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Motion Coherence Perimetry in Glaucoma and Suspected Glaucoma

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Motion direction sensitivity in glaucoma patients, glaucoma suspects and controls was assessed perimetrically at 22 visual field locations using small random dot kinematograms and a motion coherence task. For foveal stimulus presentations, mean motion coherence sensitivity was normal in both patient groups. However, nearly all glaucoma patients and about half of glaucoma suspects (all with normal visual fields as assessed with static perimetry) had some deficit of motion sensitivity. These were most pronounced and most prevalent in the superior field at 15 and 21 deg eccentricity. Glaucoma appears to produce a reduction in the normal integrative visual function necessary for the perception of global motion in textured displays and this disruption is non-uniformly distributed across the visual field. © **1997 Elsevier Science Ltd. All rights reserved.**

Glaucoma Ocular hypertension Motion perception Perimetry Motion coherence

MOTION COHERENCE PERIMETRY IN GLAUCOMA AND OCULAR HYPERTENSION

Recent psychophysical studies report that in the early stages of glaucoma visual processing may be impaired in a number of specific and subtle ways. Of diagnostic significance is the consistent observation that visual deficits may be observed in the presence of normal visual fields as assessed by conventional static perimetry. For example, deficits for detection of coloured stimuli (e.g. Johnson *et al.*, 1993; Sample *et al.*, 1993), pattern discrimination (Drum *et al.*, 1989), contrast sensitivity (e.g. Atkin *et al.*, 1979; Falcao-Reis *et al.*, 1990; Teoh *et al.*, 1990), flicker perception (Brussell *et al.*, 1985; Holopigian *et al.*, 1991; Schmeisser & Smith, 1989; Tyler, 1981; Tytla *et al.*, 1990), and motion perception (Bullimore *et al.*, 1993; Fitzke *et al.*, 1987; Joffe & Raymond, 1991; Silverman *et al.*, 1990) have all been reported among patients who perform normally on static visual field perimetry tests but are at risk for developing glaucoma (e.g. patients with ocular hypertension, OHT).

Collectively these studies provide strong evidence that significant neural damage can exist without affecting performance on a static visual field test. Indeed, post-mortem studies correlating retinal ganglion fibre loss and visual field defect indicate that as many as 35% of ganglion fibres may be lost in OHT patients who had demonstrated normal fields (Quigley, 1985), and that as many as 40% of fibres must be lost before static field defects become detectable (Caprioli, 1989, 1990; Hart *et al.*, 1978; Quigley *et al.*, 1982, 1989). The question is no longer whether retinal damage exists in a significant percentage of individuals at risk for glaucoma (and with normal static fields), but rather, how can retinal damage be best detected and, further, what retinal mechanisms are most likely to be affected. Careful psychophysical experiments, such as those investigating the role of cone mechanisms, i.e. blue-on-yellow perimetry studies (e.g. Johnson *et al.*, 1993; Sample *et al.*, 1993) have the potential to provide answers to these questions.

The purpose of the study described here was to investigate deficits of motion sensitivity in patients with glaucoma or suspected for glaucoma. Since most aspects of visual dysfunction in glaucoma are found to be non-uniformly distributed across the visual field, our main goal was to determine if perimetric variations in motion sensitivity for glaucoma and suspects were different from those for normal age-matched observers. To do this, we used small random dot kinematograms (RDKs) and a motion coherence technique that has been well-studied neurophysiologically in non-human primates (Newsome *et al.*, 1989; Newsome & Paré, 1988) and psychophysically in normal (e.g. Raymond, 1994; Snowden & Braddick, 1990) and abnormal humans (e.g. Baker *et al.*, 1991; Barton *et al.*, 1995). In this procedure observers judge the global direction of motion in a "noisy" dynamic dot pattern in which a small percentage of dots move in a single coherent direction whilst remaining dots are

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moved in random directions. A motion threshold is defined as the minimum percentage of coherently moving dots necessary for just correct direction judgement.

We chose to investigate this type of motion sensitivity because there are several anatomical reports indicating that in the early stages of glaucoma, retinal ganglion cells that project to the magno layers of the lateral geniculate nucleus (LGN) may be damaged preferentially (Glovinsky *et al.*, 1991; Quigley *et al.*, 1987). Since many aspects of motion perception, including motion coherence, are thought to be mediated primarily by the magno stream of the retinogeniculostriate pathway (e.g. Maunsell *et al.*, 1990; Van Essen *et al.*, 1992), tests of motion sensitivity may be particularly sensitive to damage from glaucoma. Moreover, this type of motion processing has been well-studied in non-human primates and many aspects of the underlying central neurophysiology are known.

Evidence that peripheral damage to the retinal fibres feeding the magno pathway may be echoed centrally resulting in impaired direction perception in glaucoma has been previously reported (Bullimore *et al.*, 1993; Fitzke *et al.*, 1987; Joffe & Raymond, 1991; Silverman *et al.*, 1990). Silverman *et al.* (1990) measured motion coherence thresholds in patients and controls using RDKs. They report small but significant elevations in motion coherence thresholds for both glaucoma and OHT patients relative to controls. In another study using similar stimuli, Bullimore *et al.* (1993) reported non-significant elevations in motion coherence threshold for glaucoma patients and for glaucoma suspects. They did report, however, that more patients could be identified as abnormal using a D_{min} measure of motion sensitivity.

