

# Peripheral contrast sensitivity in glaucoma and ocular hypertension

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## Abstract

Contrast sensitivity has been measured in patients with glaucoma and ocular hypertension, the latter graded into high, medium, and low risk clinical groups. Measurements were made centrally and peripherally at 10°, 15°, 20°, and 25° off-axis at each of the four meridians 45°, 135°, 225°, and 315°. A sine wave grating of 1.9 cycles/degree, reversing at 1 Hz was used. It was displayed on a 100-Hz refresh rate monitor. Normal values were established to compare those from 41 eyes from patients with either primary open angle glaucoma (POAG) with minimal field loss detectable on a Humphrey perimeter, or raised IOP and/or disc changes but no field loss (OH). Those with POAG had normal central contrast sensitivity, but at 20° and 25° eccentricity the values were >2 standard deviations above the normal mean. This was also the case for high risk OH, but not for low risk patients. All the high risk patients except one who had abnormal peripheral contrast sensitivity had possible field defects (threshold elevation at one or more points more than 5 but less than 10 dB above normal mean). Only one of those with normal peripheral contrast sensitivity had such 'suspect points'. The results are assessed in terms of screening of glaucoma suspects.

Primary open angle glaucoma (POAG) is diagnosed when a patient has demonstrable visual field defect and either a raised intraocular pressure (IOP) or clinical signs, such as disc changes, which are consistent with a raised IOP, and no other complicating causes for the raised pressure. There are several tests which aid diagnosis. Perimetric loss defines the condition,<sup>1</sup> but its sensitivity is not great enough to detect the earliest pathological changes: 40% of optic nerve axons are lost before any visual defect is apparent.<sup>2</sup> Increases in pressure occur long before field is lost, and the degree of increase in IOP gives an indication of the probability that the patient will subsequently develop a characteristic field loss.<sup>3</sup> However, it is impossible to screen for glaucoma on the basis of ocular pressure measurement only. No level of IOP can be found at which the percentage of 'false positives' and 'false negatives' is acceptable.<sup>4</sup> Examination of the optic nerve head, especially with stereoscopic viewing, is an efficient and effective method of detecting damage to the optic nerve, but it requires expert judgment.<sup>5</sup> A further limitation derives from the fact that patients who do not have any pathological process may have discs which seem abnormal.<sup>6</sup>

Another problem in treating glaucoma is to

decide when to begin. It is often considered to be too late to wait until a frank field defect is found<sup>7-9</sup> and so one should treat patients with raised IOP and/or disc changes but no field loss (OH) in whom the condition is progressing. Estimates from the measurement of IOP are of limited use in deciding when to treat, as is a slight increase in a high cup/disc ratio. Therefore various subclassifications of OH have been devised in an attempt to quantify the risk that field loss may develop, and only patients in high risk groups are treated.<sup>10</sup> This limits the cost of treatment and minimises the possibility of adverse reactions. To a considerable extent risk classification rests on clinical judgment. Recently some other diagnostic methods have been used to detect and grade the small degree of damage to the optic nerve which occurs in OH – for example, colour discrimination measured with desaturated colours<sup>11</sup> or colour contrast sensitivity.<sup>12</sup> The pattern electroretinogram<sup>13</sup> especially has been found abnormal in ocular hypertension and appears to be more successful than the psychophysical techniques in detecting abnormality in glaucoma and high risk OH, possibly because a large retinal region is tested, while psychophysical methods measure only macular or even foveal function.

