The Dyschromatopsia of Optic Neuritis Is Determined in Part by the Foveal/Perifoveal Distribution of Visual Field Damage

Scott E. Silverman,* William M. Harr, Jr.,* Mae O. Gordon,* and Charles Kilof

Most hypotheses of acquired dyschromatopsia invoke the mechanism of selective damage to specific components of the afferent visual system to explain the predominance of red–green and blue–yellow hue-discrimination defects found in neural and retinal disorders, respectively. However, this pattern of hue-discrimination disturbance in ocular disease may vary. There are frequent exceptions which are inadequately explained by existing hypotheses. In an effort to explain the pattern and pathogenesis of acquired dyschromatopsias better, the authors examined patients with nonproliferative diabetic retinopathy (DR) and late-stage retrobulbar neuritis (RBN) using age-corrected Farnsworth-Munsell 100-hue testing and threshold static perimetry. As expected, most DR eyes showed some degree of relative blue–yellow dyschromatopsia (89%) with few showing a greater weighting towards red–green dyschromatopsia (11%). However, an approximately equal number of RBN eyes had a relative blue–yellow (48%) versus red–green dyschromatopsia (52%). For RBN, the authors found a strong association between the spatial distribution of field defect and the type of relative hue-discrimination disturbance. Eyes with greater field depression at the fovea relative to the perifovea showed a relative preponderance of red–green dyschromatopsia (68%) as opposed to blue–yellow dyschromatopsia (32%), whereas eyes with greater relative perifoveal impairment showed a relative preponderance of blue–yellow dyschromatopsia (100%). This relationship between the relative spatial distribution of visual field damage and the relative hue-discrimination deficit in RBN was statistically significant \( P = 0.002 \). Such an association was not found for DR. Rather, a preponderance of relative blue–yellow dyschromatopsia was present in nearly all DR eyes, without regard to the spatial distribution of visual field defect. Our findings suggest that the spatial distribution of damage to the foveal and perifoveal visual field is an important determinant of the resulting hue-discrimination defect in RBN but not in DR. There are fundamentally different underlying mechanisms of color vision impairment in these two classes of disease. Invest Ophthalmol Vis Sci 31:1895–1902, 1990

The pattern and pathogenesis of hue-discrimination defects in acquired dyschromatopsias are not completely understood. In 1912, Köllner\(^1\) stated that red–green color vision defects are characteristic of optic nerve disease and blue–yellow defects are characteristic of retinal disease, a hypothesis which continues to receive support.\(^\text{2,3}\) It has been proposed that this pattern of hue-discrimination defects in ocular disease reflects an underlying selective impairment of color opponent neural channels in the afferent visual system.\(^\text{4–6}\) However, the validity of this hypothesis is unproven.

We previously used "color contrast perimetry" to test the theory of selective impairment in acquired dyschromatopsias.\(^\text{7–10}\) Color contrast perimetry is a kinetic perimetric technique for examination of hue discrimination in the central 30° of the visual field.\(^\text{7}\) A computer generates colored video screen test objects that are matched in luminance to a surrounding background of a contrasting color. The saturation of the colored test objects is varied while the target:background luminance match is maintained. The application of this technique to glaucoma\(^\text{8}\) and other diseases of the optic nerve and retina\(^\text{9}\) showed that visual field defects appear equivalent (ie. have identical spatial distributions) when measured by luminance contrast, red–green color contrast, and blue–yellow color contrast perimetry. If, in fact, opponent channels are selectively destroyed in ocular
disease, then selective hue-discrimination defects should be identified by color contrast perimetry. That is to say, an eye with an acquired red-green dyschromatopsia should show defects in red-green discrimination in areas of the visual field where blue-yellow perception has been relatively less disturbed. King-Smith et al. reported greater loss of equiluminous color contrast perception than of luminance-contrast perception when using a static form of color contrast perimetry to examine a patient with a retinal scar. However, in this case the spatial heterogeneity of the equiluminous red, blue, and yellow deficits was limited to a narrow zone in and around the borders of a localized and relatively dense visual field defect.

