Temporal Contrast Sensitivity Loss in Primary Open-Angle Glaucoma and Glaucoma Suspects

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The need for more sensitive tests for the early detection of compromised visual function in glaucoma is established by anatomic and psychophysical evidence of damage occurring to optic nerve fibers in eyes with normal visual fields. The results are reported of temporal frequency testing on 51 glaucoma suspects without visual field loss in either eye, 52 glaucoma patients with visual field deficits in the tested eye, and 11 normal subjects. Modulation transfer functions were obtained using a sinusoidally flickering 5° spatially uniform white field viewed with central fixation and plotted at six frequencies from 5–30 Hz. The results showed a frequency-specific sensitivity loss centered at 15 Hz and a nonfrequency-specific mean sensitivity loss, that was greater, on average, in glaucoma patients than in suspects. Sensitivity losses of both kinds were seen in most glaucoma patients, but only in a minority of glaucoma suspects. About 12% of suspects were indistinguishable from the lowest performing one third of these glaucoma patients. A smaller number of suspects appeared to form a second mode in the frequency distribution for temporal sensitivity at 15 and 25 Hz. In patients with glaucoma, age was found to be a significant factor associated with the magnitude of mean sensitivity loss. Age was not a significant factor contributing to sensitivity loss in individual suspect data as measured by regression analysis, but it contributed to a small and consistent sensitivity loss across frequency for group-averaged data in those older than 55 years of age. Invest Ophthalmol Vis Sci 32:2931-2941, 1991

Primary open-angle glaucoma is defined by a progressive loss of optic nerve fibers in the presence of an open iridocorneal angle and is presumed to follow from chronically elevated intraocular pressure. Visible diagnostic signs of optic nerve insult include cupping of the optic disc and defects in the retinal nerve fiber layer. Functional signs of damage are provided by psychophysical tests, with clinical kinetic and static visual field sensitivity currently used.

The place of kinetic and static perimetry in the diagnosis and treatment of glaucoma rests on the assumption that significant physiologic compromise in the neural visual fibers will appear as a corresponding loss of visual field sensitivity. Despite the success of visual field testing in glaucoma, evidence has accumulated that progressive changes in the optic disc and retinal nerve fiber layer may occur before the onset of glaucomatous visual field loss. In addition, there is histologic evidence that significant anatomic damage may be sustained by the optic nerve fibers before or disproportionate to the visual field deficits. Experimental glaucoma in monkeys resulted in greater damage to larger and medium-sized optic nerve fibers compared with the damage sustained by smaller fibers. This finding implies that psychophysical tasks selectively mediated by larger fibers may show greater sensitivity to early glaucomatous optic nerve damage than tasks mediated by a population of smaller fibers.

There is extensive psychophysical evidence in color, spatial, and temporal vision that damage to optic nerve fibers is present in glaucoma suspects without conventional visual field loss or signs of progressive optic disc cupping. For color vision, significant deficits were reported in up to 50% of glaucoma suspects in studies of color discrimination, color matching, threshold wavelength sensitivity, and wavelength discrimination, with differential deficits in the blue spectral region or in blue–yellow discrimination. Similar results showing deficits for glaucoma suspects in the absence of significant visual field loss were reported for spatial frequency performance, temporal frequency response, and a temporal discrimination task. However, these techniques have not achieved general clinical acceptance, motivating the search for more sensitive methods.

Tyler presented the most complete work on tem-
temporal frequency response in glaucoma. Using a 5° si-
inusoidally flickering field, he showed that up to 90% of a selected combined group of glaucoma patients and suspects (46 eyes of 41 patients) had significant losses in temporal frequency sensitivity near a specific frequency of 35 Hz. Selection of glaucoma patients and suspects was made on the basis that they had mild or no visual field losses on Goldmann perimetry. In addition, 75% of these patients had “marked” visual field loss in the untested eye. Temporal frequency loss was recordable at both central and 20° peripheral visual field locations. Tyler’s results agree with earlier work in finding a temporal frequency deficit in a small number of glaucoma patients21 and with other studies using methods of flicker-fusion perimetry that also report deficits in temporal performance in early stages of glaucoma when Goldmann visual fields are normal.22,23

We analyzed the temporal frequency response of a larger group of glaucoma patients and suspects than was reported by Tyler in an effort to evaluate group trends of potential clinical significance. Our goal was to identify glaucoma suspects without visual field loss, whose performance on this test more strongly resembled that of glaucoma patients than it did that of the rest of the suspect group. We hypothesized that this subgroup might be at higher risk for developing glaucomatous optic nerve damage as measured by other conventional means.

