Effects of Glaucoma and Aging on Photopic and Scotopic Motion Perception

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\textbf{Purpose.} To examine the effects of primary open-angle glaucoma and normal aging on visual sensitivity for targets known to bias responses from the magnocellular visual processing stream.

\textbf{Methods.} Contrast sensitivity was measured for the detection and direction discrimination of low-spatial-frequency (0.5 cyc/deg), drifting (4–24 Hz) sinusoidal gratings in 15 patients with glaucoma (mean age, 58.7 years), 14 age-matched control subjects (mean age 55.8 years), and 10 young control subjects (mean age, 24.4 years). As a control, sensitivity was measured for the detection of stationary stimuli. Stimuli of 4.7° square were presented at either 0° eccentricity or at 20° along the nasal horizontal meridian, under both photopic and scotopic levels of lighting.

\textbf{Results.} Across a wide range of conditions, the ability to detect and discriminate visual motion declined significantly ($P < 0.05$) with increasing age, whereas the ability to detect stationary patterns was generally unaffected. The rate of decline was adequately described by a simple linear function. Control studies showed that the age-related motion sensitivity losses could not be attributed solely to decreases in retinal illuminance associated with increasing age. Of note, however, there were no significant differences in mean sensitivity between glaucoma and age-matched control groups for any of the conditions used.

\textbf{Conclusions.} Even under conditions believed to bias the response of the visual system to the magnocellular pathway, glaucoma subjects could not be reliably differentiated from control subjects on the basis of mean sensitivity to motion stimuli. The findings have two broad implications: first, that substantial neural loss specific for motion perception occurs during the processes of normal aging, and second, that sensitivity to motion targets per se may not be a useful indicator of neural integrity in the early stages of glaucoma. (\textit{Invest Ophthalmol Vis Sci.} 2000;41:325–335)

The prediction that motion-specific sensitivity losses may provide an early indicator of primary open-angle glaucoma (POAG) is based on the assumptions that magnocellular (M) cells are lost preferentially from the glaucomatous eye during the early stages of the disease and that the M pathway plays an important role in conveying motion information. A number of reports suggest that large ganglion cells,\textsuperscript{1–3} and in particular M cells,\textsuperscript{4–5} are lost preferentially throughout the glaucomatous retina, particularly in the early stages of the disease.\textsuperscript{1} M cells provide the dominant input, via the striate cortex, to the middle temporal (MT) area (V5).\textsuperscript{6–7} This area contains large numbers of cells highly selective for stimulus speed and direction of motion and for this reason many have supposed that MT plays an important role in primate motion perception.\textsuperscript{8–12} As such, although motion selectivity is not a property of retinal ganglion cells, damage to the M system at the level of the optic nerve head in POAG could result in a reduced ability to analyze stimulus motion.

In support of this hypothesis, a number of studies report that motion perception is disrupted in POAG and ocular hypertension. POAG subjects often demonstrate elevated minimum displacement ($D_{\text{min}}$) thresholds for dot and line targets,\textsuperscript{13–15} decreased sensitivity for frequency-doubled motion stimuli,\textsuperscript{16–17} elevated thresholds for the detection of form from motion,\textsuperscript{18} elevated motion perimetry thresholds,\textsuperscript{19–20} elevated motion coherence thresholds for the directional discrimination of random-dot kinematograms (RDVs),\textsuperscript{21–22} and diminished visual evoked responses to motion stimuli.\textsuperscript{23}

However, although sensitivity to motion is depressed in many patients with established POAG, motion sensitivity tests have so far failed to gain clinical acceptance. To be of value as a clinical tool, a test of M pathway function must reliably distinguish subjects with POAG from normal subjects and must differentiate a percentage of subjects with ocular hypertension from control groups that approximates the proportion of patients with ocular hypertension that later experience glaucomatous visual field loss. No tests to date have fulfilled both criteria. As many as a third of patients with glaucoma, for example, may exhibit normal motion sensitivity,\textsuperscript{14} and as many as 40% of control subjects may show abnormally low sensitivity to moving targets.\textsuperscript{15}