Since relative impairment of color contrast neural channels may not be adequate to explain the preferential (relative) axes of dyschromatopsias found in acquired neural and retinal disease, the question arises of how to account for the apparently selective hue discrimination deficits so commonly found by Farnsworth-Munsell 100-hue testing in these disorders. In a previous preliminary study, we examined hue discrimination and visual field integrity in 17 patients with acquired dyschromatopsias resulting from diseases of the macula and optic nerve. A trend was found between the dyschromatopsia axis and the spatial distribution of visual field defect. Patients with predominantly blue-yellow dyschromatopsia axes tended to have visual field defects with foveal sparing, and those with predominantly red-green axes tended to have field defects directly involving the fovea. In the current study, we examined patients with nonproliferative diabetic retinopathy (DR) and late stage “recovered” retrobulbar neuritis (RBN), using Farnsworth-Munsell 100-hue testing and threshold static perimetry. We wanted to describe the patterns of acquired dyschromatopsias better and systematically test the relationship between the spatial distribution of visual field damage and the relative hue-discrimination defect.

Materials and Methods

Eighteen patients with nonproliferative DR (35 eyes) and 28 patients with late stage RBN (29 eyes) were included in the study. Inclusion criteria for the DR group included diagnosis of type I or type II diabetes mellitus, presence of background retinopathy confirmed by fluorescein angiography, visual acuity of 20/60 or better, and an otherwise normal ocular examination and history. Patients with evidence of neovascularization or significant macular edema were excluded. The average age of the DR group was 43.1 yr (range, 20–61). Average visual acuity was 20/21.7 (range, 20/15–20/30).

All patients in the RBN group had had an episode of RBN in the past 2–48 months, either of an idiopathic nature or associated with known disseminated multiple sclerosis. The diagnostic criteria included a history of rapid visual loss associated with abnormalities in color vision, visual field, and pupillary reaction accompanied by an ophthalmoscopically normal fundus, followed by a period of spontaneous visual recovery. Cases suggestive of an inflammatory, vascular, or infectious cause were excluded. Visual acuity of 20/60 or better was required for inclusion. The average age of the RBN group was 36.4 years (range, 18–67). Average visual acuity was 20/27.1 (range, 20/15–20/60).

All patients were assessed with automated threshold static perimetry and Farnsworth-Munsell 100-hue color vision testing. With one exception, all testing within the DR group was bilateral, with each eye tested separately. One DR patient satisfied inclusion criteria in only one eye, and unilateral testing was done. With one exception, all testing within the RBN group was unilateral, confined to the eye satisfying the inclusion criteria. In one RBN patient both eyes met the criteria, and bilateral testing was done.

Perimetric Testing

The macula program of the Humphrey field analyzer (Allergan Humphrey, San Leandro, CA), with foveal option enabled, was used for visual field examination. This program measures 16 points in the central 4.2° of the visual field. Field data were age corrected using normative values from the Statpac program (Allergan Humphrey). Foveal normative data are not available through the Statpac program and were provided especially to us by Allergan Humphrey. The primary purpose of visual field measurement in this study was to compare the extent of foveal versus perifoveal impairment. To accomplish this, the foveal sensitivity defect was compared with the defect of the perifoveal region. The four most central extrafoveal points of the macula program were used to define perifoveal sensitivity. These points are at a distance of 1° vertical and 1° horizontal from the point of fixation. The depth of visual defect at each location was defined as the difference between the value observed and the value expected for the age-related norm. To compare the impairment at the fovea to the perifovea, the defect at the fovea was subtracted from the average defect at the four perifoveal locations. This value, “perifoveal-minus-foveal defect,” quantifies the relationship between relative foveal and perifoveal impairment. For instance, a central visual field defect with greater impairment at the perifovea than at the foveal projec-
tion would result in a positive perifoveal-minus-foveal defect value, representing a distribution of relative foveal sparing. Conversely, a central scotoma with relatively greater damage at the fovea would yield a negative perifoveal-minus-foveal defect value, representing a distribution of relative foveal involvement. To summarize the overall integrity of the central visual field, the “central visual field defect” was calculated by averaging the defect values at the fovea and the four perifoveal points. These points are within the central 1.4° of the visual field.

