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Motion Perception Is Abnormal in Primary Open-Angle Glaucoma and Ocular Hypertension

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Several lines of evidence suggest that the large optic nerve fibers, which form the magnocellular retinocortical pathway, are preferentially susceptible to early glaucomatous damage. It is evident from studies of the functional architecture of the visual system that the magnocellular pathway underlies the global perception of motion. Therefore, we have developed a psychophysical technique for assessing motion detection thresholds in patients with ocular hypertension (OHT) and primary open-angle glaucoma (POAG). For this purpose we employed a dynamic random dot display that contained varying degrees of a coherent motion signal embedded within a background of random motion noise. We used this technique to measure motion thresholds in POAG patients (n = 37), OHT patients (n = 14), and age-matched controls (n = 39). Motion thresholds were elevated by 70% for the POAG group and 44% for the OHT group relative to controls. In these same patients, no significant deficit in form discrimination was found as measured by Pelli-Robson charts. Our results demonstrate that significant motion perception deficits are evident in POAG and OHT. These findings support the suggestion that significant and selective damage to the magnocellular pathway occurs in OHT and POAG and indicate that motion threshold testing may reveal preclinical optic nerve disease in early POAG. Invest Ophthalmol Vis Sci 31:722–729, 1990

The visual loss associated with early optic nerve damage often goes undetected in primary open-angle glaucoma (POAG) because a large proportion of the optic nerve fibers must be lost before a visual deficit can be detected with traditional perimetric techniques.1–2 This observation has generated considerable interest in the nature of early optic nerve damage in this disease. In histologic studies of glaucomatous optic nerve damage in humans3 and monkeys,4 Quigley has observed a selectively greater loss of large optic nerve fibers. Although no fiber size appears to be totally spared by the glaucomatous process, optic nerve fibers larger than the mean diameter are more depleted than smaller diameter fibers.

Investigations of the functional architecture of the human visual system indicate that the large and small diameter optic nerve fibers underlie two distinct functional pathways. Large optic nerve fibers provide the principle retinal input to the magnocellular visual pathway, which is considered to be primarily involved in the global interpretation of spatial organization based upon motion and depth discrimination.5–7 Small optic nerve fibers supply the majority of the retinal input to the parvocellular visual pathway, which is thought to be primarily involved in the perception of color and form.5–7 Relative to the parvocellular pathway, the magnocellular pathway is preferentially sensitive to low spatial and high temporal frequency stimuli.8

A number of psychophysical and neurophysiologic studies have provided support for the hypothesis of selective magnocellular damage in glaucoma. Psychophysical studies have demonstrated that temporal resolution9 and dynamic contrast sensitivity10,11 are depressed in POAG and ocular hypertension (OHT) patients. Evoked potential studies in humans12,13 and monkeys14 have found evidence of high temporal frequency and low spatial frequency attenuation of visual responses in patients with POAG and OHT. These findings are consistent with the concept of preferential damage to the high temporal resolution, low spatial resolution, magnocellular pathway. If the magnocellular pathway is preferentially sensitive to damage in POAG, then the study of motion perception, which is primarily mediated by this pathway, may be useful in describing functional deficits asso-
ciated with the early glaucomatous process. In summary, there is compelling histological, psychophysical and electrophysiologic evidence that the magnocellular visual pathway is preferentially sensitive to early glaucomatous damage. However, it is not clear whether certain other visual functions primarily mediated by the magnocellular pathway (i.e., motion and depth perception) are abnormal in POAG and OHT. In addition, the extent to which the loss of magnocellular function exceeds the loss of parvocellular function has not been quantified.

The aim of this study was to assess the functional integrity of the pathway mediating motion perception (i.e., the magnocellular pathway) in POAG and OHT in relation to the pathway mediating form discrimination (i.e., the parvocellular pathway). We examined motion thresholds and form discrimination sensitivities in OHT and POAG patients. We also evaluated the relationship between deficits in motion perception and the degree of visual field loss in POAG patients.