Another method claimed to detect visual changes in glaucoma and OH is the measurement of contrast sensitivity. Several techniques have been used,<sup>14-17</sup> and testing is as fast, easy to perform, and inexpensive as is required for screening. Unfortunately, early hopes of sensitivity and specificity of that method were not realised.<sup>18</sup> Although losses in contrast sensitivity at low spatial frequency occur in patients suffering from glaucoma and OH, there is an unacceptable overlap with normals, even when the grating pattern is modulated as a function of time (which improves detection of early losses<sup>19</sup>). Another unexploited value of temporally modulated gratings is that with them the Troxler effect is absent, and it is thus possible to measure contrast sensitivity in untrained subjects as far peripherally as 25–30° from the fovea. There have been no previous reports of such measurements in OH patients, but since the first field loss occurs peripherally it seems likely that the first losses of contrast sensitivity will occur in such regions. We have accordingly measured peripheral contrast sensitivity in eyes with early glaucoma and ocular hypertension at different risks of conversion of glaucoma. By the time glaucomatous eyes show the earliest evidence of visual field defect, that is, one spot of 10 dB loss, contrast sensitivity is greatly reduced when measured in the periphery. Determination of contrast sensitivity in four quadrants at 20° or 25° (four points)

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served to separate all eyes with glaucoma from normal eyes, while testing at the macula was totally ineffective. Furthermore, in 50% of eyes with high risk ocular hypertension there are definite losses of contrast sensitivity at an eccentricity of 20° and 25°.

#### Material and methods

A TV monitor controlled by a T221 grating generator (Faulkner-West Associates) was used to present a vertical sinusoidal grating in a pattern reversing mode. The refresh rate was 100 Hz. Thus there was no 'TV flicker' even for peripheral viewing. Contrasts as low as 0.1% could be generated. The TV screen was surrounded by a white card of 80×80 cm, on which small fixation spots were affixed at appropriate locations. Most of the TV screen was also covered by the card except for an 8 cm round area in the middle. In this hole 6 cycles of the grating could be seen. Patients sat in a chair at 1.2 m from the screen. At this distance stimulus and surround subtended 3.8° and 33° respectively. Spatial frequency was 1.9 cycles/degree. An abrupt reversal (square wave) with a temporal frequency of 1 Hz was used. Both remained unchanged during testing. The mean luminance of the TV screen was 80 cd/m<sup>2</sup>. Daily calibrations of contrast and luminance were carried out with a Tektronix J16 photometer. Room illumination was 10 cd/m<sup>2</sup>. The operator sat at the side of the monitor, facing the patient, to check fixation. Threshold was determined by the method of ascending limits. Contrast, defined in the usual manner, was increased slowly and smoothly by the manual operation of a 10-turn calibrated potentiometer either from zero or from a variable subthreshold value. Patients were instructed to respond as soon as they could detect motion of the pattern. At least three measurements were averaged to determine a threshold at each locus. In a few cases the stimulus lay on a scotomatous region of retina, so no meaningful measurement could be made. In such cases the threshold contrast was arbitrarily given a value of 30%. Pupil diameter was not measured, but none of the patients was treated with miotics.

Patients and subjects wore spectacles when appropriate. Tests were carried out monocularly. The grating had an unchangeable spatial frequency, so no pattern preview was given. No time limit was imposed. Contrast threshold were measured at 10°, 15°, 20°, and 25° eccentricity at the 45°, 135°, 225°, and 315° meridians: these were chosen so that the blind spot was avoided. In addition the threshold was measured with foveal viewing. A full examination – 17 loci each

eye – took about 45 minutes. All contrast sensitivity determinations were carried out by the same operator, who was not aware of the patients' condition.

Forty-one eyes (mean age of patients 57 (SD 8) years, 27% female, 73% male) with glaucoma or hypertension recruited from a glaucoma clinic and 11 eyes (mean age of subjects 49 (SD 12) years, 36% female, 64% male) with normal eyes were examined. Hospital staff members with no ocular or systemic diseases volunteered as controls.

The doctors who carried out the contrast sensitivity test did not recruit the patients and did not carry out any clinical examinations, and were not aware of the field test results.

