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ORIGINAL ARTICLE

Defining the Nature of Motion Perception Deficits in Glaucoma Using Simple and Complex Motion Stimuli

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ABSTRACT

Purpose. The purpose of this study is to determine the nature of motion perception deficits in primary open-angle glaucoma by measuring the sensitivity of simple (luminance-defined) and complex (texture-defined) motion, the latter requiring supplementary neural processing to be resolved. These findings will help address the possible extent of the cortical damage in glaucoma that has been recently demonstrated by anatomic and physiological studies. They also serve the purpose of establishing which motion paradigms would be most appropriate for assessing glaucoma-related functional loss.

Methods. Direction-identification thresholds for first-order and second-order motion were measured for 26 patients with primary open-angle glaucoma (for both phakic and pseudophakic) and 18 nonglaucomatous observers.

Results. The glaucomatous observers showed significantly increased motion thresholds for both first- and second-order motion conditions when compared with nonglaucomatous observers. However, the relative increase in threshold for first-order motion did not differ significantly from that of second-order motion.

Conclusions. These findings imply that there is no measurable higher-level cortical function damage caused by the glaucomatous process because no greater loss in second-order motion was observed. Based on the results, we suggest that motion paradigms used to assess functional loss in primary open-angle glaucoma should consist of simple, first-order type stimuli to minimize potential confounds such as those introduced by both the normal and pathologic aging process on complex motion processing (i.e., perimetry using complex motion stimuli).
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Key Words: cortical damage, glaucoma, motion, first-order, second-order, motion perimetry

Recent studies have demonstrated that glaucoma may cause changes in the Lateral Geniculate Nucleus and cortical regions of the brain as demonstrated by cell shrinkage and loss.[1,2] There is also evidence from metabolic and neurochemical studies that the central nervous system is affected in glaucoma.[3–5] Given these findings, it becomes important to determine whether both low- and higher-level perceptual losses are induced by glaucoma-related damage.

A number of psychophysical studies have focused on the development of early diagnostic methods that assess visual field loss in open-angle glaucoma.[6] Such defects associated with glaucoma are presumed to originate from ganglion cell destruction in the retina.[7–9] Specifically, such studies indicate that M-ganglion cells, which represent approximately 8% to 10% of the retinal ganglion cells in the retina,[10] are lost preferentially during the glaucomatous process, particularly in the early stages of the disease[7,8] (see also other references for an alternate view[11–13]). An attenuation of both the pattern electroretinogram[14] and low-contrast visual-evoked potentials,[15] both of which have been argued to be stimuli that bias the magnocellular pathway, have been shown in glaucoma and attributed to M-cell loss. Decreased diffuse flicker sensitivity[16,17] and temporal modulation visual fields[18–20] lowered peripheral displacement thresholds,[21] and defects in spatiotemporal contrast sensitivity[22] in glaucomatous eyes are further indicators of magnocellular pathway degeneration. Because M-ganglion cells are believed to be involved in the processing of temporal information and

to be the retinal origin regarding motion information processing,[23,24] the evaluation of motion perception in glaucoma has been used as an investigative predictor of the disease. In effect, several studies have associated motion perception impairments with advancing glaucoma. Direction-discrimination thresholds for global motion cues have been shown to be elevated in patients with primary open-angle glaucoma (POAG) and for persons with ocular hypertension (OHT),[25] and motion perimetry has revealed glaucomatous defects that were not observable using conventional perimetry.[26,27] Loss of sensitivity to motion-defined form (patients with POAG and those with OHTs[28]), elevation of perimetric motion thresholds (patients with POAG[29]), and loss in motion coherence perimetry[26] have all been demonstrated. These and other findings suggest motion perception is impaired in the early stage of the disease (see [30] for review).

