Diplopia Secondary to Aniseikonia Associated With Macular Disease

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**Objective:** To provide an explanation for diplopia and the inability to fuse in some patients with macular disease.

**Methods:** We identified 7 patients from our practices who had binocular diplopia concurrent with epiretinal membranes or vitreomacular traction. A review of the medical records of all patients was performed. In addition to complete ophthalmologic and orthoptic examinations, evaluation of aniseikonia using the Awaya New Aniseikonia Tests (Handaya Co Ltd, Tokyo, Japan) was performed on all patients.

**Results:** All patients were referred for troublesome diplopia. Six of the patients had epiretinal membranes and 1 had vitreomacular traction. All 7 patients had aniseikonia, ranging from 5% to 18%. In 5 of the patients the image in the involved eye was larger, and in the other 2 patients it was smaller than in the fellow eye. All patients had concomitant small-angle strabismus and at least initially did not fuse when the deviation was offset with a prism. Response to optical management and retinal surgery was variable.

**Conclusions:** Aniseikonia caused by separation or compression of photoreceptors can be a contributing factor to the existence of diplopia and the inability to fuse in patients with macular disease. Concomitant small-angle strabismus and the inability to fuse with prisms may lead the clinician to the incorrect diagnosis of central disruption of fusion. Surgical intervention does not necessarily improve the aniseikonia.


INOCULAR DIPLOPIA associated with epiretinal membrane formation and subretinal neovascular membranes has been described. The mechanism is postulated to be a mechanical distortion of the macula causing rivalry between central and peripheral fusional mechanisms. Patients with these conditions are unable to fuse when their deviation is offset with prisms owing to the continuing rivalry. We believe that aniseikonia is another condition associated with macular disease. It can contribute to the failure of prisms to eliminate diplopia.

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We have had the opportunity to follow up 7 patients with macular disease and binocular diplopia. All patients were referred for evaluation of troublesome diplopia. All shared the finding of substantial aniseikonia concurrent with their diplopia. This article attempts to explain the pathophysiology of binocular diplopia associated with macular disease and aniseikonia.

**REPORT OF A CASE**

A 42-year-old man (patient 1) was referred for a 4-year history of horizontal, vertical, and torsional diplopia. The ocular history of his right eye is clinically significant for a penetrating injury 7 years prior to the onset of diplopia, an episode of herpes zoster 7 months after the onset of diplopia, and an epiretinal membrane. He had a negative tensilon test result and a normal brain and orbital magnetic resonance imaging scans as part of the evaluation of his diplopia. His refraction and best-corrected visual acuity was +1.25 sphere 20/25 OD and +2.75 +.75 370 20/15 OS. His spectacles had 2.5 prism diopters (PD) of base up prism in the right lens and 0.5 PD base down in the left lens. He measured a total of 5 PD of left-sided hypertropia and 2 PD of exotropia in the primary position at 6 m, and 3 PD of exotropia with 4 PD of left-sided hypertropia at one-third m. These measurements include the prism incorporated in his spectacles. He had 200 seconds of arc of stereopsis with the Titmus test. There was no cycloptoria measured with the double Maddox rod test despite his subjectively reporting an intermittent awareness of torsion during casual viewing. When tested with the amblyoscope, he could briefly fuse with very limited amplitudes. With handheld prisms to offset his deviation in free space, he could momentarily superimpose the 2 images, but they quickly sepa-
PATIENTS AND METHODS

We identified and retrospectively reviewed the medical records of 7 patients who had binocular diplopia concurrent with macular disease. Six patients had epiretinal membranes; 1 patient had vitreomacular traction. Each patient underwent a full ophthalmologic and orthoptic examination including evaluation of visual acuity, pupillary response, slitlamp findings, refraction, ocular motility (including prism cover and testing, subjective torsion with the double Maddox rod test, and versions), and dilated fundoscopy. All examinations also included evaluation of aniseikonia using the Awaya (Handaya Co Ltd, Tokyo, Japan) New Aniseikonia Test. This test consists of plates containing matched pairs of red and green semicircles. They are arranged in a series in which the 2 semicircles in each pair are of different sizes, with the size difference varying in increments of 1%. The subject views the plates while wearing red-green spectacles, thus allowing the right eye to see one of the semicircles in each pair, and the left eye the other semicircle. The subject indicates the pair in which the semicircles appear to be of equal size. The actual size difference of the semicircles in that pair represents the percent of aniseikonia. In addition, fundus photographs were taken to document macular disease. Referrals to a vitreoretinal specialist were made when it was deemed appropriate; alternatively some of the patients were referred to us by vitreoretinal specialists. Prisms were prescribed in all cases for small-angle strabismus in an attempt to alleviate the diplopia.

rated. He had aniseikonia with a perceived image size that was 18% smaller in the right eye. Axial lengths were 23.76 mm OD and 22.80 mm OS.