The patients' eyes were examined by biomicroscopy, funduscopy, disc photography, applanation tonometry, and Humphrey computerised static perimetry, and were classified by the usual criteria as suffering from either glaucoma or ocular hypertension (Table 1). Glaucomatous eyes had a reproducible visual field defect defined as one or more spots of sensitivity loss of 10 dB or greater at least in two different occasions plus one adjacent spot of 5 dB. Hypertensive eyes were further classified into three groups.<sup>20</sup> The low risk group had an intraocular pressure below 26 mmHg and a vertical cup to disc (C/D) ratio less than 0.6. Eyes with either an intraocular pressure equal to or greater than 26 mmHg or a C/D ratio equal to or greater than 0.6 were classified as medium risk. The high risk group included eyes with intraocular pressure greater than 26 mmHg and C/D ratio greater than 0.6. Additionally eyes with OH were classified as 'suspects' when loci in the sites tested by the Humphrey 30-2 program had a sensitivity loss greater than 5 dB but less than 10 dB.

**Exclusions.** Patients and normal subjects had a visual acuity of 6/6 or better and no ocular disease particularly cataract or corneal opacities. None had systemic hypertension, diabetes, or other disease.

#### Results

##### CENTRAL RETINAL CONTRAST SENSITIVITY

Each patient or observer was asked to look directly at the grating, and contrast sensitivity was measured. The mean value of the macular region in normal control eyes was 0.91% (SD 0.13). This was not significantly less than the contrast sensitivity of low and medium risk OH groups.

The difference between the normals and the high risk group was greater, though the significance was only 0.1 > p > 0.05. Glaucomatous eyes showed a significant loss of sensitivity (p > 0.001). Of greater interest is the distribution of contrast sensitivity in the population. This is shown in Fig 1. For central viewing, in two out of eight cases of glaucoma the thresholds were just below the upper limit of normal. This sort of overlap has previously been reported and explains why central contrast sensitivity (CS) is not an adequate screening technique.

Table 1 Classification criteria

	Normal	Low risk	Medium risk	High risk	Glaucoma
IOP (mmHg)	<21	≥21, <26	≥26 or	≥26 and	Any
C/D ratio	<0.6	<0.6	≥0.6	≥0.6	≥0.6
Visual field	Normal	Normal or suspect	Normal or suspect	Normal or suspect	Abnormal

Suspect: one or more spots ≥5 dB ≤10 dB.

Abnormal: one or more spots ≥10 dB plus 1 spot ≥5 dB.

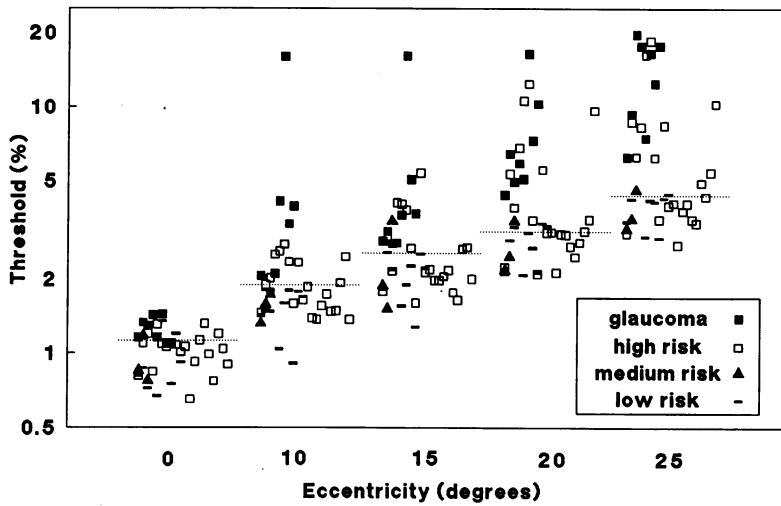


Figure 1 Distribution of mean contrast sensitivity as a function of retinal eccentricity. All individual results shown. Each symbol represents (except for central viewing) the average of results from four loci, one in each quadrant. The different symbols refer to the patients' diagnoses. The dotted line represents the upper limit of normal (mean + 2 SD). It can be seen that contrast sensitivity decreases from the centre to the periphery. Note that two glaucomatous eyes give results within the 5% confidence limit for foveal testing. However, for peripheral viewing the distinction between the results in glaucoma and the upper limit of normal is much more evident. For patients with high risk OH the proportion with reduced contrast sensitivity also increases from centre to periphery and reaches 50% at 25°.