Materials and Methods

Subjects

Thirty-seven patients with POAG, 14 patients with OHT, and 39 age-matched controls were tested. POAG and OHT patients were drawn from the glaucoma service of Washington University Medical Center. Controls were volunteers from the local community. Each subject received a comprehensive visual evaluation, and only those meeting the inclusion criteria (Table 1) were enrolled in the study. POAG subjects were additionally required to have open angles, by gonioscopy, and no evidence of secondary glaucoma (e.g., peripheral anterior synechias or contusion angle deformity). No restrictions were placed on patient medications or surgical history for glaucoma, but aphakic and pseudophakic eyes were specifically excluded.

OHT subjects were especially selected for being at a high statistical risk of developing visual field loss based upon the logistic regression model formulated by Hart et al. This model uses a weighted combination of patient age, intraocular pressure, cup to disc ratio, and family history to scale the risk of developing glaucoma. Only OHT patients with a relatively high risk factor (average = 0.74 ± 0.22) were included. Intraocular pressure ranged from 18 to 35 mm Hg at the time of testing for this group.

The average age of the POAG group (57.4 ± 8.0) did not show a statistically significant difference from that of the age-matched controls (56.8 ± 9.0). However, the average age of the OHT group (63.3 ± 8.9) was slightly greater than that of the controls. The average visual acuities of the POAG group (20/24.5, range: 20/15–20/40), OHT group (20/22.9, range: 20/15–20/30), and control group (20/25.2, range: 20/15–20/40) were not significantly different (P > 0.30).

Random Dot Motion Display

The motion stimulus was produced on a NEC Multisync CRT under control of an 8 Mhz IBM-PC compatible computer, using software developed by the authors. The display subtended 60° x 60° of the visual field. Each dot measured 16 min in diameter and had a luminance of 8.87 cd/m². The luminance of the background was 0.034 cd/m² and the contrast was 99.2%.

In the random dot display, an array of 100 randomly located dots is plotted in rapid succession on the CRT. This array of dots is replaced by a new array of randomly located dots, with each successive replot occurring at a frequency of 11.5 Hz. When viewing this display, a perception of random noise is produced (Fig. 1, left) since there is no net motion in this display. If, however, a subset of the dots is replotted at a fixed spatial offset (0.5°) in a common direction (up, down, left, or right), then a coherent motion signal is produced, embedded within the background of random noise (Fig. 1, center and right). The per-

### Table 1. Inclusion criteria for control, primary open-angle glaucoma (POAG), and ocular hypertension groups (OHT)

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Controls</th>
<th>POAGs</th>
<th>OHTs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>40–79 yr</td>
<td>40–79 yr</td>
<td>40–79 yr</td>
</tr>
<tr>
<td>Visual field</td>
<td>Full and normal</td>
<td>History of glaucomatous defect</td>
<td>Full and normal</td>
</tr>
<tr>
<td>Visual acuity</td>
<td>20/40 or better</td>
<td>20/40 or better</td>
<td>20/40 or better</td>
</tr>
<tr>
<td>IOP (at initial diagnosis)</td>
<td>19 mm Hg or less</td>
<td>21 mm Hg or more (3 or more occasions)</td>
<td>21 mm Hg or more</td>
</tr>
<tr>
<td>Ocular history and examination</td>
<td>Negative (except POAG)</td>
<td>Negative (except OHT)</td>
<td>Negative (except OHT)</td>
</tr>
</tbody>
</table>

Risk of developing field loss = \( \frac{1}{1 + e^{\text{logit} \left[ \frac{\text{risk factors}}{\text{risk factors}} \right]}} \)

\[
\text{Risk factors} = (14.64 + (-1.48 \times \text{fhx}) + (-12.20 \times \text{cdr}) + (-0.16 \times \text{iop}) + (-0.07 \times \text{age}))
\]

fhx = family history of glaucoma (neg = 0, pos = 1)

cdr = vertical cup to disc ratio

iop = intraocular pressure in mm Hg

age = subject age in years

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COHERENT MOTION (upward)

0% Coherence

25% Coherence

50% Coherence

fixed spatial offset in a common direction (e.g., upward as shown by arrows, center and right). This produces a coherent motion signal embedded within the background of random motion noise. The percentage of dots in coherent motion can be varied between 0% and 50%, producing graded intensities of the motion signal.