The reason that reliable motion sensitivity losses may not be demonstrated in established glaucoma remains unknown. A number of investigators have assumed that M cells are selectively damaged in glaucoma, but this may not be so. Findings that small P cells may sustain the same degree of damage as M cells\textsuperscript{24–26} and that red–green color perception may be com-
promised in glaucoma suggest that early neural loss in POAG may not be size specific. However, even if M and P cells are lost with equal probability in POAG, a psychophysical test that activates the M pathway preferentially may still provide a sensitive measure of early glaucomatous damage, provided the extent of receptive field overlap, or redundancy, in the M cell population is minimal. There is some evidence that this may be the case: M cells make up only 10% of the total number of ganglion cells, and a recent study suggests that although M and P cells may be lost with equal probability in glaucoma, the mean dendritic field size, and therefore the redundancy, of M ganglion cells is significantly reduced in experimental glaucoma.

Why, then, have tests of motion sensitivity in early glaucoma produced such varied results? It may be that the stimuli and experimental protocols used in most studies so far have not achieved the extent of functional isolation necessary for detecting a deficit in the M pathway alone. There is now sufficient evidence to suggest that motion information is likely to be conveyed by both the M and P pathways in the primate visual system. Nevertheless, the contribution of the P pathway to motion perception has, without exception, been ignored in all previous glaucoma trials. Many studies to date have used small, high-contrast dot and line stimuli with low to moderate drift rates in an attempt to activate the M system selectively in glaucoma. However, it is clear from behavioral measures in humans, and M-lateral geniculate nucleus lesioned monkeys, that the P pathway must play a key role in conveying information about the motion of moderate- to high-spatial-frequency targets. This fact alone dictates that dynamic dot and line stimuli, with their broad Fourier spectra, are unlikely to activate the M pathway in isolation. This conclusion is supported by recent magnetoencephalographic studies in humans, which suggest that neither broad-spectrum stimuli, nor grating stimuli with periodicities exceeding 1 cyc/deg, are likely to activate the M pathway exclusively: it is more reasonable to suppose that such stimuli will invoke the activity of both M and P pathways.

In this study, we determined the effects of POAG on sensitivity to stimuli assumed, on the basis of both human and animal studies, to be conveyed predominantly via the M pathway: namely low-contrast sinusoids of 0.5 cyc/deg, drifting at rates of up to 24 Hz.

Physiological and psychophysical evidence suggests that both M and P pathways contribute to visual sensitivity at low levels of illumination. However, although the P-cell mosaic probably limits scotopic acuity beyond 15° retinal eccentricity, it appears that, in monkeys at least, M cells are the sole conveyors of scotopic information for low spatial frequency (0.5 cyc/deg) gratings drifting at 4 Hz. For this reason, experiments were also conducted under scotopic levels of lighting.

Finally, because motion perceptual deficits have been reported in normal elderly observers, we also examined the effects of advancing age on contrast sensitivity as a possible contributing factor to the overlap between POAG and age-matched control (AMC) group sensitivities observed in many previous studies.

METHODS

Contrast sensitivity (the reciprocal of contrast threshold) was measured for both the detection and direction discrimination of drifting, luminance-modulated sinusoids, and the detection of stationary sinusoids, presented in either the central or peripheral visual field. Measurements were made under photopic and scotopic levels of illumination. For each participant, all measures were completed in two or three sessions, each lasting approximately 1 hour.

All studies followed the tenets of the Declaration of Helsinki and had Ethics Committee approval. Consent was obtained from each participant after the procedures were explained.

Stimuli

Stimuli were generated using a waveform generator (VSG2/1; Cambridge Research Systems, Rochester, UK) with 14-bit digital-analog converters and displayed on a γ-corrected 14-in. color monitor (Flexscan 9080i; Eizo). Stimuli were presented at a noninterlaced frame rate of 70 Hz and resolution 624 pixels by 878 lines. The display screen subtended 8.5° horizontally by 6.3° vertically at a viewing distance of 2 m.