Color Vision Testing

The Farnsworth-Munsell 100-hue test was used to evaluate color vision. All testing was monocular. Error scores were calculated from both anchors for each box, and cap 85 was not used. The relative axis of dyschromatopsia was determined using a quadrant analysis technique described by Smith et al. This method partitions the total error score into blue-yellow (caps 1–12, 34–54, and 76–84) and red-green (caps 13–33 and 55–75) partial error scores. A relative axis of dyschromatopsia is calculated by subtracting the square root of the red-green partial error score from the square root of the blue-yellow partial error score. (The square-root value is used because this transformation yields a normal distribution in error scores.) A positive relative axis value corresponds to a relative blue-yellow dyschromatopsia, while a negative value denotes a relative red-green dyschromatopsia. This method was previously applied to a group of 87 normal subjects, and an approximate age-correction function was calculated by regression analysis (age-corrected relative axis = relative axis – 0.061 × (age in yr) + 2.035). This function was used to correct the relative axis values for the effect of aging on hue discrimination (ie, age-related tritanopia). For purposes of the data analysis, each eye was treated as an independent observation, and the relative axis of dyschromatopsia was treated as a continuous variable.

Results

For all subjects a strong positive relationship was observed between red-green and blue-yellow partial-error scores (Fig. 1). A linear correlation was highly statistically significant for RBN (r = +0.89, P < 0.001) and DR (r = +0.75, P < 0.001).

For RBN, a positive correlation (r = +0.42, P = 0.023) was found between the depth of central visual field defect and the magnitude of color vision impairment as defined by the square root of total error score (Fig. 2, top). For DR (Fig. 2, bottom), central visual field defect did not show a significant correlation with the magnitude of color vision impairment (r = +0.013, P = 0.94).

For all RBN eyes, the mean foveal defect was 5.50 decibels (dB), and the mean perifoveal defect was 5.04 dB. For all DR eyes, the mean foveal defect was 2.16 dB, and the mean perifoveal defect was 3.82 dB. Table 1 summarizes the relationship between red-
and the foveal and peri-foveal defects (Table 1).

Pearson correlation coefficient (r) for associations between foveal and peri-foveal defect and the square root of red/green (RG) and blue/yellow (BY) partial error scores in retrobulbar neuritis (29 eyes) and diabetic retinopathy (35 eyes)

<table>
<thead>
<tr>
<th>Comparison</th>
<th>Correlation (r)</th>
<th>P value</th>
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</thead>
<tbody>
<tr>
<td><strong>Retrobulbar Neuritis</strong></td>
<td></td>
<td></td>
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<tr>
<td>Perifoveal defect and RG (Sqrt)</td>
<td>0.37</td>
<td>0.05</td>
</tr>
<tr>
<td>Perifoveal defect and BY (Sqrt)</td>
<td>0.40</td>
<td>0.03</td>
</tr>
<tr>
<td>Foveal defect and RG (Sqrt)</td>
<td>0.36</td>
<td>0.06</td>
</tr>
<tr>
<td>Foveal defect and BY (Sqrt)</td>
<td>0.20</td>
<td>0.30</td>
</tr>
<tr>
<td><strong>Diabetic Retinopathy</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Perifoveal defect and RG (Sqrt)</td>
<td>0.08</td>
<td>0.63</td>
</tr>
<tr>
<td>Perifoveal defect and BY (Sqrt)</td>
<td>0.03</td>
<td>0.88</td>
</tr>
<tr>
<td>Foveal defect and RG (Sqrt)</td>
<td>0.06</td>
<td>0.75</td>
</tr>
<tr>
<td>Foveal defect and BY (Sqrt)</td>
<td>0.08</td>
<td>0.66</td>
</tr>
</tbody>
</table>

The average relative axis value for all 29 RBN eyes (14 eyes) axes of dyschromatopsia (Fig. 3, Table 2). The spatial distribution of visual field defect showed a strong relation to the relative axis of dyschromatopsia (Pearson r = +0.38, P = 0.07) and nonparametic methods (Table 3) still confirmed a significant relationship between the distribution of visual field damage and the relative dyschromatopsia axis. Most DR eyes had relative blue-yellow axes (89%) with a few having relative red-green axes (11%) (Fig. 4, Table 4). The average relative axis for all DR eyes was —0.15, indicating that red-green and blue-yellow partial errors were nearly equally represented within the RBN group as a whole. Of the RBN eyes, 76% (22/29) showed relative foveal impairment (ie, perifoveal-minus-foveal defect < 0), and 24% (7/29) had relative foveal sparing (ie, perifoveal-minus-foveal defect > 0). The average visual acuity for RBN eyes with a relative blue-yellow axis (20/23.9; range, 20/15–20/50) was better than acuity for those with a relative red-green axis (20/30; range, 20/15–20/60), but this difference was not statistically significant (Mann-Whitney U test, P = 0.24).

