Objective Assessment of Photoreceptor Displacement and Metamorphopsia

A Study of Macular Holes

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Background: We have developed a binocular perimetry technique for the quantitative assessment of retinal photoreceptor displacement and metamorphopsia.

Objective: To study the direction and amplitude of retinal photoreceptor displacement in eyes with idiopathic macular holes using our binocular perimetry technique.

Subjects: Five healthy control subjects and 10 patients with unilateral stage 3 to 4 macular holes in one eye and a healthy fellow eye.

Method: Kinetic perimetry using red and green filter glasses, black binocular fixation targets, red and green selective monocular stimuli (Goldmann III-4-e), and fundus image superimposition of perimetry data.

Results: We found no discrepancy between the 2 visual fields in any healthy subjects. In patients with a unilateral macular hole, the central scotoma invariably extended beyond the rim of the hole. In 8 patients, each point on the rim of the scotoma had a perceptually corresponding location in the visual field of the fellow eye that was closer to the center of the visual field. In the 2 patients with the longest duration of symptoms (>2 years), no such discrepancy was found.

Conclusions: Differential perimetry enables the objective study of retinal photoreceptor displacement and metamorphopsia. We found objective evidence for radial centrifugal photoreceptor displacement in most patients with idiopathic macular holes.

SUBJECTS AND METHODS

SUBJECTS

We examined 5 healthy subjects with normal binocular vision and 10 patients with a unilateral idiopathic macular hole in one eye and a healthy fellow eye. No participant had a history of prior eye or central nervous system disease. No participant had a refractive error greater than ±3 diopters or an intraocular pressure higher than 21 mm Hg. All healthy subjects had a best-corrected visual acuity of 1.0 or better in both eyes. All patients had a best-corrected visual acuity in their unaffected eye of 0.7 or better and no ocular disorder other than a unilateral macular hole.

THEORY

Local displacement of a group of photoreceptors in relation to the rest of the retina causes a shift in the visual field projection of the involved photoreceptors. With a healthy fellow eye and normal binocular vision prior to the onset of disease, unilateral displacement of a receptor element will lead to loss of visual field correspondence with the cortically paired receptor element in the fellow eye. Because the brain continues to assign the original visual field projection to a stimulus reaching the displaced receptor, metamorphopsia and micropsia or macropsia are induced by the discrepancy between real and perceived visual field projections. This theory assumes that no remodeling of higher perceptual mechanisms has occurred.

DIFFERENTIAL PERIMETRY INSTRUMENTATION

Simultaneous and selective binocular/monocular perimetry was made using a flat white screen at a distance of 50 cm with white incandescent background illumination adjusted to the background intensity of the Goldmann perimeter (Haag-Streit AG, Bern, Switzerland) at its lowest setting. A black cross at the center of the screen, 1°6 minutes of arc wide, served as the fixation target. Since patients with unilateral macular holes are unable to see this target with their diseased eye, a concentric circle at 6° eccentricity provided a peripheral reference for binocular fusion.

The patient was seated at a slitlamp table with a headrest. Test spectacle frames corrected for refractive status and a working distance of 50 cm were mounted together with a red filter on one eye and a green filter on the other eye. The filters fully rejected stimuli of the complementary color. Red and green monocular perimetry stimuli were provided by a red diode laser (1 mW, 670 nm, Pen-Pointer, Melles-Griot, Carlsbad, Calif) and a green helium-neon ion laser (0.1 mW, 543.5 nm, 05-SGR-810, Melles-Griot). The lasers were placed on either side of the patient's head on ball mounts, allowing full 3-axis freedom of movement by manual adjustment.

Stimulus size is equal to Goldmann Ill. Neutral density filters were added to adjust stimulus intensity to correspond subjectively to Goldmann 4 e white.

To overlay the perimeter plot on a 16 × 16-cm, hard copy-digital grayscale fundus image, the perimeter plot was photocopied onto a transparent sheet using a photocopying machine with adjustable magnification. The center of the fovea and the optic nerve head were used as reference points corresponding to the center of the visual field and the physiological blind spot.