PERIPHERAL CONTRAST SENSITIVITY

Normals

Mean values for measurements in all four quadrants and the SD are shown in Table 2. Contrast sensitivity at most eccentric positions was considerably less than with central fixation, and decreased with eccentricity. The upper limits of normal (mean + 2 SD) are represented by the horizontal dotted lines on the graph of Fig 1. These results are consistent with previous findings.<sup>15 18 19</sup>

Glaucoma

The distribution of individual results is shown for peripheral CS in Fig 1. Fig 2 shows the average contrast thresholds at each retinal position. Note that for normals the symbols are at the mean plus 2 SD. In the superior quadrants

Figure 2 Contrast sensitivity at eccentric loci in glaucomatous eyes. Results are compared with upper limit of normal (mean + 2 SD). Each symbol represents the mean of thresholds of all glaucomatous eyes at a given locus. Difference between normals and glaucomatous eyes is evident at all loci in the lower field and at 20° and 25° in the superior quadrants. Loss of sensitivity in glaucomatous eyes is less pronounced in the superior nasal quadrant.

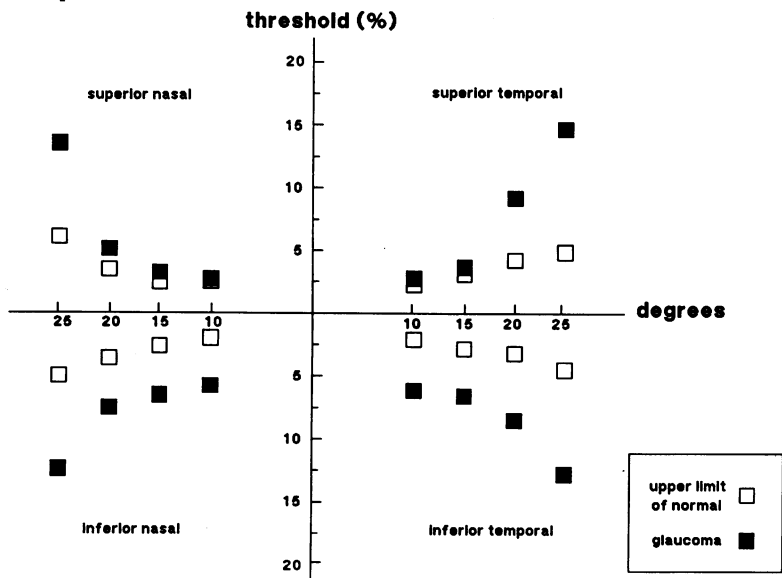


Table 2 Mean contrast sensitivity of four quadrants

Retinal position	Category of patient				Glaucoma
	Normal	Low risk OH	Medium risk OH	High risk OH	
Central					
Mean	0.91	0.93	0.93	1.03	1.25
SD	0.13	0.23	0.22	0.17	0.14
Significance*		NS	NS	<0.1	<0.001
10°					
Mean	1.57	1.51	1.54	1.89	4.34
SD	0.20	0.33	0.21	0.46	4.78
Significance*		NS	NS	<0.05	<0.05
15°					
Mean	2.01	2.03	2.27	2.59	5.00
SD	0.30	0.43	1.01	1.0	4.54
Significance*		NS	NS	<0.1	<0.05
20°					
Mean	2.56	2.76	2.69	4.50	7.58
SD	0.31	0.54	0.66	3.00	4.00
Significance*		NS	NS	<0.05	<0.001
25°					
Mean	3.63	3.90	3.76	6.47	13.30
SD	0.47	0.62	0.74	4.30	5.19
Significance*		NS	NS	<0.05	<0.001

\*† Test of normal against each class of patient: figure is probability that the result could be obtained by chance. NS = not significant.

of the visual field the difference between glaucomatous eyes and controls at 10° and 15° is small, but in the lower quadrants it is quite evident. In the two most peripheral loci (20° and 25°) the difference between the two groups is clear in all quadrants. For glaucomatous patients, contrast sensitivity, averaged over the four quadrants (Fig 5) is significantly lower than for the normals (the p values range from <0.005 to <0.001 depending on eccentricity). Note that the difference between normal and glaucomatous eyes is less in the superior nasal quadrant, where the scotomata are less frequent.