Initially, prior to our evaluation, there had been an attempt to treat his anisometropia with contact lenses that accurately corrected his refractive error. This was tried for a 3-month period without any relief of his diplopia. Later, an attempt was made to minimize the aniseikonia by magnifying the smaller image from the right eye. Using the patient’s subjective responses to a contact lens that provided additional minus power in the right eye, combined with hyperopic correction in the right spectacle lens, a Galilean telescope was created. The patient’s aniseikonia was reduced, and he reported subjective improvement in his symptoms of diplopia for 4 months. He still was aware, however, of a subjective difference in the size of the images perceived by each eye. After 4 months, he again noticed increasing awareness aniseikonia and a subsequent recurrence of his troublesome diplopia. He then underwent successful surgery to remove the epiretinal membrane in his right eye in an attempt to relieve his diplopia. Postoperatively, his visual acuity was 20/20 OD, but he aniseikonia was only reduced to 15%. His right eye continued to see the smaller image, and he has continued to experience constant diplopia.

The Table summarizes the remaining 6 cases.

COMMENT

Epiretinal membranes are fairly common and are not usually visually significant. They are nonvascularized membranes that cover and distort the macula. They can be idiopathic. Alternatively they can be associated with other ocular conditions such as proliferative diabetic retinopathy, posterior vitreous detachment, retinal detachment, and trauma. The Beaver Dam Eye Study found epiretinal membranes present in at least one eye in 11.8% of the population. In that study the membranes were not usually associated with visual symptoms in the absence of other ocular conditions. However, epiretinal membranes are known to sometimes cause mild to severe visual impairment including decreased visual acuity and metamorphopsia.

Vitreomacular traction syndrome occurs when a partial posterior vitreous detachment with persistent anteroposterior traction distorts the posterior pole. Cystic macular changes are common in this syndrome. Binocular diplopia associated with epiretinal membranes or subretinal neovascular membranes has been described. It has been suggested that in both of these disorders the diplopia is caused by metamorphopsia or macular heterotopia creating a rivalry between central and peripheral fusional mechanisms. Prisms do not alleviate the diplopia.

Acquired disruption of central fusion, or horror fusionis, is a condition that causes diplopia in all positions of gaze and is associated with strabismus. The diagnosis should only be considered after all other causes for diplopia have been ruled out. It can occur in persons who have had prolonged sensory deprivation in one eye as can occur when a previously normal eye has an untreated monocular cataract for a prolonged period. It can also occur after severe head injury, postviral syndromes, or with other central nervous system disorders. Patients with this disorder experience constant diplopia that cannot be eliminated with prisms. They can neither fuse nor suppress the images; They have lost central fusional capabilities.

It is generally accepted that 5% aniseikonia is the limit of tolerance to permit fine stereopsis. Each of our patients had substantial aniseikonia (≥ 5%) associated with macular distortion or traction concurrent with binocular diplopia. A possible contributing factor to their diplopia can be the aniseikonia. The aniseikonia may be caused by compression or stretching of the photoreceptors. The Figure illustrates how epiretinal membranes can cause the perceived image in the affected eye to either be larger (macropsia) or smaller (micropsia) depending on whether the photoreceptors are compressed or stretched, respectively. In our series of 7 patients, 5 patients perceived a larger image in the affected eye; 2 patients perceived a smaller image in the affected eye. Although many studies show that subjects may adapt to aniseikonia, those studies may have only limited applicability to the patients in this series.
They were conducted on normal subjects with visual acuity that was correctable to normal. Patients with subnormal vision or macular distortion may not exhibit the same ability to adapt to aniseikonia as a normal subject. Also, those studies induced aniseikonia by introducing anisometropic spectacle lenses. This not only induces aniseikonia, but also an anisophoria (i.e., a phoria that changes as the gaze angle changes) due to the Prentice rule. It has been shown that subjects do manifest an adjustment in their saccadic system to compensate for this anisophoria.\textsuperscript{12} The patients in our series, however, did not have anisometropia as a cause of their aniseikonia. Hence, they would not show an anisophoria. It seems problematic to make assumptions on the ability of our patients to adapt to aniseikonia based on data obtained from normal subjects with artificially induced anisometropia.

