Nonsurgical Management of Binocular Diplopia Induced by Macular Pathology

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Objective: To treat binocular diplopia secondary to macular pathology.

Methods: Seven patients underwent evaluation and treatment. All had constant vertical diplopia caused by various maculopathies, including subretinal neovascularization, epiretinal membrane, and central serous retinopathy. Visual acuity ranged from 20/20 to 20/30 in the affected eye. All except 1 patient had a small-angle, comitant hyperdeviation with no muscle paresis. Sensory evaluation demonstrated peripheral fusion and reduced stereoacuity. Neither prism correction nor manipulation of the refractive errors corrected the diplopia. A partially occlusive foil (Bangerter) of density ranging from 0.4 to 1.0 was placed in front of the affected eye to restore stable, single vision.

Results: The Bangerter foil eliminated the diplopia in all patients. Two patients elected not to wear the foil; 1 patient was afraid of becoming dependent, and the other was bothered by the visual blur. Visual acuity in the affected eye was reduced on average by 3 lines. All patients maintained the same level of sensory fusion, with only 2 having reduced stereoacuity. Symptoms returned when the foil was removed or its density was reduced.

Conclusion: Low-density Bangerter foils provide an effective, inexpensive, and aesthetically acceptable management for refractory binocular diplopia induced by macular pathology, allowing peripheral fusion to be maintained.


Inocular Diplopia secondary to macular pathology has been described previously, and when diagnosed is often left untreated. These patients classically have comitant, small-angle hyperdeviations and no cyclovertical muscle dysfunction. The diplopia does not respond to prism correction. Although the prevalence of this entity is not known, we believe it is more common than expected. The management of this unique syndrome presents a challenge for clinician and patient.

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Underlying macular diseases in the literature have included epiretinal membranes and subretinal neovascularization. When retinal wrinkling or macular derangement causes a mechanical displacement of the affected fovea, bothfoveas are no longer corresponding retinal points, and diplopia ensues.

Although effective, monocular occlusion is often an unsatisfactory option for these patients because of poor cosmesis and limitation of visual field. With this in mind, we have begun to use a graded, partially occlusive foil (Bangerter) that relieves the diplopia while still allowing peripheral fusion and acceptable cosmesis. Bangerter filters are thin, translucent, plastic foils that can be easily fixed to a spectacle lens much like a Fresnel prism. By reducing the amount of light transmitted, the foils degrade images viewed through them. They vary in strength from the most dense to the barely occlusive. The full set strengths include light perception, less than 0.1, 0.1, 0.2, 0.3, 0.4, 0.6, 0.8, and 1.0. They have been used for many years, especially in Europe, for the treatment of amblyopia and more recently in several studies to simulate cataracts and to eliminate intractable diplopia associated with strabismus.

REPORT OF CASES

Two patients are described who typify this unique syndrome and our treatment regimen. The remaining patients are described in the Table.

CASE 1

An 81-year-old man was evaluated with a complaint of vertical diplopia and metamorphopsia of 3 months’ duration. He had
PATIENTS, METHODS, AND MATERIALS

We observed 7 patients from April 19, 1994, through March 31, 1998, who had intractable binocular diplopia secondary to macular pathology (Table). These patients were examined by the same orthoptist (E.S.) and ophthalmologist (M.S.).

Patient ages ranged from 40 to 81 years, with an average age of 54 years. There were 4 men and 3 women. Underlying diagnoses included epiretinal membrane (5 patients) and choroidal neovascularization (2 patients). One of the patients with epiretinal membrane also had serious chorioretinopathy in the involved eye.

All patients underwent an ophthalmic examination, including slitlamp biomicroscopy, indirect ophthalmoscopy, and fundus contact lens inspection. Special attention was paid to the vector of gial traction using contact biomicroscopy. All patients underwent an orthoptic examination that included American Optical Vectograph slide at 6 m, Titmus stereo test at 33 cm, Bagolini striated glasses test at 6 mand at 33 cm, and Worth Four Dot test at 6 mand at 33 cm. Version assessment, Maddox double rod test, prism cover test, and Maddox rod measurements in all fields of gaze were performed. Each patient underwent testing on the synoptophore using the following superimposition slides: foveal (covering 1° of angle of vision), macular (covering 3° to 5°), and paramacular (covering 10°). In addition, retinal correspondence with after-image testing was assessed. Careful attention was paid to the patients’ symptoms, including scotomata, metamorphopsia, and image size. Amsler grid testing, subjective descriptions, and drawings were used in an attempt to document the exquisitely specific symptoms described by these patients.

We attempted to eliminate the double vision in our clinic by placing loose prisms in a Halberg clip-on over the patients’ spectacles. Patients would momentarily have single vision; however, as is typical in this syndrome, the diplopia rapidly returned. Manipulation of refractive error was also attempted. When these trials failed, a partially occlusive Bangerter foil was applied to the spectacle lens in front of the affected eye in an attempt to restore stable, single vision. The least dense foil that eliminated the diplopia was chosen. The Bangerter foil was cut to size and positioned on the entire inner lens surface (Figure). The lens was dampened with water, and the foil was pressed on, removing any air bubbles. Vision and sensory testing, including the synoptophore, were repeated with the Bangerter foil in place. Subsequent examinations included subjective assessment of whether the diplopia was relieved, and whether the Bangerter foil density could be decreased or eliminated.