Both studies and more recent replications of these findings (Trick *et al.*, 1995) have two aspects of their design which may have affected the efficiency with which they were able to detect visual deficits. First, Bullimore *et al.* (1993) and Silverman *et al.* (1990) used very large, centrally presented, random dot fields (19 and 60 deg, respectively) to test motion coherence sensitivity. A common feature of glaucoma is that once retinal neuropathy progresses to result in visual field defects, the functional loss is found typically in the peripheral, not central, visual fields. With large-field, centrally presented stimuli, spared central fibres could have mediated threshold responses even in the presence of significant peripheral pathology.

Second, these investigators used RDK display durations of 1 and 4 sec, respectively, resulting from the presentation of 67 and 46 separate stationary displays, or "frames" of dots, respectively. Not only would the long stimulus exposures used in the previous studies elicit smooth pursuit eye movements which can themselves provide a cue to stimulus direction, they also present the visual system with an extended opportunity to integrate and detect motion information. It is generally assumed that the motion coherence task requires central mechanisms to integrate a number of local motion events in order to derive a global direction perception. There is substantial evidence that the system is capable of integration not only across space but also over a number of frames (e.g. Snowden & Braddick, 1989). Even a significantly weakened low-level motion detection system, as might occur in early stage glaucoma, could probably transmit sufficient information to a central integrator if enough motion frames were presented. Thus, by assessing motion sensitivity using very large displays for long durations, the previous studies may have provided so much motion information that only faults in a highly weakened system would be detectable. The fact that glaucoma-induced deficits were observed in these studies despite the use of large fields and long durations suggest a robust abnormality which could be more efficiently probed using smaller fields and shorter durations.

In the present study we provided only a minimal motion stimulus (i.e. only five frames) and localized it narrowly in the visual field. We then measured motion coherence thresholds at 22 different visual field locations in three groups of subjects: glaucoma patients, glaucoma suspects and aged-matched healthy controls. We found that whereas motion coherence thresholds were normal for foveal presentation in both patient groups, large, significant elevations of threshold for peripheral, particularly superior, field locations were found for both the glaucoma and suspects groups.

METHODS

Subjects

Twelve primary open-angle glaucoma patients (six females, six males), 15 glaucoma suspects (six females, nine males), and 14 age-matched controls (nine females, five males) participated in the study. A summary of their ages, intraocular pressures (IOPs) and cup/disc ratios are provided in Table 1. Information about the presence of subtle optic nerve changes in the glaucoma suspects group was not available. However, none of these patients had cup/disc ratios for each eye that differed by more

TABLE 1. Clinical data

	IOP (mmHg)		C/D ratio		Age (yr)	
Group	Mean	SD	Mean	SD	Mean	SD
Control ($n = 14$)	14	2.94	0.29	0.08	55.10	14.80
Suspects ($n = 15$)	23.84	4.70	0.29	0.08	53.40	14.68
Glaucoma ($n = 12$)	28.08	5.83	0.62	0.10	56.70	13.22

IOP, intraocular pressure; C/D ratio, cup/disc ratio.

than one-tenth. Glaucoma patients were only included if they had cup/disc ratios of >0.4 whereas suspects and controls were asked to participate only if their cup/disc ratio <0.4. All glaucoma and glaucoma suspects had a documented history of elevated IOP, i.e. IOP of >21 mmHg measured on at least two occasions, whereas all controls had IOP measured at <21 mmHg (measured on one occasion only). IOP was measured on the same day as static field testing and it is these IOP data that are shown in Table 1. Half the glaucoma suspects, all the POAG patients and none of the control subjects were on medical therapy. All participants had static visual fields measured within 6 months of motion testing using the Humphrey 30-2 perimetry test and these data were used here. Only suspects and control subjects with all thresholds within 5 dB of the norms and no other indicators of abnormal static fields (e.g. normal mean defects) were included in the study. We restricted our glaucoma group to glaucoma patients who had at least one point of >15 dB, or two contiguous points of >10 dB or three contiguous points of >5. No subject had evidence of systemic or other disease, a history of neurological disease, or a history of strabismus. No aphakic or pseudophakic eyes were included. All control subjects had normal acuity, normal colour vision and no family history of glaucoma. Glaucoma patients and suspects were recruited as they were encountered at the Glaucoma Clinic at Calgary's Foothills Hospital. Control subject volunteers were recruited from staff and their families and friends at the University of Calgary. Ophthalmologic screening and static perimetry testing of all subjects was done at the Glaucoma Clinic. Informed consent was obtained after the procedures were explained.

Apparatus

RDKs were generated by a Macintosh IIx computer using custom software which limited display timing to integrals of 16 msec. Stimuli were displayed on a Moniterm 2000 19″ monitor (P20 phosphor, 72 dpi, 62.5 Hz) placed on an adjustable tripod stand 65 cm in front of the subject. Responses were recorded using a joystick (Mousestick, Gravis). A forehead restrainer and chin rest were used to stabilize head position. A black hemisphere (diameter of 10 cm) was positioned in front of the untested eye to occlude vision. Room luminance was 3.3 cd/m^2.