The stimulus was a horizontal, achromatic sinusoidal grating described by:

\[
L(x,t) = L_m + \sin[2\pi(f x + g t)]E(t)
\]

where \(E(t) = 1 + \cos(2\pi T)\), \(L_m\) is the mean luminance (25 cd/m²), \(A\) is the amplitude of the grating, \(f\) is the spatial frequency (0.5 cyc/deg), \(g\) is the drift temporal frequency (0–24 Hz), and \(T\) is the presentation time (500 ms). The luminance contrast \((C)\) of the grating was defined as Michelson contrast \([L_{\text{max}} - L_{\text{med}}]/(L_{\text{max}} + L_{\text{med}})\). The γ-corrected display was linear to 95% contrast, a value that was not exceeded.

Stimuli were presented within a square patch of 4.7° height, the sharp edges of which were attenuated using a cosine ramp of 0.75° width. The screen luminance surrounding the stimulus zone was matched to the mean luminance of the stimulus.

Observers

Fifteen patients with POAG (mean age, 58.7 ± 11.9 years), 14 age-matched control (AMC) subjects (mean age, 55.8 ± 10.5 years), and 10 young control (YC) subjects (mean age, 24.4 ± 2.1 years) participated in the study. Not all observers participated in both the photopic and scotopic experiments. All were naïve about the purposes of the experiment.

Observers with POAG were recruited from the Optometry Clinic, Department of Vision Sciences at Aston University, and the Glaucoma Department at the Birmingham and Midlands Eye Hospital. All had been known to have POAG for a minimum of 1 year before testing and were treated with timolol maleate eye drops, a nonselective β-adrenoceptor antagonist that does not affect pupil size. All patients manifested an optic nerve head appearance consistent with POAG (including increase in cup size, increase in cup–disc ratio, disc asymmetry, changes in the lamina cribrosa, loss of neuroretinal rim, pallor, evidence of papillary atrophy, vessel changes, or disc margin hemorrhage). The subjects were further selected on the basis of near-normal visual fields, because any bias toward large cell loss in POAG is thought to disappear in the later stages of the disease. Visual fields had been assessed by threshold perimetry (Henson Central Field Analyzer [London, UK] or Humphrey Field Analyzer [San Leandro, CA]) and for all sub-
Contrast sensitivity was measured using binary-choice procedures in conjunction with a three-up, one-down staircase converging to a performance level of 79% correct. Each trial consisted of a single-interval presentation accompanied by an audible tone. For the detection criterion, either a blank field or the gratings stimulus was presented at random, and observers were required to judge whether the gratings was present or absent. For the direction discrimination criterion, the stimulus was presented on each trial, and observers were required to judge its direction of motion, randomly chosen to be up or down. Responses were communicated to the computer through a three-button computer mouse. AW, who was positioned out of sight of the display screen, controlled the mouse on the observer’s behalf. No feedback was given.

Before the staircase, contrast was adjusted from a suprathreshold value to near threshold using method of adjustment. The method of adjustment threshold value was used as the initial contrast of the gratings for the staircase procedure. The termination of the method of adjustment routine and the initiation of the staircase was controlled using the computer mouse. The mean of four staircase reversals was taken as a measure of contrast sensitivity. Each datum is the mean of three staircase runs.

### Photopic Sensitivity

Fifteen observers with POAG (mean age, 58.7 ± 11.9 years [SD]), 12 AMC subjects (mean age, 56.2 ± 11.4 years), and 10 YC subjects (mean age, 24.4 ± 2.1 years) participated in the study. The room was dimly lit by a single incandescent globe directed toward the ceiling. The mean luminance of the screen was 25 cd/m². The display was viewed monocularly using natural pupils and accommodation. Contrast sensitivity was measured for test stimuli of 0.5 cycled/deg, either stationary or drifting at 16 or 24 Hz.

### Scotopic Sensitivity

Twelve observers with POAG (mean age, 58.8 ± 10.9 years), 10 AMC subjects (mean age, 56.0 ± 12.2 years), and 7 YC subjects (mean age, 24.4 ± 1.9 years) participated in the study. Experiments were performed in a darkened room after 15 minutes’ dark adaptation. Retinal illuminance was held constant at 0.6 trolands (td) by the use of a 4-mm artificial pupil positioned in the spectacle plane and neutral density filters affixed to the display screen. The illuminance of the red LED fixation target for peripheral experiments was reduced by the use of a 1.3-log unit neutral-density filter positioned over the light source. Contrast sensitivity was measured for test stimuli of 0.5 cycled/deg, either stationary or drifting at 4 Hz.