Although different motion testing paradigms show glaucoma-related loss, they do not solicit identical processing requirements. For instance, global motion (or motion coherence) necessarily requires complex spatiotemporal integration within extrastriate cortical areas,[31,32] whereas simple stimuli (luminance-defined or even flicker thresholds) solicit higher-level motion processing to a much lesser extent. If we are to assume that glaucoma is primarily an optic nerve disease, then why use complex motion stimuli to assess retinal–neural function? Using motion paradigms that necessitate complex perceptual processing may reduce the diagnostic specificity of the measures because performance on such tests may be contaminated by higher-level neural changes such as those presented during normal aging[33] in addition to glaucoma-related retinal dysfunction. Complex motion processing has been shown to be affected during the normal[34] and pathologic aging process[35–39] to a greater extent than for simple motion types.[34,38,39] On the other hand, using complex motion paradigms might be of interest if we assume that there is higher-level cortical damage in glaucoma. The recent evidence showing that cortical cells primarily associated with the nonglaucomatous eye can also show anatomic changes implies that higher-order mechanisms may also be affected in this disease.[2] If this were the case, using complex motion paradigms as opposed to simpler techniques might increase the sensitivity of the measure. Given the demographic of most patients with glaucoma, the selection of proper motion stimuli has important practical and interpretive implications.

The main purpose of this study was to determine whether motion perception deficits in POAG are the consequence of only a low-level retinal V1 system or includes additional higher-level cortical processing; in essence, to investigate the nature of motion perception deficits found in POAG. This was done by measuring direction-identification thresholds to first-order (or luminance-defined) and second-order (texture-defined) motion stimuli. These stimuli were chosen because contemporary motion models differentiate first- and second-order motion processing by the level at which they are first processed along the motion pathway.[40–44] Luminance-defined first-order motion processing begins in the retina and is followed by standard motion analysis selective mechanisms operating in the primary visual cortex that process the local luminance variations in the retinal image.[45–48] For this reason, first-order information is considered to be a "simple" type of visual information. On the other hand, additional nonlinear processing of the second-order motion signals is required (i.e., signal rectification or response squaring) before standard motion analysis can resolve the direction of this class of motion.[41,44] Such additional processing has been attributed to the cortex,[49–52] suggesting that second-order motion perception necessarily involves more cortical processing. For this reason, second-order visual information is considered a more "complex" type of visual information because it requires the implication of larger neural circuitry to be perceived. These two classes of visual information were used because a large body of psychophysical evidence has demonstrated that they are initially processed by neural mechanisms varying in complexity[40,41,44,53] and are sensitive to subtle neural dysfunctioning of visual information processing.[33,34]

The techniques used in this article have been shown to be extremely sensitive to subtle cortical alterations. For example, normal aging has been shown to affect second-order thresholds to a significantly greater extent than first-order thresholds, and this, in nonpathologic (healthy) aging where changes, if they exist, must be much more subtle than disease processes.[34] This paradigm has also been very useful to demonstrate subtle perceptual effects in autism[54,55] and fragile X syndrome.[56] Therefore, this method has been proven to be very effective to tease out whether there are condition-specific subtle changes in cortical processes requiring larger neural networks.[33]

A decrease in the perception of both first- and second-order drifting stimuli is expected in POAG given the primary mechanism for glaucoma, that being retinal ganglion cell loss. If the glaucomatous process governing open-angle glaucoma is in any way related to higher neural dysfunctions, then damage to higher cortical areas is expected, leading to a larger threshold elevation for the perception of second order. Alternatively, if there is no relative difference between the magnitude of loss for first- and second-order stimuli, we must conclude that the glaucoma-related neural damage remains at a low level and that future assessment of glaucoma-related motion sensitivity loss should use testing paradigms that limit cortical processing (simple motion).

METHODS

Observers

A total of 44 observers participated in this study (mean age, 70.5 ± 5.37 years; range, 61–78 years), all of which were recruited from among the patients of the Ophthalmology Department of the Sir Mortimer B. Davis Montreal Jewish General Hospital. Observers were considered to have glaucoma after a diagnosis or confirmation of diagnosis by the examining ophthalmologist at the patient's most recent visit. All observers with glaucoma were undergoing treatment. The observers were placed into one of four experimental groups: POAG/phakic (n = 15; mean age, 70.20 ± 5.85 years), POAG/pseudophakic (n = 11; mean age, 71.09 ± 6.09 years), nonglaucomatous/phakic (n = 10; mean age, 70.00 ± 4.29 years), or nonglaucomatous/pseudophakic (n = 8; mean age, 71.25 ± 4.86 years). Both phakic and pseudophakic observers with and without glaucoma were tested to evaluate whether lenticular senescence would contribute to loss in motion sensitivity irrespective of glaucoma. Control participants were defined as having no ocular pathologies and taking no long-term ocular medication. Mild sclerosis of the lens was permitted as long as the subject met the minimal visual acuity requirements. Minimal corrected visual acu-

ity was 20/40 and all of the observers had normal color vision as measured by Farnsworth D-15 color plates. The pupil sizes of all observers were measured. Testing was done monocularly with the observers' best correction in place. All participants were naïve to the purpose of the study and none were experienced psychophysical observers. In accordance with the tenets of the Declaration of Helsinki, informed consent was obtained from each participant before testing began.