Vertical and horizontal eye movements were monitored using an infrared corneal reflection device (Eye-Trac 210, Applied Science Laboratories; sampling rate = 50/sec, resolution = 0.5). Digitized eye position information was added to a video image (Sony HVC-2800) of the motion display. The experimenter viewed a separate TV monitor depicting this composite image to determine fixational failures.

Stimuli

Each stationary frame of the RDKs consisted of 50 small (4.3 min arc) white (37.5 cd/m^2) dots placed randomly within a borderless 3 deg circular field (dot density = 10.6 dot/deg^2) viewed against a large black (2.5 cd/m^2) background. Michaelson contrast was 87.5%. Five successive frames (each 80 msec in duration) with no interstimulus interval were presented, creating a 400 msec RDK. For each frame of the RDK, a percentage of dots was designated randomly as "signal" and displaced spatially in the next frame by 12.6 min arc (producing an effective velocity of 2.63 deg/sec) in one of four cardinal directions (upward, downward, leftward or rightward). The remaining dots were designated as "noise" dots and were randomly repositioned within the stimulus area on the next frame. A conventional wrap-around algorithm was used for dots falling outside the stimulus area. For successive displacements, assignment of dots as signal or noise was random so that the movements of any one dot did not provide a reliable cue to signal movement direction, except at high coherence values. (It is possible that with high coherence, perception of a single dot may have mediated global direction judgements, possibly aiding patients with large motion sensitivity losses.)

Procedure

At the beginning of and throughout each trial, the participant fixated a 2 deg open circle with a central crosshair. He or she then initiated a trial with a button-press which caused a white 3 deg circle to be presented for 50 msec at the location to be tested. This stimulus served to capture attention to that location (Nakayama & Makeben, 1989). Immediately following its offset, a test RDK was presented. The subject's task was to indicate the global direction of the signal dots by pointing the joystick in the appropriate direction. Immediately after responding, feedback was provided to indicate either a correct or incorrect response.

The per cent motion coherence in each trial was determined using a computer automated staircase algorithm with variable step size. For the first three trials, step size was 50% of the previous value. Step size was then adjusted to 25% of the previous value for the next three trials and thereafter was set to 12.5% of the previous value. Per cent coherence was decremented after one correct response and incremented after one incorrect response. This four alternative forced-choice staircase converges on the 50% correct point on a psychometric function on which 25% correct is chance performance. After 10 response reversals, the staircase series was terminated and threshold was computed as the average of the last six reversals.

The first two stimulus presentations in a staircase series always consisted of 100% coherent motion. These stimuli helped subjects become adjusted to the new field location and provided a check that the RDK was fully detectable at that field location. Because it was easy to judge the movement direction, the first two stimuli in each series also served the important function of encouraging participants in the task. The direction judgements for the first of these stimuli were not used in the staircase algorithm nor in the computation of threshold.