Materials and Methods

Subjects

The patient population was recruited through the Glaucoma Service Diagnostic Laboratory of the Wills Eye Hospital. Informed consent was obtained from each subject according to the guidelines set by the Institutional Review Board. All patients included in this study underwent extensive diagnostic testing, including Octopus visual fields, Program 32 (Interzeag, Schlieren, Switzerland), tonometry, measurement of anterior chamber depth, optic disc photographs, color testing (100-hue test), Snellen acuity, blood pressure, and temporal contrast sensitivity testing. In all, temporal modulation transfer function (TMTF) data were analyzed from 130 patients. These included 51 suspects selected on the basis that they had no visual field loss in either eye or other evidence of glaucomatous progression. Most suspects had a history of intraocular pressure (IOP) greater than 22 mm Hg. Almost one half of our suspects (24 of 51) had a history of treatment for elevated IOP or were receiving treatment at the time of testing. The visual acuity of suspects ranged from 20/15 to 20/50; only two subjects had a visual acuity worse than 20/30. Most (44 of 51) had an acuity of 20/25 or better. The average age of the suspect group was 50.9 years ± standard deviation of 14.0 (range, 21–76 years). The suspects were judged to have no visual field loss in either eye using a criterion requiring a defect to have at least two or more contiguous test points more than 8 dB below the age-corrected normal threshold, excluding optic disc and rim points. Using this criterion, 52 glaucoma patients (mean age, 61.6 ± 11.1) had visual field loss. The diagnosis of glaucoma was based on progressive change typical of glaucoma in either visual fields or in the appearance of the optic nerve head or both.

Eleven normal subjects were recruited from hospital employees who were naive to the temporal frequency modulation test and to most psychophysical testing techniques. All had 20/20 visual acuity or better and no history of elevated IOP. The average age of the normal group was 38.3 years ± 15.5 (range, 20–62 years). The normal subjects all were tested monocularly in both eyes with the right eye tested first.

Equipment and Stimulus Conditions

The stimulus was a 5° spatially uniform white field centered on the fovea presented in Maxwellian view through a 2-mm artificial pupil. For patients with pupil sizes greater than 2 mm (all normal subjects and suspects and most glaucoma patients), the artificial pupil eliminated varying retinal illuminance from changes in the aperture of the natural pupil. Sinusoidal flicker was introduced into the field at a fixed frequency by means of a rotating polarizer against a fixed polarizer in one of the two field channels. Flicker frequency was computer controlled, and the depth of modulation was varied from 0–100% by means of a second polarizer. The peak amplitude of the modulated channel was set at twice the amplitude of the unmodulated channel to maintain constant average illumination throughout the range of modulation depth. The Maxwellian view apparatus used a tungsten light source with a color temperature of approximately 2800° K. The test field illumination was calculated to be 690 trolands as seen through the 2-mm artificial pupil. This placed the stimulus high enough in the photopic range so that the small amount of pupil variation that could occur for miotic patients below 2 mm diameter would have little effect on sensitivity.

Experimental Procedure and Scoring

A modified method of limits (from below threshold only) was used in which the experimenter gradually increased the amount of flicker presented in the initially uniform field (a point well below threshold or at 0% modulation) until the patient signaled the first
perception of flicker. After a practice session, ten trials were averaged to estimate the threshold value at each of nine temporal frequencies ranging from 5–60 Hz (5, 10, 15, 20, 25, 30, 40, 50, and 60 Hz). Although data were gathered at 40, 50, and 60 Hz, these data are not included in the analysis that follows because of problems with possible age-dependent effects and performance artifacts at higher frequencies. At frequencies approaching the high-frequency resolution limit (critical flicker-fusion value [CFF]), performance was limited in all observers by a ceiling effect. Even though flicker can be seen in the field on some trials in this frequency range, an increasing number of trials (as frequency is increased) will yield no perception of flicker, even at 100% modulation. This effect will tend to force all observers toward the same performance level and reduce trial-to-trial response variability. The effect will occur at lower frequencies for those most affected by loss of optic nerve function in the glaucoma and suspect groups.