Apparatus and Display

The motion stimuli were presented using a Power Macintosh 7300/200 computer and presented on a standard Seiko Instruments CM 1445 screen (refresh rate of 67 Hz, GEAC Canada Ltd, Ontario, Canada) that was gamma-corrected using a color look-up table. The screen resolution was 640 × 480 pixels and its mean luminance was 38 cd/m^2 in which L_{min} and L_{max} were 3.6 cd/m^2 and 80 cd/m^2, respectively. Stimuli generation and presentation were controlled using the VPixx graphics program (www.vpixx.com). Luminance and chromaticity measurements were made using a Minolta Chromameter (CS-100, Folio Instruments Inc., Ontario, Canada).

Stimuli

All motion stimuli were presented to observers within a circular region at the center of the display subtending a visual angle of 10° in diameter when viewed from a distance of 67 cm. The stimuli consisted of first- and second-order translating patterns. The first-order motion stimuli were luminance-modulated noise patterns produced by adding static grey-scale noise to modulating sine waves, in this case a vertical sinusoid. The noise consisted of dots (1 pixel × 1 pixel measuring approximately 2.3 arc min) with individual luminances randomly assigned as a function of sin (x) in which (x) ranged from 0 to 2Π. The contrast (luminance modulation depth) of the first-order patterns was manipulated by varying the amplitude of the modulating sine wave. The amplitude of the luminance modulation for the first-order patterns could be varied from 0.0 to 0.5 defined as:

$$\text{luminance modulation depth} = (L_{max} - L_{min})/(L_{max} + L_{min})$$

where L_{max} and L_{min} refer to the average highest and lowest local luminances in the pattern. The first-order luminance modulation levels used in the constant stimuli presentations were 0.04, 0.02, 0.01, 0.005, 0.0025, and 0.00,125. These levels were chosen based on previous studies using similar stimuli.[54]

The second-order stimuli were texture-modulated noise patterns produced by multiplying rather than summing the same modulating sine waves to the grey-scale noise. The depth of the texture modulation (contrast modulation depth) was manipulated by varying the amplitude of the modulating sine wave. The amplitude of the sinusoid therefore defined the contrast of the pattern and could be varied within a range of 0.0 and 1.0 defined as:

$$\text{contrast modulation depth} = (C_{max} - C_{min})/(C_{max} + C_{min})$$

where C_{max} and C_{min} are the maximum and minimum local contrasts in the pattern (Fig. 1). Second-order contrast modulation levels used during the constant stimuli procedures were 1.0, 0.333, 0.143, 0.111, and 0.059. The spatial and drift frequency was 1 cycle per degree (cpd) and 2 Hz, respectively.

Procedure

Participants were tested individually in a dimly lit room and viewed the monitor monocularly using the eye with best visual acuity. The procedure was explained to them and a practice session followed to familiarize the participants with the procedure and confirm that they were able to complete the task. During the actual testing session, each participant was presented with trials consisting of first- and second-order stimuli moving either to the left or to the right for 750 ms. The method of constant stimuli was used to measure direction-identification thresholds for each motion condition that included six levels of luminance modulation (ranging from 0.04 to 0.00,125) and five levels of contrast modulation (ranging from 0.5 to 0.03,125). For each motion condition, the stimuli were presented 10 times in either direction (left and right) at each level of modulation for a total of 20 trials at each level of modulation. Participants were asked to identify the direction of motion by pressing either of two buttons on a keypad (two alternative forced choices). All stimuli were presented foveally at the same spatial location on the monitor. Participants were instructed to keep their gaze on a central fixation point throughout experimentation. Finally, Weibull[57] functions were fitted to the data to calculate direction-identification thresholds at 75% correct level of performance.

RESULTS

Because first- and second-order absolute motion thresholds are defined by different attributes (i.e., luminance and texture modulation, respectively), comparisons of the mean differences between the two motion classes is not informative nor is the direct comparison of first- and second-order thresholds for the same patient. For this reason, first- and second-order motion-identification thresholds were analyzed separately.