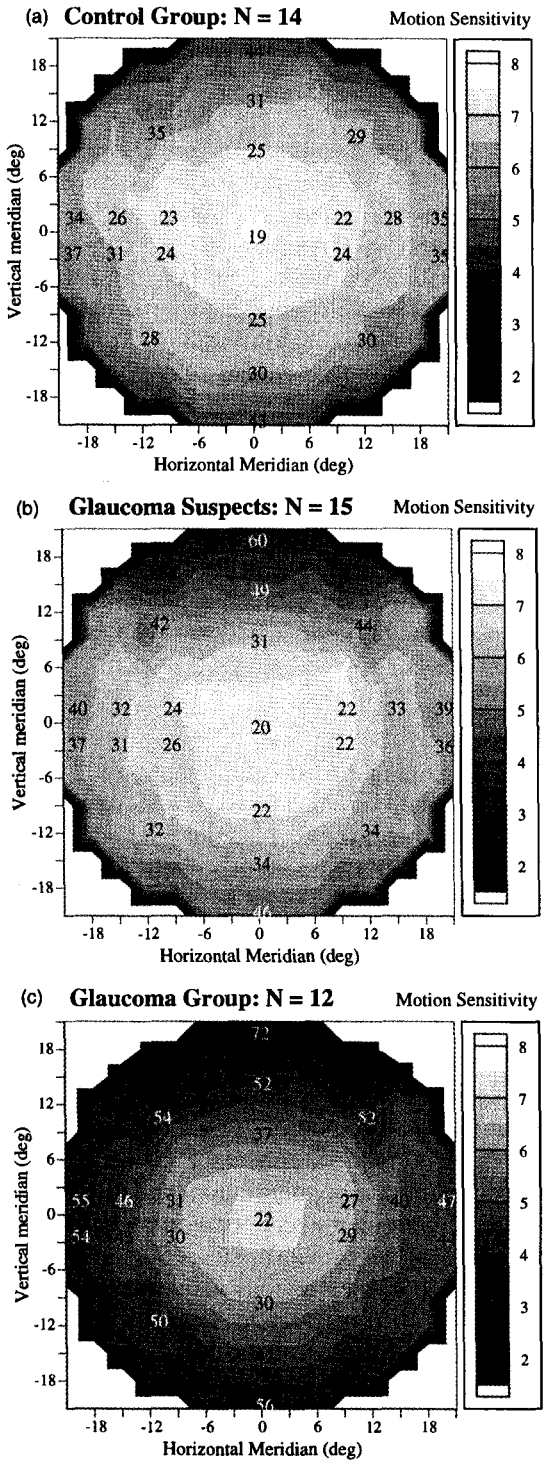

FIGURE 1. The group mean motion sensitivity as a function of visual field location (i.e. motion perimetry) for each group studied. Light areas indicate high sensitivity and dark areas indicate low sensitivity. Numbers on the figures denote group mean threshold in % coherence. Values above 45% are presented in white for clarity only. Along the horizontal axis, negative numbers indicate nasal hemifield locations and positive numbers indicate temporal hemifield locations. (a), (b) and (c) represent the control, the glaucoma suspect, and the glaucoma group data, respectively.

Within a single experiment session, 22 visual field locations, including the fovea and points at 9, 15 and 21 deg eccentricity, respectively, were tested in four separate blocks. These field locations are represented by the threshold numbers seen in Fig. 1. Stimuli along the nasal and temporal meridian were presented 2 deg above and below horizontal.

The fixation point was presented in one of the four corners on the monitor so that one-quarter of the field was tested in a block. Each block of testing included locations along the vertical, horizontal and oblique meridians. Adjustments of the monitor stand were made so that the fixation point remained directly in front of the subject's viewing eye, independently of its physical location on the monitor. The order in which blocks and locations were presented was random. Once a location was chosen, a complete staircase was conducted. Vertical and horizontal eye position was monitored throughout the session so that trials in which the subject failed to maintain central fixation could be eliminated. If, on a given trial, the subject made a saccade to a peripherally presented stimulus or to any other location, the trial was deleted from the staircase and a new RDK was then presented with the same % coherence but with a randomly chosen direction. Each session began with two practice staircases presented at two different randomly chosen locations. Rest periods were given frequently.

Subjects performed the task monocularly. For glaucoma patients and suspects, the eye with the greatest damage, or suspected damage (based on IOP and C/D ratio), was chosen. For controls, the eye to be tested was chosen randomly. Each subject participated in two testing sessions lasting *c.* 1 hr each. One threshold for each visual field location and four thresholds at the fovea were obtained in each session.

Static perimetry

Static perimetry was tested in a separate session using standard stimulus parameters for the Humphrey 30-2 field perimetry test. Conventional static perimetry thresholds are expressed in dB units converted from apostilbs, where 0 dB = 10 000 apostilbs and 50 dB = 0.1 apostilbs. Only those locations used in the motion perimetry test (Fig. 1) were included in analysis of static field performance as reported in the Results section, although all points were used to determine suitability for inclusion in the study. In those cases where static field locations did not perfectly correspond to motion field locations, sensitivity averaging was conducted.

Data analysis

Motion sensitivity. A sensitivity measure, in addition to the more conventional threshold measure, was used to ease comparisons with static perimetry sensitivity measures. The motion coherence thresholds obtained at a given location in each session were averaged and then converted into a motion sensitivity value using Eq. (1):

$$(100 - T)/10 \qquad (1)$$

where T is the average threshold percent coherence. This method of expressing sensitivity constrains the range of possible values between 0 (corresponding to a motion threshold of 100% motion coherence) and 10 (corresponding to a threshold of 0% coherence).

Field indices. Perimetric field indices were calculated for both motion and static perimetry. Mean sensitivity (MS), a global index reflecting generalized field sensitivity was computed using Eq. (2):

$$\mathrm{MS} = 1/I * \Sigma n_i \quad (2)$$

where I represents the number of locations and n represents the sensitivity value obtained at the ith field location.

Loss variance (LV) is used to index the variability in sensitivity losses across a subject's visual field. Unlike MS, LV is determined through comparison with "normal" field performance at each point in the visual field. For calculation of LV [Eq. (3)], the control group MS (M_{ctl}) for each visual field location was used as a comparison,

$$\mathrm{LV} = 1/I - 1 * \Sigma(Y_i^2) \quad (3)$$

where Y is derived using Eqs (4) and (5):

$$Y_i = n_i - (M_{\mathrm{ctl}i} - \mathrm{MD}) \quad (4)$$

where MD represents mean defect.

$$\mathrm{MD} = 1/I * \Sigma(n_i - M_{\mathrm{ctl}i}). \quad (5)$$

RESULTS

Motion sensitivity

Group mean motion sensitivity was calculated for each field location tested. Using an interpolation procedure (averaging between points tested), these data were used to produce the group mean motion coherence sensitivity maps shown in Fig. 1. Numbers on the figures denote group mean threshold in % coherence. Negative numbers along the horizontal axis represent the nasal hemifield and positive numbers represent the temporal hemifield.