Bielschowsky\textsuperscript{17} reported successfully treating a patient with aniseikonia by using magnifying spectacle lenses to equalize image size. We only attempted a similar treatment in patient 1, and it had limited success. Milder and Rubin\textsuperscript{8} suggest only small amounts of aniseikonia (up to 5\%) can be treated practically in this manner, and consequently we believe it would not be useful for most of the patients in our series. We did consider it for patient 6, because he had aniseikonia that only measured 5\%. It turned out that he also had a history of being intolerant of contact lenses, so we did not use this treatment.

Several patients (patients 2 and 5) reported some short-term improvement in their symptoms with prism therapy. They initially felt that when their angle of misalignment was offset with prisms in their spectacles, they found it easier to localize the object of regard in their visual environment. They still, however, noted aniseikonia and diplopia; the 2 images were superimposed. In both cases, after a short period, they no longer found the prisms helpful and discontinued their use.

Indication for surgical intervention for epiretinal membranes is usually based on visual acuity and the length of time symptoms have been present. Rice et al\textsuperscript{18} found that eyes with visual acuity better than 20/70 have less to gain from surgery than eyes with poorer visual acuity. Eyes with a long duration of symptoms of blurred vision also do less well. Rice et al do not recommend surgery for eyes with visual acuity of 20/60 to 20/80 if the goal is to improve visual acuity significantly. They do not have data to determine if surgery decreases symptoms of metamorphopsia at this acuity level.
It is obvious from our few patients that vitrectomy with membrane peeling does not necessarily alleviate the aniseikonia and diplopia. We are unable to comment on indications for surgery as a treatment for these symptoms. Our patients did not meet the visual acuity standards commonly used or recommended for epiretinal membrane surgery.

This study needs to be viewed in the light of several limitations. Because the series was derived from tertiary care, pediatric ophthalmology and strabismus practices and because we cannot calculate a denominator for the number of patients with macular abnormalities from which these 7 patients would have been derived, we are unable to comment on the relative incidence of aniseikonia in patients with macular disease. Similarly, we are unable to comment on the incidence of aniseikonia as a cause of diplopia. The biggest limitation of this study is that we have multiple possible reasons to have intractable diplopia. Epiretinal membranes cause image distortion that can prevent fusion. It is impossible therefore to tell the relative roles of aniseikonia or metamorphopsia in causing their symptoms. Probably there is an additive effect of these factors. Consequently we have avoided claiming that aniseikonia was the sole obstacle to fusion. Instead, we have considered it a contributing factor. Also, although cyclotorsion can be an obstacle to fusion that can be mistaken for a disruption of fusion, none of our patients had cyclotorsion of sufficient magnitude to cause intractable diplopia. We believe the amount of aniseikonia present in each patient was of a sufficient amount that it alone could possibly have been responsible for the patients' symptoms. We also believe aniseikonia should be considered a cause for diplopia in patients with macular disease before a final diagnosis of central disruption of fusion is rendered.

### Table

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<thead>
<tr>
<th>Double Maddox Rod, Degrees</th>
<th>Stereopsis, Seconds of Arc</th>
<th>Comment</th>
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<tr>
<td>3 Incyclotropia</td>
<td>200</td>
<td>Prism helped temporarily</td>
</tr>
<tr>
<td>0</td>
<td>Absent</td>
<td>CME postoperatively</td>
</tr>
<tr>
<td>5 Excyclotropia</td>
<td>400</td>
<td>Prior ERM surgery; aniseikonia postoperatively</td>
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<tr>
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<td>400</td>
<td>Prisms helped temporarily</td>
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<tr>
<td>0.0</td>
<td>100</td>
<td>No improvement with prisms</td>
</tr>
<tr>
<td>4 Excyclotropia</td>
<td>200</td>
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