PATTERNS OF EARLY VISUAL FIELD LOSS IN OPEN-ANGLE GLAUCOMA*

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INTRODUCTION

PATTERNS OF VISUAL FIELD LOSS IN LOW-TENSION GLAUCOMA AND IN HIGH-TENSION glaucoma have been studied.[1-8] Some investigators have found significant differences in patterns of field loss between the two groups, while others have found no differences. Variations in the criteria used for patient selection make direct comparisons between studies difficult. Stages of the disease and rates of progression are frequently not comparable. Most importantly, the criterion of intraocular pressure (IOP) probably does not adequately separate subpopulations of glaucoma patients. The terms "low tension" and "high tension" imply that IOP differences alone may adequately separate these two "entities,"[9,10] but the semantics are not reassuring. Functional definitions are required.

In an effort to supply additional useful information in this area, we have taken a novel approach. We examined certain characteristics of eyes of patients with open-angle glaucoma, differentiated on the basis of visual field findings alone. The criteria for visual field abnormalities were designed to separate patients whose eyes showed diffuse depression of the visual field, from patients whose eyes showed dense, localized scotomas.

METHODS

The screening criteria for selecting an initial study population are presented in Table I. These criteria were designed to reduce the possibility of including eyes with diffuse depression due to nonspecific causes, such as uncorrected refractive error, cataract, and miosis. Patients were selected from the records of the Yale Glaucoma Service and carried a diagnosis of primary open-angle glaucoma or low-tension glaucoma and also had automated static threshold visual fields (Octopus) during the last 5 years. The screening of records was performed by the technical staff at

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the Yale Glaucoma Service. The investigators remained masked as to the identity of the patients selected. Sixty-three eyes of 59 patients met the initial screening criteria. Of patients who qualified bilaterally, one eye was chosen for inclusion into the study by the toss of a coin.

Visual field indices introduced by Flammer et al[11] were used to select eyes with a relatively pure form of diffuse visual field loss and those with a relatively pure form of localized dense loss. Mean defect (MD) is the average sensitivity loss compared to age-matched normals of all points tested. While its value may be affected by any visual field abnormality, it is increased relatively little by localized scotomas but is increased greatly by diffuse depression of sensitivity. The statistical variance of sensitivity loss, compared to normal values for all points tested is termed "loss variance" (LV), and includes a component of variance over time (short-term fluctuation [SF]). Corrected loss variance (CLV) is calculated from LV by subtracting out SF. SF can be estimated by measuring the threshold of the same test location multiple times, over the course of the test. CLV (and to a lesser extent, LV) is a measure of scatter or local non-uniformity of visual field thresholds. It is not affected by uniform diffuse depression of sensitivity, but is increased by localized defects. Thus, MD and CLV are visual field indices which are sensitive to different types of loss: MD is sensitive to diffuse depression, while CLV is sensitive to localized defects.

MD and LV were calculated from each visual field (program 32), after eliminating the most peripheral loci of test locations and the three loci normally containing the blind spot. LV was used rather than CLV, since an estimate of scatter over time (SF) is limited with program 32, which tests only ten locations twice. However, selection criteria demanded that the root mean square fluctuation (a measure of SF) be ≤ 3 decibels. Correction of LV for SF (ie, the use of CLV) would not have altered the selection of patients for study. Values for MD and LV for all 59 eyes in the study population, were plotted on a scattergram (Fig 1). Arbitrary definitions were made to select patients with predominantly diffuse loss (MD > 3 decibels, LV < 10 decibels) and localized scotomas (MD ≤ 3 decibels, LV ≥ 20 decibels). These definitions identified eight eyes with diffuse field loss, and seven eyes with localized scotomas.

Information regarding each of the patients whose eyes were selected for final analysis was obtained from our patient records and from referring physicians, where appropriate. This included medical history, surgical history, ocular history, ocular medications, blood pressure, IOP, and visual field data. This information was compiled without the investigators' knowledge of the patient's identity, or knowledge as to which visual field