Control group mean motion sensitivity measured at the fovea was 8.05 (SD = 0.25), which corresponds to a motion coherence threshold of 19.5%. This value is consistent with that previously reported for naïve, healthy adult observers (Raymond, 1994). Group mean foveal motion sensitivity for the glaucoma suspect was 7.92 (SD = 0.56) or 20.8% coherence, and for the glaucoma group was 7.81 (SD = 0.54) or 21.9% coherence. An analysis of variance (ANOVA) showed that neither group mean was significantly different from that of the control group [$F(2,38) = 1.00$, $P > 0.05$]. This finding provides evidence that all subjects understood the task equally. It further suggests that non-visual factors such as unfamiliarity with the task or fatigue effects cannot account for the motion sensitivity deficits found elsewhere in the visual field in the patient groups.

Normal visual sensitivity at the fovea is consistent with some previous studies (Brussell *et al.*, 1985; Tytla *et al.*, 1990; Falcao-Reis *et al.*, 1990) and supports the notion of macular sparing until late in the disease process. However, normal foveal sensitivity is inconsistent with Tyler's (Tyler, 1981) report of foveal flicker sensitivity loss in both glaucoma patients and suspects and is also inconsistent with central field losses reported for OHT and early glaucoma patients using blue-on-yellow luminance sensitivity (Adams *et al.*, 1987).

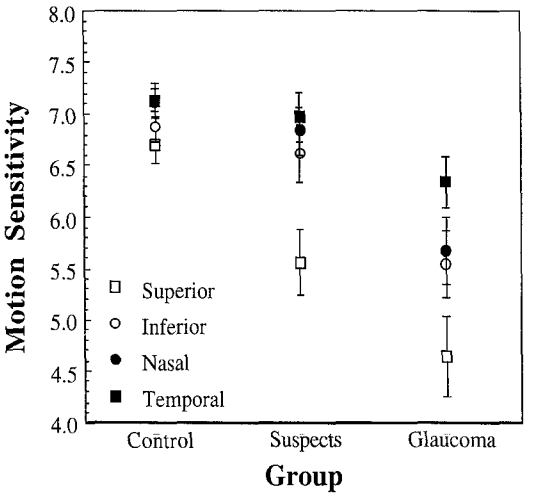

FIGURE 2. Group mean motion sensitivity for each area of the visual field for each group. Vertical lines indicate ±1 SEM.

Motion sensitivity decreased with increasing eccentricity for all groups. However, eccentricity effects were larger for both the glaucoma patient and suspect groups compared to that observed for the control group. The decrement in MS measured at 21 deg compared to that obtained foveally was 1.83, 2.23 and 3.28 for the control, suspect and glaucoma groups, respectively. To express this in another way, the group mean peripheral (i.e. 21 deg eccentricity) thresholds for the control, suspect and glaucoma groups were 37.8, 43.1 and 54.7%, respectively. An ANOVA on these data showed a significant group effect [$F(2, 38) = 10.25$, $P < 0.001$]. *Post hoc* Scheffe tests showed significant differences ($P < 0.001$) between the glaucoma and control groups but non-significant differences between the suspect and control groups.

As can be seen in Fig. 1(A), the change in control group sensitivity with eccentricity was relatively similar for the nasal, temporal, superior and inferior areas of the visual field. In contrast to this, sensitivity in the peripheral superior hemifield for the suspect [Fig. 1(B)] and glaucoma [Fig. 1(C)] groups was more reduced than in other areas. To examine this effect, we averaged motion sensitivity in each visual field area (superior, inferior, nasal and temporal) of each individual. For the superior and inferior areas, sensitivity values obtained at 9, 15 and 21 deg along the vertical meridian and 15 deg along the oblique meridians in the superior or inferior fields, respectively, were averaged. For the nasal and temporal areas, values obtained just above and below the appropriate half of the horizontal meridian were averaged. The group means are plotted in Fig. 2.

Motion sensitivity for the control group was similar in each of the four areas and was lowest in the superior area, a result consistent with a previous report (Raymond, 1994). Both patient groups showed the greatest deficit in the superior area and the smallest deficit in the temporal area when compared to controls. A multivariate analysis

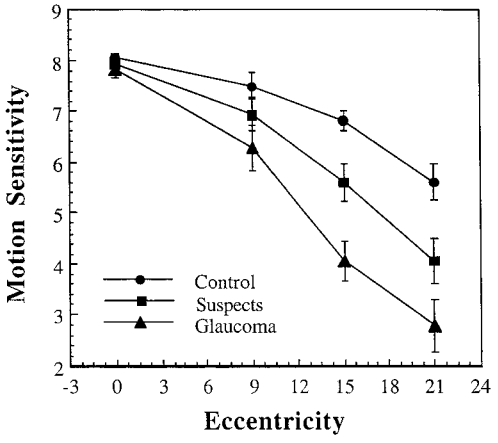

FIGURE 3. Group mean motion sensitivity as a function of eccentricity along the superior vertical meridian for each group. Vertical lines indicate ±1 SEM.

of variance on these data revealed a significant main effect of group [$F(2,38) = 11.15$, $P < 0.001$] and area [$F(3,38) = 46.14$, $P < 0.001$] as well as a group X area interaction [$F(6,114) = 3.71$, $P < 0.01$]. Planned comparisons showed that the suspect group was significantly ($P < 0.01$) less sensitive than the control group in the superior area only, whereas the glaucoma group was significantly ($P < 0.01$) less sensitive in all four areas in comparison to the control group.