Threshold at each frequency was defined as the average of the ten trials at that frequency. Before testing, the concept of the test was explained, and emphasis was placed on the need to respond to the first sign of flicker in the field. A practice session consisted of showing the patient examples of obvious flicker (as reported by the patient) and no flicker (zero modulation) and then presenting ten practice trials at 15 Hz with feedback on performance to encourage a sensitive response criterion.

The setting of the potentiometer encoding the position of the 90° rotating polarizer (which controlled modulation depth) was read automatically by the computer at each response of the patient and stored on a computer disk. The computer program translated this voltage reading into percent modulation and printed the averaged threshold values and standard deviations for each frequency. A graphic representation of each patient's TMTF response also was produced at the end of each session.

Results

Response Variation as a Function of Age

Our results for a small group of normal subjects are shown in Figure 1. The average age of this group (38.3 years) was lower than that of the glaucoma and suspect patient groups, even though the age range spanned 20–62 years. The difference in average age between the normal and patient groups posed potential problems for performance comparisons between these groups (for subjects up to 55 years of age10). Tyler16 found age effects to be minimal at lower temporal frequencies (< 15 Hz), and they increased only gradually at higher frequencies. The Tyler study suggested that age effects would not be a major factor at lower temporal frequencies. However, because the ages of our patients extended substantially beyond the range studied by Tyler, we concede there may be a possible effect of age in our results. The possible effects of age in our study were minimized by restricting analysis to a frequency range of 30 Hz and below and by making intergroup comparisons only at frequencies below 20 Hz.

Furthermore, we attempted to evaluate aging effects in our data by analyzing results from the suspect patient group under the conservative assumption that age effects would be the same or greater in this group compared to normal. Larger age effects might be caused by any latent glaucomatous process that itself is a function of age. Figure 2 shows a plot of sensitivity versus age for the 51 suspects at 5, 15, and 30 Hz with the best-fitting linear-regression lines. Although the regressions appear to show a general tendency toward a mild decrease in sensitivity with age, none of the correlations of sensitivity versus age was significant (P > 0.05). However, in the group of glaucoma patients, all six correlations of sensitivity with age were signifi-
Age Effect for Suspects (n=51)

Fig. 2. Figure 2 plots temporal frequency sensitivity versus age of glaucoma suspects at 5, 15, and 30 Hz, along with best fitting regression lines, to illustrate the effects of aging on sensitivity.

Figure 2 shows significant effects for the six temporal frequencies. The significant correlations ranged from 0.22–0.31 with significant levels from $P < 0.05$ to $P < 0.01$.

An additional way of looking at possible age effects in the patient data is to separate the glaucoma and suspect patients into older and younger subgroups by age as shown in Figure 3. A division point of age 55 years was chosen because it is approximately at this age that accelerating visual deficiencies, probably related to the aging process, have been noted. The suscepts showed only a small difference in sensitivity between the subgroups. However, these differences were consistent across frequency. The glaucoma subgroups showed a much larger age effect that was maximal in the midfrequency range.

Mean Temporal Frequency Sensitivities

The mean temporal frequency sensitivity for glaucoma suspects with no visual field loss was compared with that for glaucoma patients with visual field loss in Figure 1. The mean sensitivity for the normal subjects also is shown for comparison. The difference function generated by subtracting the glaucoma sensitivity values at each temporal frequency from the mean suspect values is shown in Figure 4. This function revealed a frequency-specific loss peaking at 15–20 Hz and a loss of mean sensitivity spread across frequencies. Comparison of both suspects and glaucoma patients with normal subjects showed a peak loss located at about 15 Hz.

Glaucoma Suspects

The suspect group was ranked by temporal frequency sensitivity at 15 Hz and then subdivided into

Fig. 3. Age-related differences in temporal frequency sensitivity for the glaucoma patient and suspect groups. Suspects are shown in the top half of the Figure separated into two subgroups at age 55. Glaucoma patients are separated into two similar subgroups in the lower part of the Figure.