Procedure

The subjects were seated in a darkened room and viewed the display monocularly at 16.7 cm. One eye of each subject was tested while the other eye was covered with a patch. Prior to testing, intraocular pressure was measured by applanation tonometry and pupil diameter was assessed by visual inspection. Sixteen trials were presented at each of 11 motion coherence levels, ranging from 0% to 50% coherence, using the method of constant stimuli. The trial presentation order was randomized for each subject. Subjects were required to indicate the direction of perceived motion with a joystick, using a four-alternative, forced choice technique. The stimulus was presented for a duration of 4 sec in each trial, after which time the screen blanked and the subject was required to respond. This forced response initiated the progression to the next trial. If the subject responded within the 4 sec stimulus presentation period, the screen was immediately blanked and progression to the next trial was initiated. There was a 1 sec pause between trials and the entire test took approximately 12 min.

Form Discrimination Testing

For psychophysical testing of form perception, we used the Pelli-Robson chart (Clement Clarke Inc., Columbus, OH). This chart uses the Sloan letter set with characters at various contrasts. The charts are produced by a computer-generated, variable-dot-density half-tone typesetting process. Although the individual letters are large, subtending 2.8° at the viewing distance of 1 m, they also contain distinct (i.e., high spatial frequency) edges. Thus, visual processing of high spatial frequency information, a parvocellular function, should play a major role in this form discrimination task.

Form discrimination sensitivity was measured in all OHT subjects and a subset of the control (27 of 39) and POAG (25 of 37) subjects. The same eye as tested for motion threshold was assessed. All testing was monocular with the other eye occluded. Standard testing conditions and procedures were used. Subjects were seated 1 m from the chart which was uniformly illuminated to produce an 80 cd/m² chart luminance.

Visual Field Scoring

In order to evaluate the relationship between the magnitude of visual field loss and motion thresholds in the POAG group, the most recent visual fields were collected from each patient’s clinical record and scored for severity of defect. All patients were tested with either Goldmann kinetic perimetry, Humphrey automated static perimetry (program 30-2), or Octopus automated static perimetry (program 34). A masked observer (WMH) scored the visual fields using the following categories of field defect severity: (1) no defect; (2) nasal step; (3) paracentral scotoma; (4) arcuate scotoma; (5) quadrant defect; and (6) hemi-field defect.

Results

For each subject, the percent of correct responses was determined at each coherence level. The percent of correct responses was plotted as a function of mo-
tion coherence and probit analysis was used to produce a best fit curve. The motion threshold, defined as the percent coherence necessary for 75% correct responses, was extrapolated from the best fit psychometric function (Fig. 2). The average motion thresholds for each group were then calculated. In the control group, the average motion threshold was 11.2 (% coherence), while the average motion threshold for the POAG group was 19.2. For the OHT group, average motion threshold was 16.1. These threshold elevations represent increases of 71% and 44% for the POAG and OHT groups, respectively. Both of these threshold elevations were significant statistically (Table 2).

Results of motion threshold testing from a representative subset of the OHT and POAG groups are shown in Figure 3. An elevated motion threshold is apparent both as a decrease in the slope and as a shift to the right of the psychometric function relative to control subjects (refer to Fig. 2). Many POAG patients showed markedly elevated motion thresholds but in others motion thresholds were either normal or only moderately elevated (Fig. 4). For the OHT group, a bimodal distribution of motion thresholds was observed (Fig. 4). In this group, most of the motion threshold values were within the normal range but a small subset of the patients exhibited markedly elevated thresholds.