We then examined individual differences in motion sensitivity for each of the four areas of the visual field. Using the control group mean and standard deviation (SD) to calculate a 95% normal limit, we observed that 11 of 12 glaucoma patients and 8 of 15 glaucoma suspects could be identified as abnormal on the basis of their MS in the superior field. Sensitivities in the remaining three areas of the field did not identify any further glaucoma patients. Nasal hemifield sensitivity was useful, however, at identifying visual deficits among two additional suspects, both of whom had normal sensitivity in the superior field. All controls appeared normal except one who was abnormal in both the superior and nasal fields.

Since performance for stimuli presented to the superior field seemed to provide the most sensitive indicator of visual deficit, these data are represented in more detail in Fig. 3. Both patient groups exhibited lower sensitivity than controls at all eccentricities, with greatest deficits at the most eccentric location tested. Whereas control subjects, on average, could just accurately judge motion direction in RDKs presented 21 deg eccentrically along the superior meridian when there was only 44.0% coherence in the display, glaucoma suspects and patients needed 59.5% and 72.2% coherence, respectively, to achieve the same level of performance. Comparisons between the suspect and control group means revealed a significant reduction in sensitivity at both 15 and 21 deg ($P < 0.01$). Glaucoma group sensitivity was significantly worse than controls at all eccentricities ($P < 0.01$).

We also analysed motion sensitivity data using more conventional visual field indices, i.e. MS and LV. MS provides a single measure of general sensitivity and is consequently relatively insensitive to local area of deficit. In contrast, LV is a measure of the variability in sensitivity loss across an individual's visual field. A single localized areas of deficit will produce a relatively high (i.e. abnormal) LV value and a relatively high (i.e. normal) MS value, whereas a uniform depression in sensitivity across the field will produce a low LV value and a low MS value.

Group mean motion MS and LV values are shown in Table 2. A one-way ANOVA revealed group differences in motion MS values [$F(2,38) = 10.63$, $P < 0.001$] and subsequent planned comparisons showed that the glaucoma group MS values were significantly less than that for the control or suspect group ($P < 0.01$). Differences between the suspect and control group were non-significant. A similar statistical analysis of motion LV values also showed a main effect of group [$F(2,38) = 9.85$, $P < 0.001$] and that these values were significantly greater in both glaucoma patients ($P < 0.01$) and OHT patients ($P < 0.02$) compared to controls.

Static perimetry

The inclusion criteria for our study required that glaucoma suspects and controls had normal static visual fields and that our glaucoma patients had evidence of deficits on this test. Our goal in reporting indices of static perimetry is to provide more information about the magnitude of the deficits in the glaucoma group and to provide evidence that the OHT and control group had no deficits at the field locations probed in the motion test. To do this, we derived our measures of luminance sensitivity using only the static perimetry data obtained from the same (or as near as possible) locations as were tested in the motion perimetry procedure.

As with motion sensitivity, luminance sensitivity measured foveally did not differ significantly among

TABLE 2. MS and LV indices calculated on motion and static sensitivity data

	Motion MS		Motion LV		Static MS		Static LV	
Group	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Control	6.98	0.53	0.69	0.26	30.60	1.55	1.28	0.35
Suspects	6.55	0.85	1.06	0.47	30.39	1.11	1.44	0.35
Glaucoma	5.63	0.87	1.50	0.55	25.09	2.83	6.39	2.94

MS, mean sensitivity; LV, loss variance.

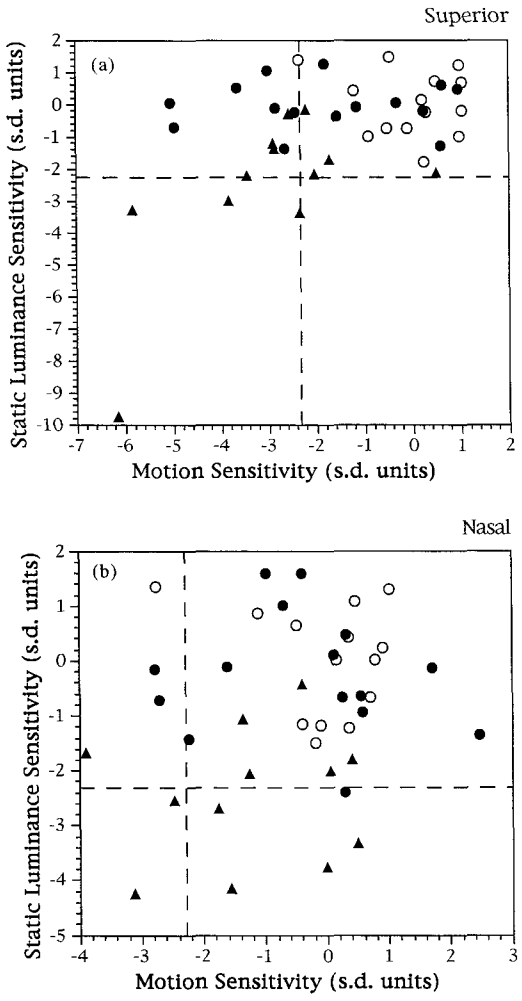

FIGURE 4. Individual performance on each test expressed in terms SD units different from the control group mean. ○, Control data; ●, glaucoma suspect data; and ▲, glaucoma data. The dashed lines indicate the lower 95% limit of the control group data for each measure. Points falling below or to the left of the dotted lines indicate abnormality on the static and motion tests, respectively. (a) Data measured in the eccentric superior field. (b) Data measured in the eccentric nasal field.