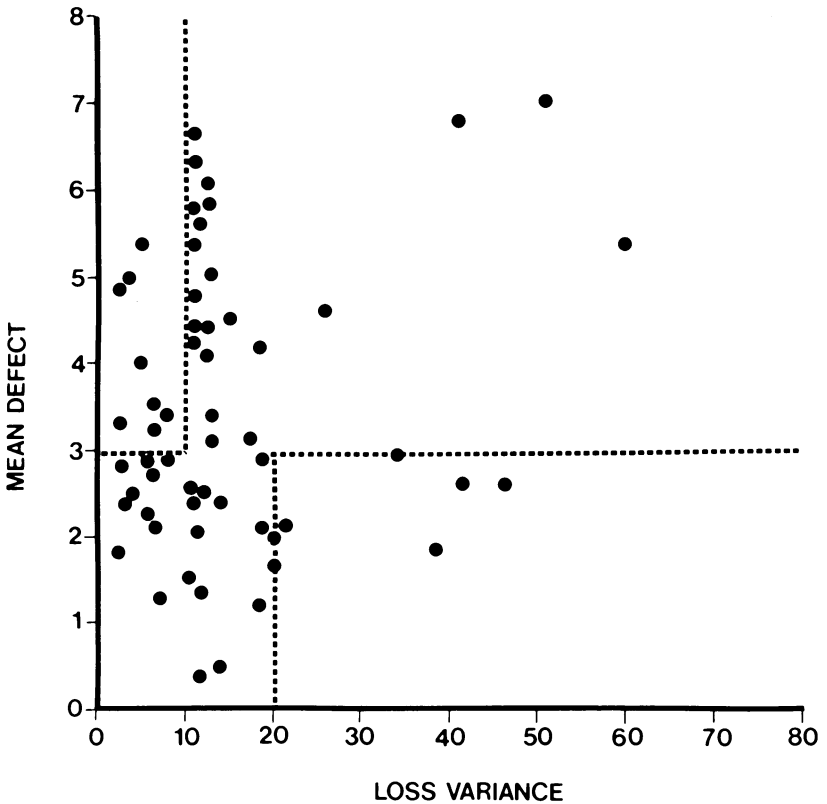

FIGURE 1
MD (decibels) versus LV (decibels) in 59 eyes meeting the selection criteria. *Dotted lines* represent criteria for MD and LV, designed to identify two groups of patients with relatively pure types of visual field loss.

group the patient belonged.

It is impossible to compute a single number which adequately describes an eye's IOP history. Therefore, we took several approaches. The mean, median, high, and low values of recorded pressures were identified for each eye. In addition, eyes were assigned to IOP categories in a masked fashion as follows: eyes with recorded pressures frequently above 25 mm Hg were included in a "high" IOP group; eyes with pressures frequently above 20 mm Hg but infrequently above 25 mm Hg, were assigned to an "intermediate" IOP group; and eyes with IOP infrequently above 20 mm Hg were assigned to a "low" IOP group. The optic disc of each patient was analyzed from stereoscopic color photographs taken on the same day the visual field was obtained. While viewing a photographic

TABLE I: CRITERIA FOR SELECTION

Diagnosis	Primary open-angle glaucoma or low-tension glaucoma
History	No eye surgery No other eye disease No other reason for visual field loss
Age	50–80 years
Refractive error	≤ 3 diopters ammetropia ≤ 2 diopters astigmatism
Visual acuity	≥ 0.67
Visual field	Total loss 100 to 300 decibels Root mean square ≤ 3.00 decibels Rate of false (+) and false (−) ≤ 10% Pupil size ≥ 3.0 mm

pair stereoscopically, the disc rim and cup rim were traced on a projected image for each eye using contour clues only; measurements of rim width, normalized to the disc diameter, were made along eight meridians.

Data were analyzed with the help of Student's *t*-test and chi-square testing, with a significance level of $P < 0.05$.

RESULTS

The diffuse loss group included four male and four female patients, and the localized loss group included two males and five females. Two patients in the diffuse group and three patients in the localized group had been treated for systemic hypertension. One patient in the diffuse group was receiving systemic beta blockers, while three patients in the localized group were similarly treated. One patient had a history of significant systemic hypotension and belonged to the localized group. All patients in the diffuse group carried a clinical diagnosis of primary open-angle glaucoma, while four of the seven patients in the localized group carried the diagnosis of low-tension glaucoma. Three patients in the diffuse group had a family history of glaucoma, while two patients in the localized group had such a history.

Data regarding age, visual acuity, refractive error, pupil size, number of eye medications, and visual field are given (Table II). There were statistically significant differences between visual field MD and LV, due to patient selection criteria. There was no difference in the root mean square value betwen the two groups, nor was there a statistically significant difference in total loss.

IOP data are given in Tables III and IV. The diffuse group manifested significantly higher pressures than the localized group, regardless of the

TABLE II: CLINICAL DATA (MEAN ± SEM)

	DIFFUSE (n = 8)	LOCALIZED (n = 7)
Age (years)	68.0 ± 3.3	65.4 ± 3.3
Visual acuity	0.81 ± 0.05	0.89 ± 0.05
Refractive error (spherical equivalent, diopters)	0.9 ± 0.6	0.9 ± 0.4
Pupil size (mm)	3.5 ± 0.3	4.0 ± 0.5
Eye medications	2.0 ± 0.3	1.5 ± 0.3
Visual field (decibels)		
Mean defect	4.0 ± 0.3	2.1 ± 0.2
Loss variance	5.5 ± 0.8	29.5 ± 4.0
Root mean square	2.0 ± 0.2	1.9 ± 0.2
Total loss	211 ± 15	166 ± 24

TABLE III: INTRAOCULAR PRESSURE (mm Hg, MEAN ± SEM)

	DIFFUSE (n = 8)	LOCALIZED (n = 7)	
Mean	21.9 ± 0.9	17.6 ± 0.8	$P < 0.01$
Median	22.0 ± 0.9	17.7 ± 0.6	$P < 0.01$
High	27.6 ± 1.2	22.4 ± 1.4	$P < 0.02$
Low	17.0 ± 1.0	13.3 ± 0.8	$P < 0.05$

TABLE IV: IOP CATEGORIES*

	DIFFUSE	LOCALIZED
High	3	0
Intermediate	5	2
Low	0	5

$P < 0.01$.
*See text for criteria.