Effect of Lenticular Senescence on Motion Sensitivity

As already mentioned, both phakic and pseudophakic participants with and without glaucoma were evaluated to determine

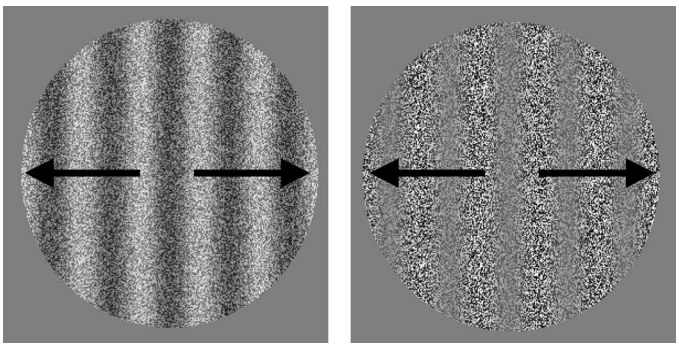

FIGURE 1.
First- (left panel) and second-order (right panel) motion stimuli used in the present study.

whether lenticular senescence would contribute to loss in motion sensitivity independently of the presence of glaucoma. Figure 2A shows the mean direction-identification thresholds for phakic (white bars) and pseudophakic (black bars) observers with and without glaucoma for the first-order motion condition. A two-way analysis of variance (ANOVA) (group × lenticular senescence) revealed no significant difference in first-order direction-identification thresholds between phakic and pseudophakic observers in either the glaucoma ($F_{1,24} = 0.428$, $p > 0.05$) or the control group ($F_{1,16} = 0.454$, $p > 0.05$). A separate two-way ANOVA was used to compare the direction-identification thresholds between phakic and pseudophakic observers for the second-order motion condition. As was found for the first-order motion type, phakic and pseudophakic observers showed no significant difference in direction-identification thresholds for either glaucomatous ($F_{1,24} = 0.432$, $p > 0.05$) or control participants ($F_{1,16} = 0.834$, $p > 0.05$) for the second-order motion condition (Fig. 2B). Because the direction-identification thresholds were not affected by lenticular senescence for either glaucomatous and control observers for both motion conditions, the thresholds were collapsed across lens condition and averaged. These results demonstrate that, unlike minimum motion color thresholds,[58,59] lenticular senescence does not affect motion sensitivity in our conditions for either luminance- or texture-defined motion information for observers with and without glaucoma.

Effect of Glaucoma on First- and Second-Order Motion Sensitivity

After the data were collapsed across lens condition, 26 observers fell into the glaucoma category (mean age, 70.56 ± 5.85 years; range, 61–78 years) and 18 nonglaucomatous observers served as age-matched control subjects (mean age, 70.56 ± 4.46 years; range, 63–78 years). Figure 3 illustrates the direction-identification thresholds for first-order (left panel) and second-order (right panel) motion conditions for both glaucomatous and control participants (phakic and pseudophakic patients included in each experimental group). Statistical analysis of the grouped data showed that direction-identification thresholds were significantly higher for the observers with glaucoma when compared with normal observers for both the first ($F_{1,42} = 19.114$, $p = < 0.05$) and second-order ($F_{1,42} = 12.787$, $p < 0.05$) motion conditions. More importantly, the magnitude of the threshold elevation was similar for both motion conditions; 1.56 times greater for the first-order class (from 0.005 to 0.0078) and 1.40 times greater for the second-order class (from 0.1787 to 0.2506).

Effect of Retinal Illumination on First- and Second-Order Motion Sensitivity

Optical factors such as reduced retinal illumination caused by decreased pupil size contribute to age-related decline in contrast sensitivity.[60] To ensure that differences found in motion sensitivity were not a consequence of reduced retinal illumination, the pupil size of each observer comprising the different experimental groups was measured and compared. On average, each experimental group had comparable mean pupil sizes: POAG/phakic group (n = 15; mean pupil size, 3.20 ± 0.68 mm), POAG/pseudophakic group (n = 11; mean pupil size, 3.00 ± 0.63 mm), nonglaucomatous/phakic group (n = 10; mean pupil size, 3.30 ± 0.67 mm),