For the patients with late-stage RBN the relative spatial distribution of visual field defect showed a strong relation to the relative axis of dyschromatopsia (Fisher’s exact test, P = 0.002). All seven RBN eyes with field defects characterized by foveal sparing had blue-yellow axes (Fig. 3, Table 2). However, those with relative foveal impairment showed mostly relative red-green defects (68%). Additionally, all 15 RBN eyes with relative red-green defects displayed relative foveal impairment. Further statistical analysis by linear regression also demonstrated a relationship between relative foveal/perifoveal defect and the relative axis of dyschromatopsia (Pearson r = +0.30, P = 0.11). All eyes in the RBN group had visual acuity of 20/30 or better except for two with 20/50, and two with 20/60 acuity. When these four eyes were excluded, statistical analysis by parametric (Pearson r = +0.38, P = 0.07) and nonparametric methods (Table 3) still confirmed a significant relationship between the distribution of visual field damage and the relative dyschromatopsia axis.

Comparison Correlation (r) P value

<table>
<thead>
<tr>
<th>Spatial distribution of visual field defect</th>
<th>Relative dyschromatopsia axis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Foveal sparing</td>
<td>n = 7</td>
</tr>
<tr>
<td>Foveal impairment</td>
<td>n = 0</td>
</tr>
</tbody>
</table>

Fisher’s Exact Test, P = 0.002.

Relative foveal sparing

Relative foveal impairment

n = 6

n = 7

n = 15

Fisher’s Exact Test, P = 0.015.

Table 3. The spatial distribution of visual field defect and the relative dyschromatopsia axis in retrobulbar neuritis eyes with visual acuity of 20/30 or better (25 eyes)

<table>
<thead>
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<th>Spatial distribution of visual field defect</th>
<th>Relative dyschromatopsia axis</th>
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<tbody>
<tr>
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<td>n = 6</td>
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<td>Foveal impairment</td>
<td>n = 0</td>
</tr>
</tbody>
</table>

Fisher’s Exact Test, P = 0.015.

Relative foveal sparing

Relative foveal impairment

n = 7

n = 12

Fisher’s Exact Test, P = 0.015.
was +2.16, reflecting the predominance of blue-yellow errors within the DR group. Of the DR eyes, 57% (20/35) had relative foveal sparing, and 43% (15/35) had relative foveal impairment. For DR, the relative axis of dyschromatopsia was unrelated to relative foveal/perifoveal defect (Fisher’s exact test,  = 1.00;  = 0.13,  = 0.44; Fig. 4, Table 4). Nearly all eyes in the DR group showed relative blue-yellow axes of dyschromatopsia, without regard to the spatial distribution of visual field damage in and around the fovea.

**Discussion**

It is generally accepted that the dyschromatopsia associated with DR is predominantly along a blue-yellow axis

Although red-green defects have been reported.

Our finding that 89% of nonproliferative DR eyes had a relative blue-yellow axis of dyschromatopsia is consistent with previous studies. Roy et al found abnormal hue discrimination in 54% of DR eyes, with most errors along a blue-yellow axis. Bresnick et al found abnormal hue discrimination in 54% of DR eyes, with most errors along a blue-yellow axis.

Red-green defects have been reported in RBN, both acutely and after visual recovery. Cox found 83% of RBN patients to have red-green dyschromatopsias. Perkin and Rose reported red-green axes in 76% of eyes with abnormal color vision. However, blue-yellow defects have also been reported. Mullen and Plant found discrimination errors to be distributed across all hues without a tendency to show either relative red-green or blue-yellow defects. We also found relative red-green and blue-yellow defects in nearly equal frequency. These findings indicate that relative hue-discrimination defects in the later stages of RBN can occur along either a blue-yellow or red-green axis and suggest that RBN may represent an important exception to the classifications of acquired dyschromatopsias put forth by Kölln and Verriest. It is important to note that we used a quantitative and age-corrected method of relative axis determination; nearly all previous studies of color vision defect in RBN used qualitative assessment strategies without age correction. This may partially explain the discrepancy between our findings and those of previous studies.