groups [$F(2,38) = 1.99$, $P > 0.05$] and had an average value of 36.5 dB. The glaucoma group sensitivity was significantly reduced [$P < 0.001$] in all four visual field areas when compared to either control or suspect groups. On average, the greatest deficit (22.33 dB, SD = 4.4 dB, compared to the mean of 30.58 dB, SD = 1.5, dB for controls) was found for the nasal area and the smallest deficit (27.65 dB, SD = 2.18 dB, compared to the mean of 30.61 dB, SD = 1.53, dB for controls) was found in the temporal area. Differences between the suspect and control groups for all areas were non-significant.

Group mean static MS and LV values are shown in Table 2. An analysis of variance on these data revealed a significant group effect for both MS [$F(2,38) = 34.53$, $P < 0.001$] and LV [$F(2,38) = 41.36$, $P < 0.001$]. MS and LV values for controls and suspects were non-significantly different as would be expected based on the inclusion criteria. Predictably, the glaucoma group was significantly ($P < 0.001$) different from the control group on both indices.

Motion vs static perimetry

Although our study examined a relatively small number of individuals and can provide only a cross-sectional comparison, we used the available data to examine the effectiveness of motion vs static perimetry at identifying visual abnormalities in individuals. To do this we compared four measures derived from the perimetry data: sensitivity in the superior field, sensitivity along the nasal meridian, MS and LV.

We chose to compare sensitivity in the superior field because it appears to be particularly sensitive to abnormalities in motion sensitivity. Sensitivity along the nasal meridian was compared because it is more likely to reveal abnormalities using the static test. In all cases, an average value for sensitivity measured at 15 and 21 deg was calculated for each field since these locations were observed most often to reveal deficits in the glaucoma suspect and patient groups. We then calculated the control group mean and SD for both measures for both areas of the field. Each subject's performance was quantified in terms of SD units different from the control group mean. Figure 4 shows the distribution of the values obtained for each subject. It also shows how deficits found with the motion test correlate with deficits found with the conventional perimetry test measured at the same locations. The dashed lines indicate the lower 95% limit of the control group data for each measure.

For the superior field, 8 of 15 (53%) suspects had motion sensitivity values lower than the normal limit and none had static values that fell outside the normal limit. One control subject also had a motion value outside the normal limit. Of the 12 patients in the glaucoma group, 10 (83%) were observed to be abnormal on the motion measure. Note that four of these patients had normal static sensitivity and that one control and one glaucoma patient had abnormal static sensitivity and yet performed well within the normal limits on the motion test.

For the nasal field, one control was abnormal on the motion test. Only one glaucoma suspect appeared abnormal on the static measure and two were abnormal on the motion measure. Of the glaucoma group, six were abnormal on the static measure and three showed significant deficits on the motion test. As with the data from the superior field, there is evidence of dissociation of deficits on the two measures. There were five patients who appeared normal on the motion test but were abnormal on the static test at the same locations. Four different subjects were abnormal on the motion test and normal on the static test. Such dissociations point to an advantage of using a perimetric technique in motion assessment, particularly from a theoretical perspective, because they suggest strongly that different neural mechanisms may mediate these thresholds.

Using MS values, we observed that four suspects (27%) and no controls had MS values lower than the lower 95% limit for the motion test. No-one in either

group was abnormal for the static MS test. Eight (67%) of the glaucoma patients were abnormal using the motion MS measure compared to the ten glaucoma patients (83%) observed to have static MS deficits. No controls and seven suspects (47%) had motion LV values higher than the 95% confidence limit. One suspect and one control subject (7%) were found to be abnormal using the static LV measure. The motion LV measure identified nine glaucoma patients (75%) as abnormal whereas 100% of glaucoma patients were identified as abnormal using the static LV measure.

DISCUSSION

We measured perimetric sensitivity to the global direction of motion using small-field, partially coherent RDKs in glaucoma patients with visual field defects and in a group of age-matched controls. We also measured motion sensitivity in patients who were suspect for glaucoma, i.e. patients who had elevated IOP but who had no visual field defects as assessed by conventional static perimetry. Three important findings emerged. First, in addition to their static field losses, glaucoma patients were found to have large deficits in motion sensitivity. Second, a large number of glaucoma suspects were found to have significant deficits of motion perception, indicating the presence of visual neural pathology in these patients. Third, motion sensitivity losses were not uniformly distributed across the visual field. In both patient groups, motion sensitivity losses were greatest in the superior visual field and were absent in the fovea.