Not only are the differences between means significant, but the discrimination between affected and normal is nearly complete if the results from each of the four quadrants are averaged as in Fig 1.

There is only one false negative at 10° and none at 15°, 20°, and 25°. It might be considered that this averaging biased the result, since contrast sensitivity would be zero over a scotoma. Contrast sensitivity is not directly related to luminance incremental threshold (which is measured by perimetry, but evidently when increment threshold is infinity (that is, the stimulus lies on an absolute scotoma) the contrast threshold must also be immeasurably high. Only five eyes had a threshold higher than 30%. Among these one glaucomatous eye had a threshold higher than 30% at four retinal loci in one quadrant. These were scored as 30% (see 'Methods' section). Thus the results given in the figures under-represent the difference between normal and glaucomatous eyes.

OCULAR HYPERTENSION

Low risk group

Some differences between normal persons and OH patients can be observed. Low risk OH eyes had an average contrast sensitivity that was consistently less than the upper limit of normal (Fig 3) at all retinal eccentricities. When the results of all quadrants were averaged, the results

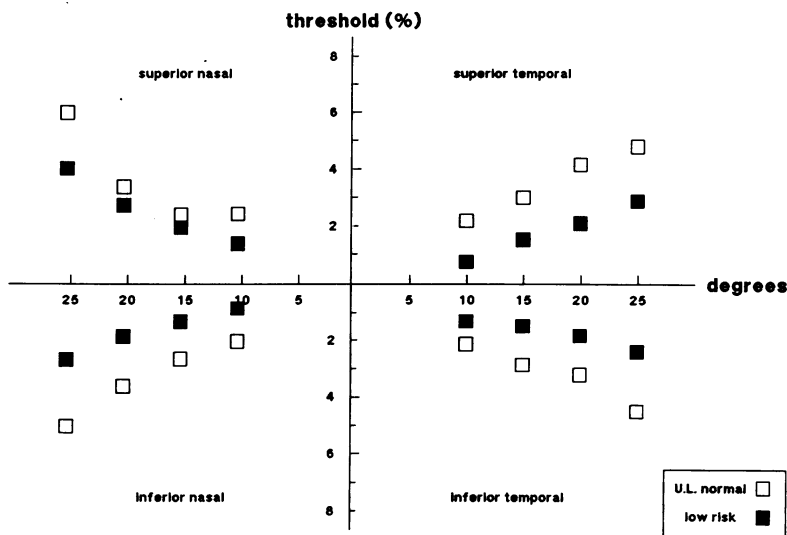


Figure 3 Contrast sensitivity at eccentric loci in low risk hypertensive eyes. Results are compared with upper limit of normal (mean + 2 SD). Each symbol represents the mean of thresholds of all eyes at a given locus. Thresholds are similar in all quadrants. Mean contrast sensitivity is consistently under the upper limit of normal.

were also under the upper limit of normal for all eccentricities (Fig 5). As might be expected, the differences between this group and normal controls were not statistically significant (Table 2). Fig 1 shows the individual results, averaged over four quadrants, for the low risk group: only in two individuals, at 20°, were the results above the upper limit of the normal mean. Even in some low risk patients there was evidence of retinal damage, and a proportion of patients progressed to frank glaucoma. The few high thresholds encountered may indicate such lesions, but to provide evidence for this would require prolonged follow-up. More important, the finding that most low risk patients have results within our normal range shows that there is a low incidence of false positives.