Figure 1 shows the strong association found between blue-yellow and red-green partial-error scores for the DR and RBN groups. A shift is seen to the right for the DR data relative to the RBN values. This shift represents the disparity along the relative dyschromatopsia axis between these two groups. In other words, for a given amount of red-green discrimination error, the DR group had more blue-yellow errors than the RBN group. However, substantial red-green and blue-yellow errors were seen in both DR and RBN. It appears that simultaneous, significant impairment to red-green and blue-yellow color discrimination occurs in both DR and RBN. It is the relative proportion of this impairment that defines the relative dyschromatopsia axis.

Our data for RBN eyes are most similar to those of Mullen and Plant. Although there is a strong, linear correlation between blue-yellow and red-green partial-error scores for RBN eyes (Fig. 1), there is no apparent tendency toward a relative prevalence of one form of hue-discrimination defect over the other at any overall level of dyschromatopsia. Rather, the relative weight of one form over the other appears to be randomly distributed, with a nearly equal representation for the two forms.

Investigations of the spatial distribution of visual field defects in RBN have been inconsistent. Burde and Gallin found decreased sensitivity in perifoveal regions with normal foveal sensitivity (ie, foveal sparing) in all nine RBN patients studied. In our study the opposite trend was found. Relative foveal involvement was seen more frequently than foveal sparing (76% versus 24%). Others found all regions of the central visual field equally affected. The distribution of visual field involvement in optic neuritis, therefore, appears variable.

**Table 4.** The spatial distribution of visual field defect and the relative dyschromatopsia axis in diabetic retinopathy (35 eyes)

<table>
<thead>
<tr>
<th>Spatial distribution of visual field defect</th>
<th>Relative dyschromatopsia axis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Blue/yellow</td>
</tr>
<tr>
<td>Relative foveal sparing</td>
<td>n = 18</td>
</tr>
<tr>
<td>Relative foveal impairment</td>
<td>n = 13</td>
</tr>
</tbody>
</table>

Fisher’s Exact Test,  = 1.00.
It is important to note that the distribution of color perception in the central 2° of the visual field is not homogeneous. Human psychophysical experiments have found significant variations in color perception in this region. Although red–green hue-discrimination sensitivity is roughly constant throughout the central 2°, sensitivity to blue light is reduced dramatically at the central fovea (approximately 30') compared with the perifovea.24,23 Cytochemical techniques show a definite heterogeneous cone distribution in the primate retina, consistent with human psychophysical findings.26,27 Although there is a high density of red and green cones across the entire central 2° of the primate visual field, blue cones are nearly absent at the fovea (approximately 25') and increase in density peripherally, reaching a peak at about 75°–1.5° eccentricity.26

Pigment tests of hue discrimination like the Farnsworth-Munsell 100-hue test use colored caps which subtend approximately 2° of the visual field. Spatially heterogeneous damage in this restricted area of the visual field, centered at the foveal projection, would be expected to have a profound effect on the magnitude of relative hue-discrimination defects, as measured by such tests. This relationship was confirmed for RBN (Fig. 2, top) but not for DR (Fig. 2, bottom). The dissociation of relative foveal–perifoveal luminance-contrast defect and relative hue-discrimination defect in DR is not easily explainable and suggests a different pathophysiology of color vision loss in DR than that for RBN.

Diseases of the retina and optic nerve can differentially involve the fovea or perifoveal regions of the visual field. Central scotomas caused by macular disease frequently preserve foveal function.28 Similarly, glaucoma selectively impairs the function of arcuate nerve fiber bundles, producing extensive loss of peripheral portions of the central visual field, often without any disruption of foveal function. Compressive and demyelinating neuropathies on the other hand more frequently produce impairment of papillomacular nerve fibers with a corresponding impairment in foveal function. Optic neuritis, specifically, frequently impairs foveal function, but as previously noted, the distribution of field loss is variable.

We proposed that the spatial distribution of damage to the central visual field may be an important determinant of the color vision defect in ocular disease.29 For instance, a disease process that predominantly destroys afferent channels subserving the fovea is likely to destroy a disproportionate number of red–green discrimination channels and would be expected to result in a relative red–green axis of dyschromatopsia. On the other hand, a disease predominantly affecting extrafoveal regions is likely to destroy a disproportionate number of blue–yellow discrimination channels, preserving red–green discrimination at the fovea; it would, therefore, be expected to produce a blue–yellow color defect. Our knowledge of the general pattern of acquired dyschromatopsias supports this idea. Glaucoma and retinopathic disease often preserve foveal function and generally produce relatively greater blue–yellow hue-discrimination defects. Compressive and demyelinating neuropathies generally impair foveal function and often have relatively greater red–green hue-discrimination defects.