### Results

Mean contrast sensitivity for each stimulus condition is shown for the POAG, AMC, and YC groups in Table 1. Unless otherwise stated, one-way analyses of variance (unrelated) were used to estimate statistical significance that meant sensitivity measures were different among the three experimental groups.

### Photopic Sensitivity

**Central Vision.** Figure 1 shows foveal contrast sensitivity for the detection of stationary (a) and drifting (b, c) gratings...
and for the direction discrimination of drifting gratings (d, e). Each symbol represents the contrast sensitivity of one observer, classified as YC, AMC, or POAG. Note that in all three groups, and for both drift temporal frequencies, thresholds for direction discrimination approximated those for detection.

Sensitivity for the detection of stationary gratings was not significantly different between groups. However, significant group differences in mean contrast sensitivity were evident for both the detection and direction discrimination of gratings drifting at 16 and 24 Hz ($P < 0.001$). Post hoc analyses confirmed that the YC group was significantly more sensitive to moving targets than both the POAG and AMC groups ($P < 0.05$; Tukey’s honestly significant difference [HSD]). However, there was no significant difference in mean contrast sensitivity between POAG and AMC subjects for either stimulus drift rate.

To characterize the decline in motion sensitivity with normal aging, contrast sensitivity for the detection of 16-Hz drifting gratings was replotted as a function of increasing age for young (filled symbols) and age-matched (open symbols) control observers and subjected to multiple regression analyses (Fig. 2). The age-related decline in sensitivity for motion stimuli was best described by a linear function (slope $= -1.5$) and was significant ($P < 0.01$).

It is possible that reduced retinal illumination, arising as a result of increased lenticular absorption and/or decreased pupil size associated with aging, may affect contrast sensitivity for moving targets selectively. To assess whether decreased retinal illumination alone could account for the motion-specific sensitivity losses observed in the aged groups, contrast sensitivity for the detection of stationary and drifting gratings presented in central vision was measured for two experienced psychophysical observers (the authors) under three levels of retinal illumination. Retinal illumination was manipulated by varying artificial pupil size. Pupils were dilated to 9-mm diameter using 0.5% Tropicamide, a muscarinic antagonist causing mydriasis with minimal cycloplegia. Artificial pupils of 4 mm (314 td), 6 mm (707 td), and 8 mm (1257 td) were cut from...
black card and positioned in the spectacle plane using a trial frame. To ensure that the centers of the artificial and natural pupils were in alignment, each observer varied the position of the artificial pupil until the perceived contrast of a stationary sine-wave grating of 1 cyc/deg and 90% luminance contrast was maximized.

Figure 3 shows, for both observers, contrast sensitivity for the detection of test stimuli as a function of increasing pupil diameter. Results are shown for drift temporal frequencies of 0 Hz (circles), 16 Hz (squares), and 24 Hz (triangles). Over the range of values tested, sensitivity for stationary and drifting targets was not significantly affected by varying pupil diameter. It is unlikely, therefore, that the age-related decline in motion sensitivity observed in this study could be accounted for solely by reductions in retinal illumination associated with the processes of normal aging.

To determine whether contrast sensitivity declined with age within the older groups, POAG and AMC data were replotted as a function of increasing age and subjected to multiple linear regression analyses (Fig. 4). When data from both groups were combined (solid lines), multiple regression analyses revealed significant declines in sensitivity with age for the detection of stationary gratings ($P < 0.05$), and for both the detection and direction discrimination of drifting gratings ($P < 0.001$). The slope of the function for the combined AMC and POAG data sets, and its significance, is indicated on each panel.