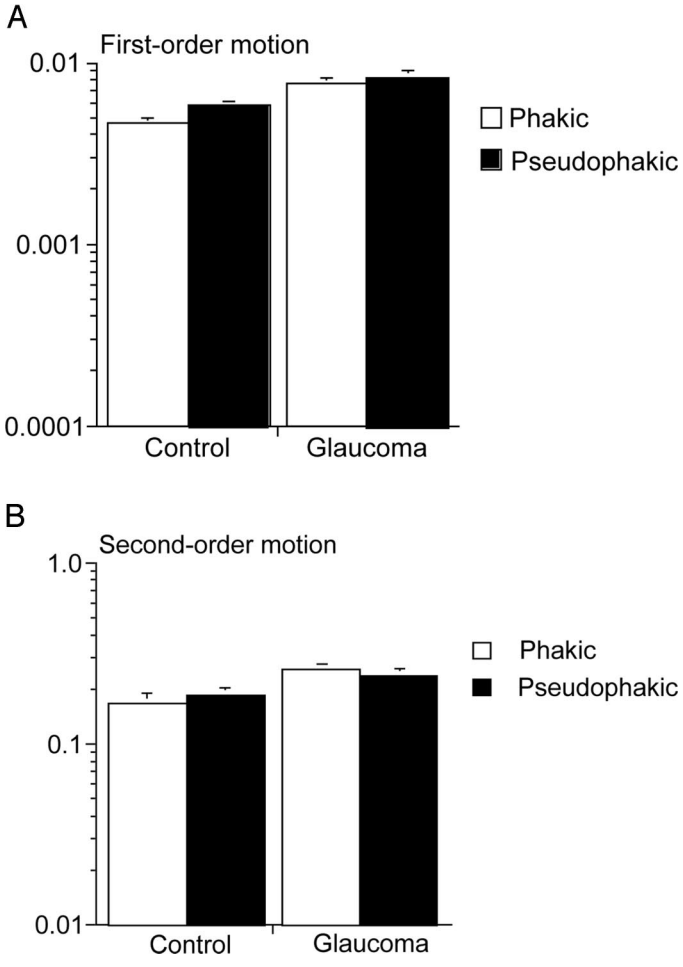

FIGURE 2.

Mean first-order (A) and second-order (B) direction-identification thresholds for phakic (n = 25, 15 glaucomatous and 10 nonglaucomatous) and pseudophakic (n = 19, 11 glaucomatous and 8 nonglaucomatous) participants for both the control and glaucoma conditions.

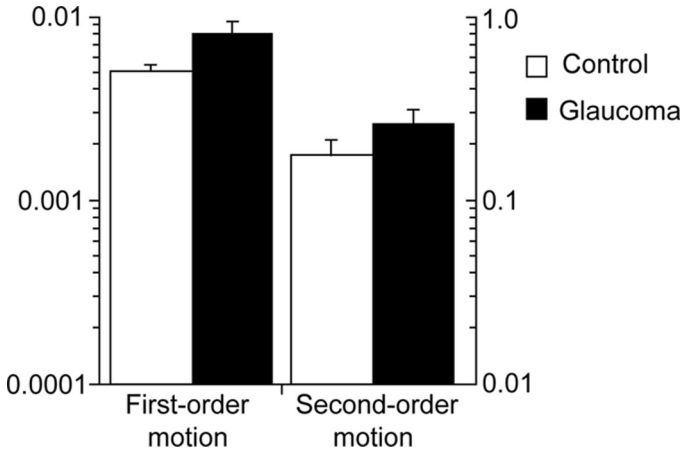

FIGURE 3.

Mean first-order and second-order motion direction-identification thresholds for control and glaucomatous observers. The first-order scale is shown on the left of the figure and the second-order scale is shown on the right.

and nonglaucomatous/pseudophakic group (n = 8; mean pupil size, 3.00 ± 0.53 mm). Additional statistical analysis demonstrated that pupil size (4 mm, n = 12; 3 mm, n = 26; and 2 mm, n = 6) did not significantly affect direction-identification thresholds for either the first-order ($F_{2,41} = 0.971$, $p > 0.05$) or second-order ($F_{2,41} = 1.824$, $p > 0.05$) motion, irrespective to which group the patient belonged to.