The average contrast sensitivities on the Pelli-Robson chart was calculated for each group. Average form discrimination sensitivities were nearly identical for the control, OHT, and POAG groups (Table 3), indicating that form discrimination was not depressed in either the OHT or POAG group.

The relationship between the severity of visual field defect and the motion threshold in the POAG group was assessed. Since visual fields were scored as an ordinal variable, nonparametric analysis was used (Fig. 5). This analysis failed to reveal a statistically significant association between these two measures (Spearman rho = 0.09, \( P = 0.62 \)).

The association between motion threshold and both pupil size and intraocular pressure was assessed using linear regression analysis. No significant relationships were evident (\( P > 0.10 \) for all groups). A similar regression analysis was used to investigate the association between subject age and motion threshold (Fig. 6). For the control group, age did not show a statistically significant association (\( r = 0.18, \ P = 0.27 \)) with the motion threshold, and this was also true for the OHT and POAG groups.

**Table 2.** Average motion thresholds for the control, ocular hypertension (OHT), and primary open-angle glaucoma (POAG) groups (the OHT and POAG groups showed statistically significant motion threshold elevations relative to controls)

<table>
<thead>
<tr>
<th>Group</th>
<th>( N )</th>
<th>Average motion threshold</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>39</td>
<td>11.2 ± 4.0</td>
</tr>
<tr>
<td>OHT</td>
<td>14</td>
<td>16.1 ± 7.9*</td>
</tr>
<tr>
<td>POAG</td>
<td>37</td>
<td>19.2 ± 7.4†</td>
</tr>
</tbody>
</table>

* Statistically different from control (\( P = 0.03 \)) by \( t \)-test.
† Statistically different from control (\( P < 0.001 \)) by \( t \)-test.

**Discussion**

In an effort to better elucidate the pattern of optic nerve damage and the pathogenesis of the associated visual loss in glaucoma, we examined motion and form perception in patients with POAG and OHT. We found that motion perception thresholds were elevated in both groups of patients while form discrimination sensitivities were normal. These results are consistent with the findings of previous studies which have been interpreted as indicating a selective sensitivity of the magnocellular visual pathway to glaucomatous damage. While more generalized optic nerve damage also may have been present in these patients, it was not severe enough to influence either
Fig. 3. Representative motion threshold results for primary open-angle glaucoma (POAG) and ocular hypertension (OHT) groups. POAG patient #218 (top left) and OHT patient #239 (bottom left) displayed marked elevations in motion threshold as represented by a decrease in the slope and a shift to the right of the psychometric curve relative to a typical control (refer to Fig. 2). POAG patient #227 (top center) and OHT patient #031 (bottom center) showed moderate elevations in motion threshold. Normal motion thresholds were seen in POAG patient #228 (top right) and OHT patient #112 (bottom right).

form discrimination as assessed by Pelli-Robson charts (Table 3) or Snellen visual acuity, each of which was normal for both the POAG and OHT patients. Nevertheless, this may simply reflect a low sensitivity of both the Pelli-Robson and Snellen charts to glaucomatous visual dysfunction, rather than less damage to the parvocellular pathway.

Dynamic random dot displays have been employed extensively in the investigation of motion perception in nondiseased human eyes and have many advantages for the study of motion perception. When random dot displays are used, measures of motion perception are not confounded by the influence of form and position dependent mechanisms. This type of display also allows for the intensity of the motion signal to be precisely varied while keeping other stimulus parameters constant (e.g., mean luminance, and contrast, as well as spatial and temporal frequency). In the only previous attempt to examine motion processing in glaucoma, Fitzke found that peripheral displacement thresholds were elevated in patients with POAG and OHT. However, peripheral displacement thresholds cannot be assumed to reflect only motion sensitivity since form- and position-dependent mechanisms also may be involved.