DIFFERENTIAL PERIMETRY TEST STRATEGY

Throughout the procedure, the patient fixated on the central target. The extent of the central scotoma of the diseased eye was mapped using manual kinetic perimetry. A stimulus visible only to the diseased eye was moved slowly from the fixation target and outward along 1° of a succession of 8 meridians until the patients indicated that they saw the stimulus. Following this, an analogous mapping of the physiological blind spot was made on the same eye.

A static stimulus visible only to the diseased eye was then positioned at the rim of the central scotoma. A kinetic stimulus visible only to the healthy eye was introduced near the fixation target on the same meridian. The patient verbally directed the examiner to move the kinetic stimulus outward until it was seen by the patient to coincide with the static stimulus. The examiner marked the location of both spots on the screen using clear ink (visible only under UV illumination). The 2 sets of stimulus positions define the outlines of the objective scotoma on the diseased eye and its projection on the healthy eye, i.e., the subjective scotoma. Point reproducibility was assessed by reexamination of 2 meridians at the initial session, and by reexamination 1 day later of 2 healthy subjects and 2 patients.

Healthy subjects viewed a red fixation target invisible to the fellow eye wearing the green filter, which simulated a unilateral central scotoma. A concentric black circle at 6° eccentricity maintained fusion. A static monocular stimulus was presented at 8 points in a grid pattern extending from 1° to 8° eccentricity. For each location, a kinetic monocular stimulus seen by the fellow eye was introduced at 5° distance, whereupon the subject directed the examiner to move the kinetic stimulus outward until it was seen by the subject to coincide with the static stimulus.

As the 2 stimuli approach each other, it becomes increasingly difficult to determine their relative positions. In this situation, the test subject’s perception of the stimuli was enhanced by flashing 1 of the stimuli at a rate of about 2 Hz. Since intense fixation often leads to fading of the stationary stimulus, the patient was allowed to close the eyes for about 1 second when necessary to make the stimulus reappear. The test was completed in 15 to 20 minutes.

FUNDUS PHOTOGRAPHY

Digital black and white fundus photography in red-free illumination was made using a retina camera (TRC-50X, Topcon, Tokyo, Japan) equipped with a digital backpiece (Mega plus model 1.4, Eastman Kodak Co, San Diego, Calif) and a PC-based image handling system (Ophthalmic Imaging Systems Inc, Sacramento, Calif). The diameter of the macular hole was determined from the fundus images using the optic nerve head diameter as an internal reference, assuming a vertical diameter of 1500 μm. The extent of the scotomata was expressed by the linear dimension calculated from their fundus projections.

STATISTICAL ANALYSIS

Correlation between variables was estimated with Kendall’s rank correlation analysis for paired samples and by multiple regression analysis.

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One patient (No. 1) demonstrated concentric objective and subjective scotomata on the initial examination, whereas a second examination later in the day showed 3° horizontal displacement between the 2 scotomata. Exophoria was confirmed by clinical examination. All other patients consistently had concentric objective and subjective scotomata.

The diameter of the macular hole varied between 260 and 610 µm (Table). All patients had objective scotomata that were considerably larger than the macular hole, ranging from 180% to 310% of the diameter of the hole itself (Table).

Photoreceptor displacement was confirmed in 8 of 10 patients, each of whom had a subjective scotoma that was distinctly smaller than their objective scotoma (range, 58%-79%; Table). The discrepancy between real and perceived visual field projections is shown in Figure 1.

In 2 patients with macular holes, we found large central scotomata, but no evidence of photoreceptor displacement at the rim of the scotomata. These 2 patients had the longest duration of symptoms (>2 years). An example of a patient with equal diameter objective and subjective scotomata is seen in Figure 2.

When the Watzke-Allen test was used, all patients reported metamorphopsia, ie, seeing the projected slit as having the shape of an hourglass. Some patients constantly or intermittently noted a dehiscence of the halves of the hourglass. There was no consistent relationship between these observations and the perimetry findings, however, and all patients found it difficult to determine categorically whether dehiscence was present.

Visual acuity in patients with a macular hole was inversely correlated with the diameter of the hole ($P = .004$). After accounting for the diameter of the hole, there was no independent effect of the correlations found in paired analyses with duration of symptoms ($P = .05$), the diameter of the objective scotoma ($P = .04$), and the diameter of the subjective scotoma ($P = .04$).