DISCUSSION

Simple and Complex Motion Perception in Glaucoma

The purpose of this study was to investigate the nature of motion perception impairment in POAG by measuring the sensitivity of motion stimuli varying with respect with the amount of neural processing involved in resolving their motion direction. Our general findings are consistent with previous studies demonstrating decreased motion perception sensitivity for patients with advancing glaucoma.[21,26,28,29,61] In the present study, direction-identification thresholds for both simple (first-order) and complex (second-order) motion types were significantly elevated for patients with glaucoma. One implication of this finding is that the decreased sensitivity to luminance-defined first-order motion stimulus, which is processed in part by the retina, is in accordance with M-cell pathway damage in glaucoma.[15,17,21] Increased first-order motion thresholds suggest ganglion cell loss and damage within the retina itself. Our results also demonstrated a significant increase for second-order motion thresholds for the observers with glaucoma. However, this difference in threshold did not exceed the difference in threshold measured between the glaucoma and normal observers for the first-order motion condition. The finding that the complex-motion perception (that is mediated by a more complex neural network) was not selectively affected by glaucoma implies that there is no measurable higher-order cortical damage caused by the glaucomatous process, at least at early corticovisual areas (areas V2 and V3) where second-order motion is believed to solicit more processing than first-order mechanisms.[40,50,52]

Aging and Its Relation to Motion Perception in Glaucoma

Aging affects both peripheral and central aspects of visual information processing.[33,62,63] For example, gradual decline of temporal modulation visual fields,[64] form-from-motion processing,[65] and temporal processing speed[66] have been shown to be associated with an increasing age, as has global motion perception.[35] Previous studies using motion stimuli similar to those used in the present study have demonstrated that aging (i.e., in the absence of eye disease) affects the perception of complex motion stimuli to a greater extent.[34] Together, these findings suggest less efficient neural processing of complex visual motion information in the elderly. The present finding that the relative first- and second-order motion loss is similar in our glaucoma subjects suggests that these findings are not the consequence of normal aging, which would have resulted in a greater loss for the more complex, second-order stimuli. At least for the luminance and texture-based motion stimuli used in the present study, both lenticular senescence and retinal illumination had no significant effect on motion direction discrimination. Although the mean pupil size of our experimental groups was very similar, no correlation was found between pupil size and motion sensitivity when analyzed irrespective of group inclusion. Similarly, lenticular senescence did not affect sensitivity to the motion stimuli, whether they were defined by spatiotemporal variations in luminance (first-order) or texture (second-order). Based on these findings, we suggest that the decreased motion sensitivity demonstrated between the control and glaucoma groups were not confounded by either lenticular senescence and/or retinal illumination.

Clinical Application of Findings

The interpretation of results from studies using existing psychophysical tests used to detect early stages of glaucoma has been questioned, particularly on their ability to selectivity isolate either M or P system functioning.[67,68] The present study does not attempt to do so. Rather, by using two different motion stimuli differentiated only by the neural processing involved in resolving their motion direction (i.e., same spatial and temporal characteristics), an assessment of M pathway functioning at two different levels was possible, the second-order motion condition implicating increased neural processing. The finding that glaucomatous individuals were equally less sensitive to first-order and second-order motion stimuli has important clinical considerations regarding the type of stimuli to be used in glaucoma studies. Although both simple and complex motion analysis is initially based on the efficient spatiotemporal functioning of retinal ganglion cells, complex motion analysis (i.e., second-order motion, global motion) usually involves neurointegrative processing beyond the primary visual cortex (i.e., V2/V3 in the case of second-order motion and the medial temporal [MT] area in the case of global motion perception). Therefore, motion testing using complex motion stimuli like random dot or global motion[25–27,29] is dependent on both simple *and* complex neural analysis. If this is the case, there is an increased probability that elevated random dot/global motion may be the result of neural dysfunction, if present, in addition to any ganglion loss caused by glaucoma. As already mentioned at the beginning of this article, an example of such a scenario would be a glaucomatous person with dementia (i.e., Alzheimer disease). Complex motion perception has been shown to correlate with age[34] and significantly elevated for persons with dementia.[35–39] Given the similar demographics shared by persons with dementia and presenting with glaucoma, the sensitivity of global motion may be confounded by neural dysfunction resulting in, at the least, a less specific diagnostic method for assessing POAG.

CONCLUSIONS

Because our findings point to glaucoma as not involving higher-order motion sensitive cortical mechanisms, motion testing using simple stimuli that are processed at the eye and in the primary visual cortex (i.e., simple motion) may be an advantageous alternative with respect to its specificity for the early detection of the glaucomatous process.

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