Fig. 4. Difference function showing the comparative loss in temporal frequency sensitivity for glaucoma patients compared to suspects.
six approximately equal subgroups. The actual number of subjects in each subgroup was eight for the highest three sensitivity subgroups and nine for the lowest three (Fig. 5). The subgroups ranked lower in mean response at 15 Hz also showed a change in shape of the temporal frequency function toward that of a low-pass filter. An important feature of Figure 5 is the separation of the lowest ranking suspect subgroup from the upper five subgroups. The upper five subgroups were covered largely by the 95% normal confidence bounds in the frequency range around 15 Hz. Subgroup 5 also showed a significant difference compared with normal in the region near 15 Hz.

The differences from normal noted in Figure 5 are illustrated in Figure 6 by plotting the six difference functions generated by subtracting the mean normal values from the flicker sensitivity values of the six suspect subgroups. Results from testing above 20 Hz are not shown to avoid possible age effects.

Figure 7 shows a histogram of sensitivity values at 15 Hz for the suspect group along with the 95% confidence limits set by the normal data at this frequency. A total of 16 suspects (28%) had sensitivities below the lower 95% probability limit. However, six of these (12%) appeared to be separated from the main suspect group into a possible secondary mode of the distribution. Histograms for 10 Hz showed a similar pattern, with five suspects apparently separated into a low sensitivity subgroup. Histograms for 20, 25, and 30 Hz also showed similar tendencies toward separation of a second mode on the low sensitivity end of the distribution.

Because drug treatment for elevated IOP might affect visual performance, an evaluation of the treatment variable in the suspect group was made by comparing the distribution of sensitivities at 15 Hz for the treated and untreated subgroups. Comparison of the
The distribution of temporal frequency sensitivity for glaucoma suspects at 15 Hz. The solid curve estimates the distribution of sensitivity values for normals, under an assumption of an underlying Gaussian distribution, with the 95% confidence limits indicated by the vertical dashed lines and shaded areas. The dashed curves estimate the positioning of two apparent sub-distributions, which are also assumed to be Gaussian. The distribution suggested to be at the far left contains the six suspects who most resemble glaucoma patients in their response characteristics.

The difference functions in Figure 9 show a tendency toward loss of high frequency sensitivity, but the greatest loss is centered at 15 Hz especially for Ranks 3 through 6. The values above 20 Hz are not shown because of possible age effects. The pattern of temporal sensitivity loss shown by the difference function for Subgroup 6 of the suspect group strongly resembled that of the three lowest-ranking glaucoma subgroups.

Correlation of Sensitivity Loss Across Frequency

A correlation matrix was calculated to estimate the degree of independence of loss across temporal frequency. The sensitivities at each frequency were correlated with those at each of the 5 other frequencies generating 15 correlations for the 6 temporal frequencies for each subject group. The normal subjects showed a pattern of significant correlations for closely

histograms revealed no significant differences. This observation was supported by the summary statistics. The means of the two groups were virtually the same at -0.025 for treated compared with -0.024 for untreated patients, with standard errors of 0.06 and 0.04, respectively. Variance was somewhat greater for the treated group (0.076) compared with the untreated group (0.047).

Glaucoma Patients

Similar to the suspect group, the 52 glaucoma patients with visual field loss were ranked by sensitivity at 15 Hz and subdivided into six approximately equal subgroups (Fig. 8). The number of subjects in each subgroup was eight for the highest two subgroups and nine for the lowest four. Figure 9 shows the differences in sensitivity from normal by subgroup. The top subgroup had a sensitivity profile similar to normal. However, Subgroups 2 through 6 departed significantly from the normal pattern. Only Subgroups 1 and 3 showed peak sensitivity at 15 Hz similar to normal; Subgroups 2, 4, 5, and 6 had a sensitivity peak at 10 Hz. Subgroup 6 showed low-pass characteristics, with a virtually flat response between 5 and 10 Hz and a steady decline in sensitivity at higher frequencies. The projected CFF value for this rank appeared to be near 40 Hz. Another feature of the functions from Subgroups 2 through 4 was the tendency of crossings to occur at both lower and higher frequencies than the ranking frequency of 15 Hz. This tendency for crossing was not seen for the suspect subgroups and may reflect reduced correlations for sensitivities of neighboring frequencies in the glaucoma patients relative to suspects.