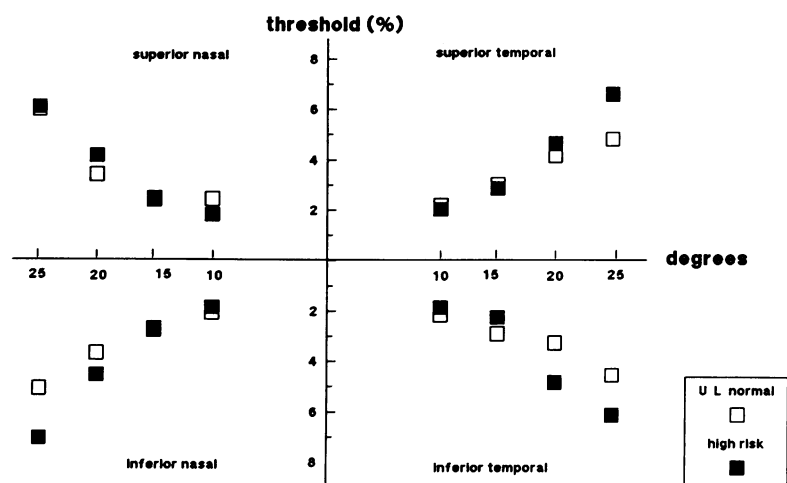
Figure 4 Contrast sensitivity at eccentric loci in the eyes of patients with high risk OH. Results are compared with the upper limit of normal (mean + 2 SD). Each symbol represents the mean of thresholds of all eyes at a given locus. There is overlap between the upper limit of normal and high risk OH eyes. Again, loss of sensitivity is less accentuated in the superior nasal quadrant. Note the different scale of ordinate compared with Figure 2.

Medium risk group

The medium risk group included only three eyes and results for each quadrant are not shown. Fig 5 shows the mean contrast sensitivity of all quadrants, and it can be seen that is within normal limits.

High risk group

Fig 4 shows the mean results obtained in each locus in the four quadrants. These are compared with the upper limit of normal from the normal



population. For 10° and 15° off fixation the two sets of symbols overlap, but for 20° and 25° in seven of the eight determinations the OH eyes' thresholds were significantly raised.

The exception was the upper nasal quadrant at 25°. Therefore the results for all four quadrants were averaged, as shown in Fig 5. Loss of sensitivity in the two most peripheral positions is very evident in Fig 5, and this difference between high risk OH and normals is significant (0.005 > p > 0.001). Abnormal thresholds at 25° and 20° were found in 50% and 40% respectively of high risk eyes (30% and 24% of total OH eyes). If individual results are considered, at 25° of eccentricity the group of high risk eyes and a single medium risk eye had abnormal thresholds. Among the high risk cases which were abnormal were eyes in which there was a 'doubtful' field defect, defined as a determination in which threshold was raised at one or more spots by more than 5 dB but less than 10 dB. 50% of high risk eyes had thresholds under the upper limit of normal. Table 3 shows that there was a nearly complete separation of the two groups. The presence of possible field defects was associated with reduced contrast sensitivity, and in those patients who had normal contrast sensitivity 'possible' field defects were infrequent. It should be emphasised that this finding was made only after the end of the project, when the codes were broken. The Fisher exact probability that the two factors are unrelated is vanishingly small, p = 0.0000238.

Discussion

In this study simple techniques were used to measure contrast sensitivity in patients' peripheral retina. Such measurements can be made reliably in our population, which, however, is selected: the patients know they are in several clinical trials and are highly motivated. Likewise our normal values are not obtained from spouses, and are not strictly age matched. However, this is not of importance for this project, in which the relation between peripheral and central contrast sensitivity (in the same individuals) is to be compared. Problems related to general screening are discussed below.

