels, but a valuable simulation tool at high environmental light levels typical of those encountered during daytime in the outside environment.

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Figure 1.

The resulting absolute scotoma (black), and relative scotoma (grey) induced by a contact lens with a central opacity with radius h on an eye with pupil radius p . θ_1 = angle of incident ray relative to the visual axis, that, after refraction, passes from one side of the contact lens opacity to the same side of the pupil margin, θ_T = the angle of the incident ray that after refraction passes the edge of the CL opacity and the opposite pupil margin. Refer to ray tracing section for all variable definitions.

Figure 2.

Photographs taken of two eyes fitted with contact lenses containing a 3.2 mm diameter central opacity from the visual axis (A), a mid-peripheral field location (approximately 20° temporal) (B), and a far peripheral field location (approximately 40° temporal) (C).

Figure 3.

Sample calculations of the proportion of the pupil illuminated for an eye viewing a Goldmann bowl perimeter target with a background luminance of 1155 cd/m² with either a 2.8 mm or 3.2 mm diameter centrally located contact lens opacity plotted as a function of visual field angle. Pupil size was 2.04 mm (red) or 2.14 mm (green), CL opacities were 2.8 and 3.2 mm, anterior chamber depth was 3.52 mm, and corneal radius was 7.71 mm. θ_1 = the angle beyond which the pupil is partially illuminated and θ_T = the angle beyond which 100% pupil illumination is achieved.

Figure 4. Predicted absolute scotoma diameters plotted as a function of ocular pupil diameter for CL opacity diameters of 2.8 mm, 3.0 mm, and 3.2 mm.

Figure 5.

Visual field plots from the right eye of one subject fitted with a CL containing a 3.2 mm diameter central opacity. The outer isopters represent the extent of the visual field for the eye without the centrally opaque CL (blue dashed line) and with the centrally opaque lens on the eye (green line). The inner isopter (red dots and line) represents the extent of the absolute scotoma for anterior chamber depth (w) of 3.3 mm and pupil diameters (d) of: 2.7 mm, achieved with 585 cd/m^2 (Figure 5A) and 2.4 mm, achieved with 1155 cd/m^2 (Figure 5B). Each ring represents a 10° eccentricity, and blind spot mapping is also shown in green. The black circle represents the equivalent area circle of the absolute scotoma whose radius was equal to the average distance in degrees from the scotoma centre to its borders along each meridian. The centre of each plot represents the location of the perimeter bowl fixation target.

Figure 6.

Visual field plots of the right eye from four subjects wearing a 3.2 mm diameter CL opacity for Goldmann bowl luminance of 1155 cd/m^2 . Insert shows pupil diameter (d) and anterior chamber depth (w) for each case. Graph employs same design and structure as Figure 5.

Figure 7. Scatter plot showing measured absolute scotoma diameters (for all contact lens opacities and luminance levels) as a function of the difference between CL opacity and entrance pupil diameters.

Figure 8.

Figure 8A: Relationship between central measured and predicted scotoma diameter for 0% pupil transmittance (black circles), and 10% pupil illuminance (red circles) criteria. Purple triangles show the relationship between central measured and predicted scotoma diameter after removing the expected false expansion due to the dynamic perimetric methodology.

Figure 8B: Relationship between eccentric viewing measured and predicted scotoma diameter for 0% pupil transmittance (black circles) and 5% pupil illuminance (red circles) criteria. Purple triangles show the relationship between eccentric viewing measured and

predicted scotoma diameter after removing the expected false expansion due to the dynamic perimetric methodology.

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Subject 1 using a 3.0 mm CL opacity with $w=3.7$ mm and $d=2.1$ mm achieved at 1155 cd/m^2

Figure 9.

Visual field plots of the right eye for each of four eccentric fixation patterns, where Subject 1 was instructed to place one edge of the scotoma at the edge of the bowl fixation circle (see methods). Subject 1 was fitted with a 3.0 mm diameter centrally-opaque CL with a pupil diameter (d) = 2.1 mm achieved at 1155 cd/m^2 . Anterior chamber depth (w) was 3.7 mm. Graphing conventions are the same as Figure 5.

The mean (SD) of monocular measured scotoma diameters at the different fixation locations.

Table 1

		Mean Measured Scotoma Diameters (deg)				
		Fixation relative to scotoma				
		Central	Superior	Inferior	Left	Right
Perimeter Luminance Levels (cd/m ²)	10	0° (0°)	0° (0°)	0° (0°)	0° (0°)	0° (0°)
	585	18.0° (5.6°)	16.0° (3.7°)	17.4° (5.1°)	16.4° (4.4°)	17.8° (5.7°)
	1155	23.6° (5.3°)	21.2° (3.4°)	20.7° (3.8°)	21.2° (4.0°)	21.6° (4.5°)