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Frequency Specific and Interocular Loss

Two statistics were calculated to evaluate the distribution of suspect group sensitivities further. First, there was a difference between temporal frequency sensitivities at 15 Hz and 5 Hz (called the 15-5 difference), plotted in the histogram in Figure 10. This statistic highlights the loss in sensitivity at 15 Hz relative to the smaller loss seen in the average sensitivity functions at lower frequencies. The 95% confidence limits were estimated by calculating the same statistic for the normal group. One subject was deleted from the normal group who had a difference of more than three standard deviations below the mean compared with the other ten normal subjects. Exclusion of this aberrant value changed the left-hand 95% confidence limit from -0.048 to 0.115 log sensitivity units. Using the data thus corrected, 16 of the 51 suspects showed values below the normal limit, with some tendency toward the appearance of a secondary peak below a difference value of about 0.

The second statistic was the difference in sensitivity between the two eyes; this is plotted in histogram form in Figure 11. The distribution for this statistic showed five suspects separated from the main distribution by more than three standard deviations. Sixteen additional suspects were grouped near the 95% confidence limits.

Gaussian Assumption for Sensitivity Distributions

Interpretation of apparent outliers from the temporal frequency sensitivity distributions relies on the assumption that the underlying distributions for nor-
Fig. 11. Distribution of inter-eye differences in temporal frequency sensitivity for glaucoma suspects. The superimposed curve is an estimate the distribution of sensitivity values for normals similar to Figure 10. Five suspects appear to be separated from the main distribution, lying more than three standard deviations from the mean.

Table 1. P-values for test of sensitivity distribution shape against the Gaussian assumption

<table>
<thead>
<tr>
<th>Temporal frequency</th>
<th>Glaucoma suspect</th>
<th>Glaucoma</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 Hz</td>
<td>0.42</td>
<td>0.49</td>
</tr>
<tr>
<td>10 Hz</td>
<td>0.55</td>
<td>0.17</td>
</tr>
<tr>
<td>15 Hz</td>
<td>0.038*</td>
<td>0.35</td>
</tr>
<tr>
<td>20 Hz</td>
<td>0.81</td>
<td>0.037*</td>
</tr>
<tr>
<td>25 Hz</td>
<td>0.020*</td>
<td>0.015*</td>
</tr>
<tr>
<td>30 Hz</td>
<td>0.33</td>
<td>0.0008*</td>
</tr>
<tr>
<td>40 Hz</td>
<td>0.26</td>
<td>0.0004*</td>
</tr>
</tbody>
</table>

* Significant at the $P < 0.05$ level.
tion. The same cannot be said for the distribution of sensitivities in the glaucoma group. For glaucoma patients, departure of the sensitivity distribution from normal at 20 Hz and above could not be corrected by removing outliers. This result is consistent with the induced changes in temporal frequency sensitivity in most glaucoma patients.

The difference in sensitivity for suspects at 15 Hz compared with 5 Hz (Fig. 10) reflected a reduced low frequency decline in sensitivity. Such a decline is expected in normal subjects but is less apparent in glaucoma patients and in some suspects. Failure of the low frequency decline to occur, or conversely, failure of a mid-frequency sensitivity peak to appear, is not normal. Sixteen suspects fell below the 95% confidence limit set by normal subjects. Although only one of these obviously was separated from the main distribution, there was a tendency toward occurrence of a secondary mode below a difference value of 0. Ten of the 51 suspects did not have such a decline in low frequency sensitivity compared with one apparently aberrant normal value below 0.

A tendency toward appearance of a secondary mode also occurred in the distribution of interocular difference in sensitivity at 15 Hz for suspect eyes (Fig. 11). For this statistic, the expected difference was 0, with a 95% confidence limit for normal subjects on either side of 0 of about 0.25 log units. Five suspects appeared to be separated from the main distribution with differences greater than three standard deviations from the normal mean. The position of these suspects in the distribution suggests they were at a somewhat higher risk of having further functional visual deficits.