We have shown that, by comparison with normal people, patients with glaucoma have raised contrast thresholds for gratings of low spatial frequency, but this is true only for peripheral retina. (It would be difficult to investigate high spatial frequencies in the retinal periphery.) Abnormal peripheral contrast sensitivity in glaucoma has already been reported.<sup>21,22</sup> In addition we have investigated patients with OH, and in some of these too contrast thresholds are raised. In patients with no definite field defects, but with clinical signs which suggest that retinal damage is occurring, there seems to be a highly significant loss of peripheral contrast sensitivity. This might be expected, for it is known that in experimental glaucoma those neurons responsible for the detection of low spatial frequencies and of movement, which subservise the magnocellular system, are selectively affected early in the course of disease.<sup>23</sup>

Furthermore, the present results show normal

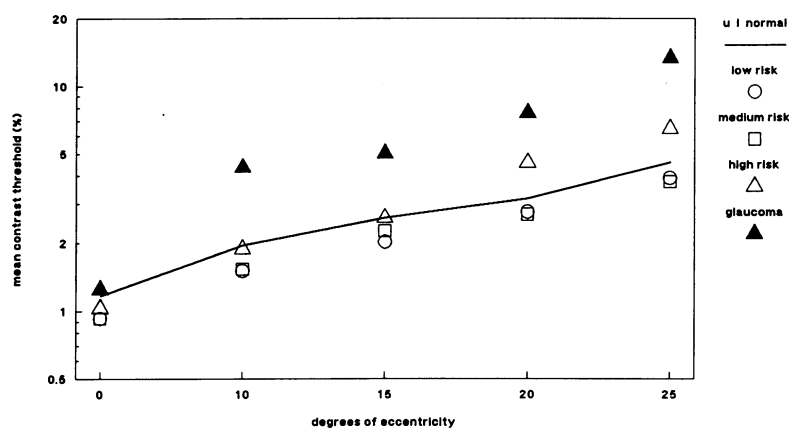


Figure 5 Mean contrast sensitivity averaged over all four quadrants. The symbols refer to different categories of patient, as indicated. The figure shows how the average of contrast sensitivity at four quadrants changes with eccentricity. The line represents upper limit of normal (mean + 2 SD). It can be seen that the thresholds of high risk OH eyes are above normal but only at eccentricities of 20° and 25°. The eyes of patients with field loss have thresholds that are just above normal at the fovea but become greatly elevated at all other loci. Low risk and medium risk OH eyes have thresholds that are under normal limits both at the centre and at all peripheral loci.

contrast sensitivity in the retinal periphery of patients with low and medium risk OH. In these patients there is only a low probability that field loss will later develop. Thus it is plausible that the 50% of cases of high risk OH eyes with abnormalities represent that subpopulation which will progress to frank field loss. If so, this would represent a significant improvement in deciding the prognosis of all suspects.

These findings once more raise the possibility that contrast sensitivity might be useful for glaucoma screening. Our results cannot be extrapolated to the entire age group at risk, in which contrast sensitivity losses occur for a variety of reasons.

One way of improving the discrimination would be to test separately in the central and peripheral retina. It should be noted that for central losses our results are less impressive than, for example, Atkin *et al* found.<sup>19</sup> This suggests that our estimate of the upper limit of normal was if anything too high, and the discrimination attainable between normals and OH and glaucoma in the peripheral retina is the more interesting. Thus it is likely that measurements in the retinal periphery might reliably detect (and quantify) retinal damage which precedes field loss.

In order to make screening practicable, our

Table 3 Analysis of results in 20 patients with high risk OH

	Abnormal CS*	Normal CS
Normal visual field	1	10
Suspect visual field	8	1
Total	9	11

\*Contract sensitivity. Values of CS are the mean of four quadrants at 25°. Fisher exact,  $p=0.000238$ .

technique requires modification. The prime need is to increase the speed of the test and to use forced-choice methods to minimise the subjective elements. In addition it appears that tritan colour vision losses are more prominent than luminance losses,<sup>12</sup> and this is the case especially for the retinal periphery.<sup>24</sup> Once modifications in technique along such lines have been tested, an alternative to prolonged field testing seems possible.

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