Assessed separately, the slope of the linear function for the POAG group (dotted lines) was greater than that of the AMC group (dashed lines) for all stimulus conditions. Note that sensitivity for both the detection of stationary gratings and the direction discrimination of drifting gratings declined significantly with age in the POAG but not AMC group. However, the rate of decline in sensitivity with age was not significantly different between the POAG and AMC groups for any condition of the test stimulus ($P > 0.05$; analysis of covariance).

**Peripheral Vision.** Figure 5 shows contrast sensitivity for the detection of stationary (a) and drifting (b, c) gratings and the direction discrimination of drifting gratings (d, e) presented at 20° in the nasal visual field. As with central vision, mean sensitivity for drifting stimuli was significantly lower in the POAG and AMC groups compared with YC subjects for drifting ($P < 0.05$; Tukey’s HSD), but not stationary stimuli. Unlike central vision, several subjects with POAG demonstrated contrast sensitivities that fell below that of the lowest measure recorded in the AMC group. However, differences in mean sensitivity between the POAG and AMC groups failed to reach statistical significance for either drift temporal frequency of the test ($P > 0.05$; Tukey’s HSD).

YC and AMC data for the detection of 16-Hz drifting gratings presented at 20° eccentricity were replotted as a function of age to determine the nature of the decline in peripheral motion sensitivity associated with normal aging (Fig. 6). The age-related decline in sensitivity was best described by a linear function (slope = −0.8) and was significant ($P < 0.01$).

Figure 7 shows the POAG and AMC data shown in Figure 5 replotted as a function of increasing age. Linear regression analysis to the combined AMC and POAG data sets (solid lines) showed a significant age-related decline in sensitivity for drifting ($P < 0.05$), but not stationary, gratings (slope and statistical significance are shown on each panel). As for central vision, the rate of decline in the POAG group was greater than that of the AMC group for both drift temporal frequencies of the test (compare dotted and dashed lines, respectively, in panels b through e). Further, the rate of decline in sensitivity with age was not significant for the AMC group for temporal frequencies below 24 Hz. However, differences in the rate of decline with aging between the two groups failed to reach statistical significance for any stimulus condition ($P > 0.05$; analysis of covariance).

**Scotopic Sensitivity**

**Central Vision.** One-way analyses of variance (unrelated) showed that mean contrast sensitivity was significantly different between groups for the detection of both stationary and drifting gratings presented in the central visual field ($P < 0.05$). Post hoc analyses showed that YC subjects performed significantly better, on average, than AMCs or subjects with POAG ($P < 0.05$; Tukey’s HSD), whereas the mean sensitivities of the AMC and POAG groups were not significantly different. Between-group differences in contrast sensitivity for the directional discrimination of 4-Hz drifting targets were not significant.

Figure 8 shows scotopic contrast sensitivity for the detection of stationary gratings (a) and for both the detection (b) and direction discrimination (c) of 4-Hz drifting gratings presented in the central visual field. Each datum in the left panels (a, b, and c) represents the mean sensitivity of one observer, classified as YC, AMC, or POAG. The right panels (d, e, and f) show...
the contrast sensitivity data depicted in a, b, and c replotted as a function of age for the AMC (open symbols) and POAG (filled symbols) groups. When data from both groups were combined (solid lines), linear regression analyses showed no significant decline in contrast sensitivity with age for either stationary or drifting gratings. When assessed separately, neither the AMC (dashed lines) nor the POAG (dotted lines) group showed a significant age-related decline in contrast sensitivity for any condition of the test. The slopes of the functions were not significantly different between groups (P > 0.05; analysis of covariance).

**Peripheral Vision.** For all conditions of the test stimulus, mean contrast sensitivity for the YC group was significantly greater than that for both the AMC and POAG groups (P < 0.05; Tukey’s HSD). Again, however, there was no significant difference in mean sensitivity between the AMC and POAG groups for any condition used.