Our main finding that motion sensitivity is degraded with glaucoma and OHT replicates previous reports of motion sensitivity losses in these patient populations (Bullimore *et al.*, 1993; Fitzke *et al.*, 1987; Joffe & Raymond, 1991; Silverman *et al.*, 1990). We report that 83% of the glaucoma patients and 53% of the glaucoma suspects studied here could be identified as abnormal on the basis of their sensitivity to coherent motion in peripheral locations within the superior hemifield. Although we tested a relatively small number of patients and our study is cross-sectional in design, these data suggest that motion perception testing may be usefully developed into a sensitive test of visual dysfunction. Since we did not obtain detailed analysis of subtle optic nerve changes in the glaucoma suspects, it is possible that some of these individuals could have been equally well identified as glaucoma patients on this basis.

Using the behavioural data, our detection rate is considerably higher than the 17% of glaucoma patients and 0% of glaucoma suspects that Bullimore *et al.* (1993) reported as having abnormal motion coherence thresholds. It is also somewhat higher than that identified as abnormal by Silverman *et al.* (1990) using a similar technique. Although they report 44% of OHT patients as abnormal, when the same conservative criterion that we used is applied to their data, only *ca* 21% of their OHT patients would be considered abnormal.

There are a number of reasons why our methods may have been more sensitive to deficits than those previously used. First, both Bullimore *et al.* (1993) and Silverman *et al.* (1990) used very large, centrally presented, random dot fields (19 and 40 deg, respectively) to test motion coherence sensitivity. Since we observed that all of our glaucoma patients had intact sensitivity for foveal stimuli it is most likely that in these previous reports, many patients were able to use their foveal regions to mediate threshold responses. Second, these investigators used display durations of 1 and 4 sec, respectively, resulting from the presentation of 67 and 46 separate stationary displays, or "frames" of dots, respectively. In contrast, our stimuli were present for a total duration of 220 msec and consisted of only five frames.

The long stimulus exposures and large stimulus areas used in the previous studies presented an extended opportunity for the perceptual mechanisms to detect and integrate information about local motion both in space and time. In order to perform the motion coherence task, the subject must make use of consciously available perceptual signals regarding movement direction which are presumably derived from cortical integration, i.e. smoothing, of "noisy" local motion events. The very observation that in normal subjects only a very small percentage of coherently moving dots is needed to produce this global directional percept indicates that the central integrator is capable of producing a perceptually accessible signal with a very limited amount of information. Thus, even a severely weakened low-level (i.e. peripheral) network of motion analysers would be able to supply a central integrator with sufficient information to perform the task well if the number of motion frames and the stimulus area were large enough. By providing only a minimal motion stimulus (i.e. few frames) and localizing it narrowly in the visual field, we were able to tax the central integration system sufficiently to reveal visual pathology.

Using a perimetric approach to assess motion sensitivity allowed us to observe patterns in the location of "motion scotomas". In both the glaucoma patient and suspect groups, motion deficits were greatest in the superior visual field, especially at 15 and 21 deg eccentricity, and least observable in the temporal visual field. This pattern of superior field loss, consistent with some perimetric studies of colour (Sample & Weinreb, 1990), flicker (Tytla *et al.*, 1990), and temporal contrast sensitivity (Falcao-Reis *et al.*, 1990), suggests that the inferior pole of the optic disc is highly susceptible to damage in the presence of elevated IOP. Indeed, anatomical (e.g. Caprioli, 1989; Carassa *et al.*, 1991) and physiological (Bray *et al.*, 1991) evidence indicates that the greatest damage to ganglion cells occurs in the inferior pole of the optic nerve head, with minimal damage to fibres mediating temporal field sensitivity. The observation that motion sensitivity losses were greatest at 15 and 21 deg eccentricity supports the possibility that arcuate fibres are particularly susceptible early in the disease (Airaksinen *et al.*, 1984).

The observation of deficits in motion sensitivity in glaucoma patients and suspects has a number of

important implication for visual function in these people. It is widely believed that many aspects of motion perception, and especially those aspects measured here, depend heavily on the input processed via the magnocellular stream of the primary visual pathway (Maunsell *et al.*, 1990; Newsome *et al.*, 1989; Newsome & Paré, 1988; Van Essen *et al.*, 1992). This neural stream provides the primary input to the dorsal visual pathway leading from the striate visual cortex through extrastriate cortex and into the parietal lobe. The dorsal extrastriate structures and their projection sites in the parietal lobe are believed to play a fundamental role in the perception of motion, smooth pursuit eye movements, and the visual control of action and locomotion (Goodale, 1993; Van Essen *et al.*, 1992). If the motion perception deficits observed here in glaucoma patients are a reflection of pathology to the magnocellular system, then our findings suggest that these patients may be dysfunctional on a wide range of functions dependent on visual motion information such as visually guided reaching, eye movements and visually guided locomotion. Continued study of visual motion deficits in this group may not only be useful in developing methods for early detection of visual pathology in patients at risk for glaucoma, but may also provide insights into the perceptual and functional correlates of this disease.

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