Our analysis of statistical distributions and our conclusions concerning the appearance of aberrant values in the distributions for suspects was based on an assumption that the underlying population distributions for normal performance are Gaussian. As shown in the results, the underlying distributions for our normal subjects at all temporal frequencies were indistinguishable from a Gaussian distribution. For suspects, two of seven distributions were not Gaussian if all suspects were included. However, these two distributions become Gaussian if a small number of outliers are removed (n = 3). For glaucoma patients, the Gaussian assumption was upheld only for the three lowest temporal frequencies and not for the four highest. For these patients, removal of outliers did not make the distributions normal.

IOP

The effect of varying IOP on temporal contrast sensitivity measurements in our study was not investigated. However, elevated IOP was shown by others to have a measurable reversible effect on temporal contrast sensitivity in normal subjects. The IOP also was reported to have a reversible effect in ocular hypertensives, 50% of whom showed improvement in performance from an abnormal sensitivity level to within the normal range during a time period of 3 months after treatment initiation. The clinical significance of the reversibility of any IOP-related performance deficit was not clear. However, the magnitude of a reversible IOP-related effect may correlate with susceptibility of the optic nerve to permanent damage and disease progression. The importance of this factor for interpreting psychophysical deficits in suspects is important and merits further investigation.

Age Effects

Our analysis was affected potentially by age differences between our experimental groups. The difference in average age between the normal and patient groups poses particular problems unless it can be shown that the change in performance expected with age was small compared with the magnitude of the effects attributed to the disease process. Normal data for this study were gathered with the expectation that age-related changes in temporal response were minimal, particularly at lower temporal frequencies and under the conditions of our test. This assumption was supported in part by the preliminary results of Tyler and strengthened by a later study designed to measure the change in performance expected with increasing age (but only up to the age of 55 years). The later Tyler study, in particular, showed no aging effect from 16–55 years of age for a sinusoidally varying 5-Hz uniform stimulus. A small decline in sensitivity with age of ~0.029 log units per decade was evident at 20 Hz and increased to ~0.048 log units at 36 Hz. These declines were small compared with the differences in average performance shown at lower temporal frequencies between our normal and our glaucoma and suspect groups in the same age range. For example, the difference in sensitivity at 15 Hz between normal subjects and suspects was 0.18 log units; between normal subjects and glaucoma patients, it was 0.38 log units; and between suspects and glaucoma patients, it was 0.20 log units. However, the Tyler study cannot be extrapolated to those older than 55 years of age, and the question of age effects after this age still must be left open.

Analysis of our own suspect data for ages older than 55 years also support the conclusion that age effects are minimal for the experimental conditions we used, especially for temporal frequencies below about 30 Hz. Nevertheless, we minimized analysis that de-
recommended on comparison with the normal data because of the age difference between these data and those of the patients. Wherever possible and appropriate, our major findings were supported by comparison of glaucoma patient results to those of glaucoma suspects.

**Duration of Disease**

The results in Figure 3, showing a much greater performance difference related to age for glaucoma patients than for suspects, may be interpreted as showing an effect related to duration of disease. However, other factors in these data also may contribute, notably the 10-year difference in average age between the suspect and glaucoma groups. It is possible that age alone is an important factor in determining vulnerability of the optic nerve to damage. Unfortunately, information on the duration of disease in these patients was difficult to establish with sufficient accuracy to justify further analysis.

**Damage to Larger Fibers**

We also considered how well our results agreed with the finding in monkeys of differential damage to large and medium-sized optic nerve fibers compared with relative sparing of smaller fibers. The observation in this study that the distribution of optic nerve fiber sizes shifted toward smaller values in glaucoma would appear to predict a differential loss of function at higher temporal frequencies. The larger and faster fibers tend to carry information from relatively larger, transient type, achromatic receptive fields. In support of these results, there was some indication in our study that the earliest temporal frequency loss may occur above 40 Hz. However, this frequency region provided the least reliable data for comparison with the normal group in our study, both because of possible age effects and because of the performance ceiling effect noted in our results.

The magnitude of the temporal frequency sensitivity loss in the 15–20-Hz region in a small percentage of suspects makes them most resemble the temporal frequency response pattern of most glaucoma patients. We hypothesize that these patients, who do not yet show measurable visual field loss, may be in the highest risk category for developing glaucomatous optic nerve damage in the future.

**Key words:** open-angle glaucoma, glaucoma suspect, temporal frequency, contrast sensitivity

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**References**