Figure 9 shows the contrast sensitivity of individual subjects for the detection of stationary gratings (a) and for both the detection (b) and directional discrimination (c) of drifting targets, plotted as a function of increasing age. The right panels (d, e, and f) show these data replotted as a function of age for the AMC (open symbols) and POAG (filled symbols) groups. Linear regression analyses of the combined AMC and POAG data sets (solid lines) showed a significant, age-related decline in sensitivity for drifting (P < 0.05), but not stationary, targets. When assessed separately, the AMC group (dashed lines) showed a small, but significant decline in contrast sensitivity with age for both stationary and drifting targets (P < 0.05), whereas the age-related decline in sensitivity within the POAG group (dotted lines) was only significant for the detection of 4-Hz drifting gratings. However, the rate of decline in sensitivity with age in the POAG group was never significantly different from that of the AMC group (P > 0.05; analysis of covariance).

**DISCUSSION**

In this article, we used protocols designed to activate the M pathway preferentially, namely motion stimuli in conjunction with a direction-discrimination criterion, to assess visual sensitivity in observers with POAG and normal observers. Our results show that, under photopic conditions, sensitivity to moving targets in both central and peripheral vision declined significantly (P < 0.01) with increasing age, whereas sensitivity to stationary targets did not (Figs. 4 and 7). Under scotopic light levels, a significant decline in sensitivity was only evident for peripheral motion targets (Fig. 9). Control studies show that these motion-specific sensitivity losses cannot be attributed solely to a decrease in retinal illuminance associated with aging, lending support to previous suggestions that motion sensitivity deficits have a neural, rather than optical, source.45
Under photopic conditions, the rate of decline in sensitivity to visual motion was consistently greater in the POAG group than in the AMC group (Figs. 4 and 7). However, this difference did not reach statistical significance and is unlikely to be a useful clinical measure of glaucomatous damage. Indeed, despite using a wide range of experimental parameters, we were unable to demonstrate any significant motion-specific sensitivity deficits in glaucoma patients other than those that could be attributed to the normal processes of aging. Our results belie many reports suggesting that motion sensitivity is selectively depressed in glaucoma, and suggest that sensitivity to motion targets per se may not be a useful indicator of neural integrity in glaucoma.

We argued in the introduction that high velocity, low-spatial-frequency targets of the type used in this study are more likely to activate the M pathway in isolation than are broad-spectrum stimuli, such as RDKs and lines. Nevertheless, some of the largest visual deficits in POAG have been reported for broad-spectrum stimuli in conjunction with tasks of motion coherence (using RDKs) and motion displacement (using line targets). Given that the P pathway must play a significant role in processing motion stimuli of high spatial frequency, the differences between our findings and those of previous studies are unlikely to reflect the superiority of dot or line stimuli in isolating M pathway function.

It is equally unlikely that a global motion task, such as motion coherence, is better able than the local motion task used in this article to bias the response of the visual system to the M pathway. Although there is evidence to suggest that cortical area MT, known to rely almost exclusively on input from M cells through area V1, plays a role in the percept of global motion, more recent work shows that motion coherence thresholds may be only marginally elevated after lesions of MT in monkeys. As a result, global motion percep-
tion may not be mediated exclusively by MT via magnocellular neurons.

Why is it that global motion tasks (using RDKs) and motion displacement thresholds (using line targets) seem better able to differentiate glaucoma-affected subjects from normal control subjects than local motion tasks (using drifting sinusoids)? Visual sensitivity within a region of glaucomatous field upset is not necessarily uniform; there may be highly localized pockets of depressed sensitivity dispersed among regions of higher sensitivity. Motion tasks of the type described above (i.e. global motion/motion displacement) necessarily demand the integration of local motion signals over space and time. If it is the case that sensitivity within a given glaucomatous scotoma is nonuniform, it may be more difficult to perceive motion with spatially nonredundant patterns such as dots and lines than to perceive local motion with spatially redundant patterns such as drifting sinusoids. A similar argument has recently been advanced by Joffe et al.\(^{22}\) in an attempt to explain the variability evident in previous glaucoma trials. They suggest that glaucoma causes a reduction in the normal integrative visual function necessary for the perception of global motion in textured (RDK) displays.