Our results indicate that motion thresholds are significantly elevated in POAG (Table 2). However, there was considerable overlap in motion thresholds with individual control and POAG patients, indicating that a subset of the POAG patients had normal motion thresholds (Fig. 4). This is not surprising...
since we monitored the motion threshold with a large field (60°) stimulus and were, therefore, summing responses over both normal and pathologic regions of the visual field. Among the OHT patients, motion thresholds also were elevated significantly relative to the control group (43%) although the effect was less pronounced. Interestingly, while a majority of the OHT patients fell within normal limits, a distinct subgroup exhibited markedly elevated thresholds (Fig. 4).

Epidemiological evidence suggests that 0.5-2.0% of OHT patients with mild to moderate elevations in intraocular pressure (ie, 21-35 mm Hg) will develop glaucomatous field loss each year.21 The percentage of OHT patients in our sample with significant elevations in motion threshold (21%) is similar to the percentage thought to develop glaucoma within 10 years. The motion perception defects seen in this subset may represent subclinical glaucomatous optic nerve damage, but further testing will be necessary to adequately evaluate this issue.

Table 3. Form discrimination sensitivity as measured by the Pelli-Robson contrast sensitivity chart for the control, ocular hypertension (OHT), and primary open-angle glaucoma (POAG) groups (form discrimination sensitivities were not depressed relative to controls for either the OHT or POAG groups).

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>Form discrimination sensitivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>27</td>
<td>1.66 ± 0.07</td>
</tr>
<tr>
<td>OHT</td>
<td>14</td>
<td>1.69 ± 0.11</td>
</tr>
<tr>
<td>POAG</td>
<td>25</td>
<td>1.66 ± 0.14</td>
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We measured intraocular pressures at the time of motion threshold testing and examined the relationship between this value and the motion threshold. No significant associations were revealed for the OHT, POAG or control groups. This result indicates that short-term variation in intraocular pressure within the physiological range does not appear to be associated with changes in motion perception thresholds.
However, this result does not rule out the possibility of a relationship between the average long-term intraocular pressure and the motion threshold.

In the POAG group, motion thresholds did not show a significant relationship to the severity of visual field defect. POAG patients with early visual field defects showed equivalent motion thresholds to those with more advanced defects (Fig. 5). This may indicate that the primary damage to motion perception occurs early in the glaucomatous disease process, perhaps as part of the diffuse (as distinct from focal) loss of visual function, as described by Drance and co-workers.22-24 Alternatively, the association between field defect severity and motion threshold may be a subtle one, requiring the examination of a larger sample size, a wider range of field defects and more quantified field analysis.

When the motion threshold data were analyzed as a function of age, no statistically significant age effect was found for any of the subject groups (Fig. 6). However, thus far we have examined motion thresholds over a relatively restricted subject age range (40-74 years). Ball and Sekuler have previously reported a small but statistically significant difference in motion sensitivity between a group of young (average age = 21 years) and elderly (average age = 68 years) subjects.25 It remains to be determined whether this discrepancy in results is due to methodologic differences or the truncated age range of our sample. There is a significant age-related deterioration in many visual functions, including spatial contrast sensitivity,26-28 temporal contrast sensitivity,29,30 visual acuity,27,31 and visual field sensitivity.32-34 This deterioration in visual function has been attributed in part to the loss of optic nerve fibers and retinal ganglion cells that occurs in normal aging.35-37 If, in fact, motion perception thresholds do not deteriorate significantly with age, this would suggest that the neural mechanism responsible for the perception of motion (ie, the magnocellular pathway) is relatively spared by the aging process or has a greater physiological reserve.

In summary, we have used a dynamic random dot display to measure motion perception thresholds in OHT and POAG patients. This psychophysical technique appeared to be unaffected by pupil size and subject age. We found significant elevations in motion thresholds in POAG and OHT patients. In these same patients we found no significant deficit in form discrimination. These findings support the suggestion that significant and selective damage to the magnocellular pathway occurs in POAG and OHT and indicate that motion threshold testing may reveal preclinical optic nerve disease in early POAG.

Key words: motion perception, parallel processing, glaucoma, ocular hypertension

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