A 68-year-old man was seen for a 6-month complaint of metamorphopsia and constant vertical diplopia not relieved by incorporated prisms. Results of a full medical examination performed elsewhere, including magnetic resonance imaging of the head, were unremarkable. He had age-related macular degeneration with a history of choroidal neovascularization, which was successfully ablated with argon laser photoagulation, improving his visual acuity from 20/40 to 20/20. The diplopia developed following the laser treatment. Examination revealed best-corrected visual acuity of 20/20−2 OD and 20/20 OS. Fundus examination of the right eye revealed a dry juxtafoveal chorioretinal scar with drusen and pigment atrophy in the macular region. The left eye had mild pigment degeneration without exudative changes. We measured a comitant right hypertropia of 4 PD, which was corrected by the patient’s incorporated prism spectacles. However, the patient remained diplopic. There was no evidence of a paretic muscle. He demonstrated vertical diplopia on AO Vectograph slide and results of Worth Four Dot test at near. He had fusion (results of Worth Four Dot and Bagolini tests) at near and reduced stereoaucity (Titmus stereo test, 200 seconds of arc on circles). A Bangerter foil of 0.6 density applied to the right spectacle lens relieved the diplopia. Visual acuity was reduced to 20/60 at distance and 20/50 at near. He suppressed the right eye on the AO Vectograph slide and had fusion on results of Worth Four Dot test at distance.
near he continued to maintain fusion. Stereacuity was unchanged.

Synoptophore testing before the foil placement showed normal superimposition on foveal, macular, and paramacular slides. With the foil, there was suppression of the right eye on the foveal slide, yet the patient continued to superimpose the macular and paramacular slides. He described a central scotoma of the right eye on the latter slides. At 8-month follow-up, the patient remained pleased.

The Bangerter foil cut to size and positioned on the inner lens surface.

Seven patients with intractable vertical binocular diplopia due to macular pathology underwent evaluation. No patient had monocular diplopia. Previously, binocular diplopia has been reported with preretinal1 as well as subretinal lesions.2 To our knowledge, this is the first combined series of patients with pathology at different levels of the fundus and the only report of a successful therapy in this challenging group of patients.

In all cases with epiretinal membranes, detailed fundoscopic evaluation showed that the membranes caused retinal wrinkling through the foveal region. As others1,3 have reported, 4 of 5 patients with epiretinal membrane had hyperdeviations of the involved eye. In our series, some cases appeared to have more traction inferiorly; however, there was not a uniform correlation between the hyperdeviation and the vector of glial traction. Of the 2 patients with choroidal neovascularization, 1 patient had a hyperdeviation of the affected eye and the other had no deviation (patient 5). Patient 3 with a juxtapfoveal chorioretinal scar had the largest hyperdeviation of 4 PD, yet had no evidence of retinal traction on contact lens inspection. In contrast to patients with epiretinal membrane where glial traction is visible, it is possible that the fibrotic scar was exerting subretinal traction that could not be viewed using conventional ophthalmoscopy.

The diplopia developed in 4 of the 7 patients after laser and/or retina surgery. Perhaps this is due to improved vision in the involved eye, which previously was decreased enough to preclude diplopia (eg, patients 1, 3, and 5). Alternatively, laser photocoagulation may lead to changes in the internal limiting membrane. For example, patient 6 had a long history of central serous retinopathy with multiple recurrences, but did not complain of diplopia until after laser photocoagulation. The role of retinal surgery and laser photocoagulation in causing the diplopia is not well understood and warrants further investigation. The remaining 3 patients had no retinal therapeutic intervention.

Similar to previous reports,1-3 our patients had relatively good vision, with visual acuity in the affected eye ranging from 20/20 to 20/30 at distance and near. All affected eyes were the nondominant eye, except patient 4. Every patient (except patient 5) had a comitant, small-angle hyperdeviation with no evidence of torsion or cyclovertical muscle paresis. All had normal retinal correspondence with no history of strabismus or diplopia. Despite extensive efforts, prism correction was unable to provide stable single vision. Two patients, in fact, had incorporated prisms prescribed elsewhere that did not alleviate their symptoms.