Marré30 proposed the concept of “localization-fixation-color vision defect” in an attempt to explain the basic phenomenon of acquired color vision defects. This theoretic framework explains acquired dyschromatopsias based on the anatomic depth of the disease process (pre-receptor, receptor, or post-receptor) and the integrity of foveal fixation. The theory predicts, for instance, that optic nerve disease (a postreceptor process) generally results in a red–green dyschromatopsia, but this depends on whether foveal fixation is maintained. If foveal fixation is not maintained (eccentric fixation), a red–green defect is predicted due to the lower sensitivity of red–green color vision mechanisms (ie, lower density of red–green cones) outside the fovea. If foveal fixation is preserved, then a blue–yellow defect is predicted, due to a higher susceptibility to damage of the blue color vision mechanism compared with the red–green color vision mechanisms.

Although our results are largely consistent with this theoretic model, there are some inconsistencies. In contrast to a prediction of the Marré model, we found RBN eyes with relative red–green axes to have visual acuity impairment no different from those with relative blue–yellow axes. It seems implausible that the eyes with relative red–green axes had eccentric fixation, since most (12 of 15) of the eyes had average visual acuities of 20/30.0, and none had acuity worse than 20/60. We suggest that the spatial distribution of damage to the central field of vision is a primary determinant of the relative hue-discrimination defect in RBN, independent of the fixation mode.

For DR, color vision defects showed no relation to the spatial distribution of central visual field damage. Instead, blue–yellow defects were present in nearly all DR eyes, without regard to the distribution of foveal–perifoveal damage (Table 4, Fig. 4). This indicates that the dyschromatopsia of DR is determined by factors other than the spatial distribution of central visual field damage. The pathophysiology of color vision impairment in retinopathic and neuropathic disease is likely to be fundamentally different, considering that these disease processes occur at dif-
ferent anatomic levels and by entirely different pathogenetic mechanisms. The pathophysiology of damage to vision by demyelinating optic neuropathies is known to be located at a postreceptor level, the myelinated portions of ganglion cell axons in the retrobulbar optic nerve. Diabetic retinopathy on the other hand could affect visual function at all three levels of the Marré model. There is evidence that DR eyes have damaged blue-yellow discrimination at both prereceptor and receptor levels. Selective depression of short wavelength sensitivities has been reported in diabetic eyes even before the onset of clinically detectable DR changes, suggesting a possible receptor dysfunction. Using blue-yellow color perimetry corrected by a measure for ocular media light absorption at short wavelengths, found evidence that there is also a significant prereceptor contribution to the reduced blue-yellow contrast sensitivity in DR eyes. Since we did not measure the ocular media absorption properties of each DR eye, it is possible that individual variations in the ocular media absorption properties may have obscured an underlying relationship between the spatial distribution of visual field damage and the dyschromatopsia axis in the DR group.

Our results support the concept that the spatial distribution of visual field damage is a significant determinant of the pattern of hue discrimination defect in RBN. Both foveal and perifoveal damage were found to be associated with red-green discrimination errors, but only perifoveal damage correlated with blue-yellow errors (Table 1). This result is consistent with previous studies that report a low density of blue cones in the central fovea. We also found a relationship between the spatial distribution of visual field damage and the relative dyschromatopsia axis (Table 2, Fig. 3). Eyes with a relative preponderance of blue-yellow hue discrimination defects tended to show greater perifoveal versus foveal damage, compared with those having relative red-green defects. These results indicate that the relative axis of dyschromatopsia seen in RBN is related to the spatial distribution of damage in the foveal and perifoveal visual field. Although our results do not refute the possibility of selective opponent channel damage (ie, relative loss of blue-yellow or red-green channels at the same location), it appears that the spatial distribution of damage to the central visual field is an important determinant of the hue-discrimination defect found on the later stages of RBN.

Key words: optic neuritis, diabetic retinopathy, visual field, color vision, dyschromatopsia

Acknowledgment

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References