Psychophysical studies that have successfully used dot and line stimuli in conjunction with motion tasks to differentiate patients with glaucoma from normal subjects have often been cited as evidence in support of the hypothesis that M cells are selectively damaged in glaucoma. But the argument outlined above for the apparent superiority of these stimuli over sine-wave gratings is not dependent on one pathway being more affected by glaucoma than another pathway. What seems to be important in the psychophysical identification of any neuropathy, at least for dot and line stimuli, is whether the observer’s task demands an integration of visual signals over space. If this is the case, we submit that the interpretation of many previous psychophysical trials using broadband motion stimuli should be re-evaluated. In brief, poor motion sensitivity measures obtained using dot and line stimuli cannot be taken as evidence for selective damage within the M pathway in POAG.

Contrary to our findings, however, one class of narrow-band stimuli has proved to be useful in discriminating between glaucoma patients and AMC subjects; namely, frequency-doubled patterns.\(^{16,17}\) These are rapidly counterphased (\(>15\) Hz) sine-wave gratings: the term frequency-doubled (FD) refers to the fact that their perceived spatial periodicity is twice their actual periodicity. FD patterns are similar to the sine-wave gratings used in this study, in that they are coarse (\(\leq 0.5\) cyc/deg), large field (\(>2\) cycles) motion targets. The discrepant results between FD studies and our own are unlikely to reflect methodologic differences in stimulus position or size: effective screening for glaucomatous damage has been reported using quadrant, hemifield, central field (5–17.5° radius), and small, localized FD targets.\(^{16,17}\) Rather, the discrepant findings may reflect the type of cells activated by the different stimulus types: our stimuli were designed to activate the entire popula-

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**Figure 7.** Effect of aging within AMC and POAG groups: photopic, peripheral viewing. Contrast sensitivity data from Figure 5 replotted as a function of increasing age for AMC (○) and POAG (●) groups. Each line in (a) through (e) is the fit of a linear function to either the AMC group (dashed line), the POAG group (dotted line), or the combined AMC and POAG data sets (solid line). The slope of the function to the combined AMC and POAG data set is given in each panel. *\(P < 0.05\); **\(P < 0.01\).
tion of M cells, whereas FD patterns are believed to activate nonlinear M cells (Mγ cells), which comprise only 15% to 25% of the M-cell population. Because the M pathway constitutes only 10% of the total number of optic nerve fibers, we would expect to find only 1.5% to 2.5% Mγ fibers. Therefore, the apparent success of FD patterns over other stimulus types may have more to do with the fact that Mγ cells form a sparse sampling array than it has to do with the selective attrition of large cell fibers in glaucoma (see introduction; see also Johnson and Samuels17).

The debate over whether M cells are selectively damaged in the early stages of glaucoma continues. Regardless of the outcome of this debate, if it is the case that M cells form a minimally redundant sampling array, psychophysical tests of M pathway function may still provide some of the most sensitive measures of glaucomatous damage. The standard means of biasing responses from the M system is to use motion stimuli of one kind or another, as we have done in this study. But evidence is accumulating to suggest that performance measures involving visual attention and object localization may also be useful. The properties of single M cells, and single units in M-dominated regions of visual cortex, make the M pathway suited to less conscious visual functions, such as redirecting gaze to peripherally detected targets in a visual scene.34,56 M cells are proportionally more abundant in the peripheral retina31 and may transmit visual information more rapidly than P cells by virtue of their superior contrast sensitivity and shorter conduction velocity.56 In addition, the parietal cortex, known to receive most of its input from retinal M cells,7 has been strongly implicated in redirecting visual attention,57 object localization,58 and the control of pursuit eye movements.59 A psychophysical test designed along these lines would have significant practical importance for the development of procedures useful for the detection of glaucoma in its earliest stages.

CONCLUSIONS

Neural deficits specific for motion perception occur during normal aging, and this must be taken into account when designing psychophysical tests for the detection of glaucomatous changes. Behavioral measures of sensitivity to motion targets may not provide a useful means of indicating neural integrity in glaucoma unless used in conjunction with a task that demands the integration of visual signals across space. Whether M cells are selectively damaged in glaucoma or not, we have argued that it is still worthwhile pursuing psychophysical measures of M pathway function. For this purpose, it may be advantageous to combine measures of object localization with a test demanding the integration of signals across space and time.
References