Prism correction may momentarily correct the diplopia by superimposing the foveal images; however, the intact peripheral retina is then shifted. Although there is momentary foveal alignment, the peripheral fusional

<table>
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<th>Summary of Patients*</th>
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<tr>
<td><strong>Patient No./Age, y</strong></td>
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<tr>
<td>1/81 ERM</td>
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<td>2/59 ERM</td>
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<td>3/68 NVM</td>
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<td>5/40 NVM</td>
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<td>6/59 ERM, CSR</td>
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<td>7/70 ERM</td>
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*VA indicates visual acuity; ERM, epiretinal membrane; NVM, neovascular membrane; CSR, central serous retinopathy; LHT, left hypertropia; RHT, right hypertropia; Ortho, no ocular deviation; X, exodeviation; E, esodeviation; and ellipses, test not done because patient elected not to wear foil. All patients’ unaffected eyes had VAs of 20/20; all affected eyes were the nondominant eye except in patient 4.

† Indicates at near only.

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mechanism is stronger,9,10 and both foveae are pulled apart. The diplopia then returns. We hypothesized that the only way to treat this syndrome would be to exclusively suppress the fovea of the involved eye. Anecdotally, Burgess et al described a patient who placed a small circle of surgical tape on the lower portion of her reading glasses to block the central vision in the affected eye.

All patients received a Bangerter foil of a selected density over the affected eye, and had relief of their diplopia. Five of 7 patients used the Bangerter 0.6 foil. Two patients (patients 6 and 7), however, elected not to wear the Bangerter foil. They differed from the others in that both had diplopia for more than 4 years before consultation and had learned to accept their condition. One patient was afraid of becoming “dependent” on the foil, whereas the other was bothered by the induced visual blur. Perhaps these 2 patients imply that the long-term natural course of this disorder is tolerance. However, we were impressed with the aid that the foil provided to the other patients in the early stages of the syndrome. To assess the success of the treatment, at each visit we attempted to reduce the density of the foil or remove it entirely. Patients immediately noted the return of double vision, a finding also reported by McIntyre and Fells.8

Although these patients are bothered by true binocular diplopia, they still are able to demonstrate fusion at near and stereoeacuity ranging from 140 to 400 seconds of arc. (Patient 5 had no stereoeacuity.) Sensory testing at distance yielded more variable responses. For example, patient 1 had vertical diplopia on the AO Vectograph slide, but fused with results of the Worth Four Dot test. At near, our standard sensory tests examine peripheral fusion, which remains intact in these patients. However, at distance, more central stimulation elicits vertical diplopia.

The Bangerter foil reduced visual acuity on average by 3 Snellen lines. However, we noticed a wide range in the decrease of acuity from foils of the same density. The variability in visual acuity was somewhat surprising, particularly in patient 4, who had a reduction to 20/100 at distance and near. The reason for these variable responses is not entirely clear. Mitsuyu and Zimmer7 found there was reproducible correlation between foil density and visual acuity, whereas our results compare more similarly with those of Janknecht and Funk.6

Evidence of the efficacy of the Bangerter foil comes from synoptophore testing. On the synoptophore, having corrected any preexisting deviation, 6 patients (all except patient 5) indicated superimposition using foveal, macular, and paramacular slides. With the Bangerter foil in place, these patients uniformly suppressed the affected eye on the foveal slide, but continued to maintain macular and paramacular superimposition. Furthermore, 2 patients reported a central scotoma in the involved eye when viewing the paramacular slides. Based on the results of synoptophore testing, it is our belief that the Bangerter foil is effective in eliminating the rivalry between central and peripheral fusion by degrading the image quality and inducing foveal suppression.

Sensory testing was repeated with the Bangerter foil in place. Among those who elected to wear the foil, peripheral fusion was maintained. Stereoeacuity was unaffected in 2 patients, slightly reduced in 1 patient (from 140 to 200 seconds of arc), and significantly reduced in 1 patient (140 to 4000 seconds of arc). The remainder of patients had no stereoeacuity or elected not to wear the foil. The singular advantage of the Bangerter foil over other modalities such as total occlusion is the ability of the patient to maintain peripheral fusion. This, in combination with the transparent cosmesis, makes the foil such an effective treatment.

The clinical picture in patient 5 was somewhat different from that of the others in that she had no deviation and yet experienced metamorphopsia, scotomata, and vertical diplopia while reading. We could not document diplopia on sensory testing at near. This may reflect the fact that bifoveal fixation is not necessary to demonstrate fusion on near sensory testing. We were impressed with the relief provided by the weakest density foil. Her case illustrates the multiple binocular visual complaints that many patients with macular pathology experience. For example, 4 patients had metamorphopsia in addition to the diplopia. With the Bangerter foil in place, no patient complained of distortion. A similar finding has been previously reported.8 This serendipitous finding may be due to a decrease in contrast sensitivity induced by the Bangerter foil; however, this was not quantified. We are now in the process of evaluating the foil in patients who have metamorphopsia, with or without diplopia.

To our knowledge, this is the first reported use of this modality for intractable binocular diplopia induced by macular pathology. The efficacy and cosmesis of the foil make it an especially attractive option for this complicated group of patients. The only disadvantage we encountered was that the foil lost some of its adhesiveness in extreme humidity (eg, a steamy bathroom). At this time, we are trying to develop a more permanent semifrosted lens that would duplicate the effect of the Bangerter foil.

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REFERENCES