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### Comments

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**Characteristics of Patients With Macular Holes**

<table>
<thead>
<tr>
<th>Patient No./Sex/ Age, y</th>
<th>Best-Corrected Visual Acuity</th>
<th>Subjective Duration of Symptoms, mo</th>
<th>Symptoms</th>
<th>Photographic Hole Diameter, mm</th>
<th>Scotoma, mm†</th>
<th>Discrepancy ‡</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Diseased Eye</td>
<td>Healthy Eye</td>
<td>Blurring, micropsia</td>
<td>0.38</td>
<td>1.2</td>
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<td>1/M/59</td>
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<td>Blurring, metamorphopsia</td>
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<td>0.50</td>
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<td>1.25</td>
<td>6</td>
<td>Blurring</td>
<td>0.54</td>
<td>1.1</td>
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<td>3/F/63</td>
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<td>1.1</td>
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<td>4/F/65</td>
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<td>0.9</td>
<td>24</td>
<td>Blurring, metamorphopsia</td>
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<td>5/F/66</td>
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<td>1.0</td>
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<td>Blurring, metamorphopsia</td>
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<td>1.1</td>
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<tr>
<td>6/F/66</td>
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<td>0.7</td>
<td>6</td>
<td>Metamorphopsia</td>
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<td>1.1</td>
</tr>
<tr>
<td>7/F/67</td>
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<td>0.8</td>
<td>5</td>
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<td>0.43</td>
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<td>8/F/69</td>
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<td>3</td>
<td>Blurring</td>
<td>0.26</td>
<td>0.49</td>
</tr>
<tr>
<td>9/M/69</td>
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<td>Metamorphopsia, micropsia, central scotoma</td>
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<td>6</td>
<td>Blurring, metamorphopsia</td>
<td>0.55</td>
<td>1.1</td>
</tr>
</tbody>
</table>

* All patients had stage 3 to 4 macular holes.
† Objective scotoma denotes extent of central scotoma on visual field of macular hole eye as superimposed on fundus image. Subjective scotoma denotes extent of projected visual field defect on healthy fellow eye.
‡ Discrepancy is evident when the subjective scotoma is significantly smaller than the objective scotoma. Plus indicates yes; minus, no.
ferential perimetry in patients with idiopathic macular holes clearly documented radial centrifugal photoreceptor displacement away from the center of the retina in 8 of 10 patients. This confirms that most idiopathic macular holes are formed by neuroretinal tissue dehiscence at the umbo and gradual distention without significant loss of tissue.¹⁻³

We have shown that idiopathic macular holes are accompanied by metamorphopsia, micropsia, and a central scotoma, but subjectively few patients complain of micropsia or manifest a well-defined central scotoma on Amsler grid testing. During biomicroscopic slitlamp examination, the patients detect deformity and usually discontinuity in a slitbeam transecting the hole.⁴ We found central scotomata extending outside the rim of the hole, indicating that the reduced retinal sensitivity over the partially detached neuroretinal cuff explains why discontinuity is seen, although presumably no significant loss of photoreceptors has occurred. We assume that a relative scotoma corresponding to the cuff area surrounds an absolute central scotoma corresponding to the hole itself, and that the depth of the relative scotoma determines whether dehiscence of the slitbeam is or is not seen.

The 2 patients without detectable photoreceptor displacement (Nos. 4 and 5) presumably had a reduction of sensitivity below threshold throughout the displaced part of the retina. Since both patients had distinct micropsia and metamorphopsia, centrifugal photoreceptor displacement must have been involved in the formation of their macular holes. Theoretically, some degree of remodeling of higher perceptual mechanisms may have occurred, but no evidence is available to support the existence of a central mechanism for the correction of irregular image transformations (metamorphopsia).

In conclusion, we have made photoreceptor displacement and metamorphopsia accessible to objective analysis by a new method, differential perimetry, which enables an accurate and detailed study of retinal diseases involving mechanical attenuation or crowding of photoreceptors, such as macular holes or macular pucker. Potential applications include the study of the response to surgical treatment intended to reposition displaced photoreceptors.

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REFERENCES


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