TABLE V: RIM/DISC RATIO (MEAN ± SEM)

POSITION*	DIFFUSE (n = 8)	LOCALIZED (n = 7)	
12:00	0.13 ± 0.03	0.10 ± 0.02	
10:30	0.18 ± 0.02	0.07 ± 0.02	$P < 0.01$
9:00	0.15 ± 0.02	0.06 ± 0.03	$P < 0.05$
7:30	0.16 ± 0.01	0.05 ± 0.03	$P < 0.01$
6:00	0.20 ± 0.01	0.12 ± 0.08	
4:30	0.22 ± 0.02	0.16 ± 0.01	$P < 0.02$
3:00	0.22 ± 0.02	0.18 ± 0.02	
1:30	0.20 ± 0.01	0.19 ± 0.02	
Mean	0.19 ± 0.01	0.11 ± 0.02	$P < 0.05$

*The clock-hour position is standardized to the right eye (ie, 9 o'clock is temporal).

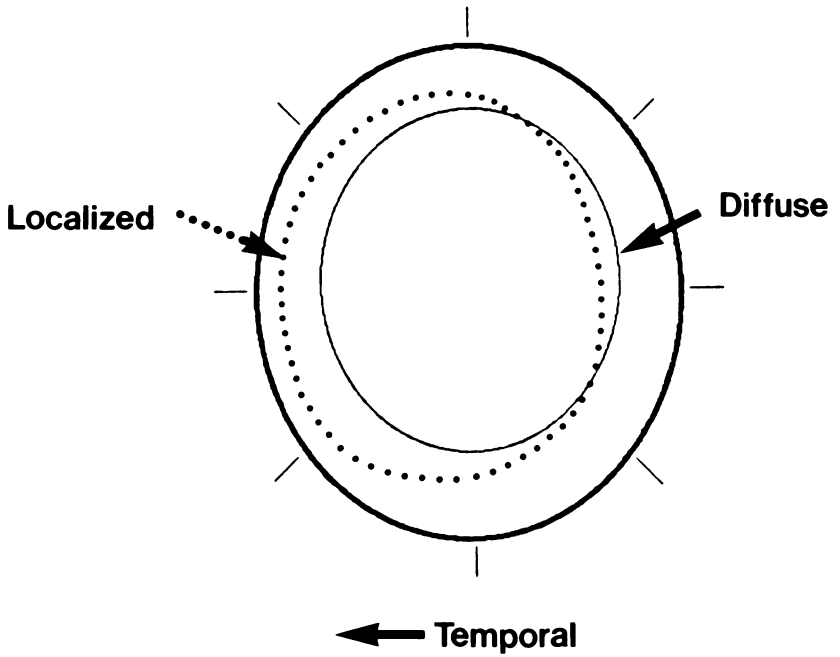

FIGURE 2
Diagrammatic representation of mean optic nerve rim width in patients with diffuse visual field loss, and in patients with localized visual field loss. There are significant differences in rim width temporally (Table V).

statistic used. That is, the diffuse group had higher mean and median pressures, and had higher extremes of IOP than did the localized group. The difference in IOP values between the two groups ranged from 3 to 5 mm Hg. Table IV summarizes the IOP categories assigned to eyes of the two groups. The diffuse group was skewed toward the high range of pressure, while the localized group was skewed toward the low range of pressure. However, overlap between the two groups is apparent.

Analysis of stereoscopic photographs of the optic nerve head revealed the disc rim to be significantly thinner in the localized group compared to the diffuse group. The largest differences were noted in the temporal area of the rim (Table V). The mean position of the cup edge in each of the two groups is demonstrated graphically (Fig 2). The cup is larger and shifted eccentrically toward the temporal and inferotemporal disc rim in the localized group compared to the diffuse group.

DISCUSSION

Several investigators have searched for differences in patterns of visual field loss, in patients with low-tension glaucoma compared with high-tension glaucoma.[1-8] While some have found no differences in types of visual field loss between the two groups,[1,2,8] others have found significant differences.[3-7] Varying patient selection criteria probably accounts for the apparent differences in these results. Clearly, IOP criteria alone do not adequately subdivide groups of patients, unless extremes of IOP are used to divide patients. Then clear patterns can emerge.[7,9] Recently, King et al[8] published a study examining the distance from fixation of scotomas, in patients with normotension and high-tension glaucoma. Although pressure criteria were more widely separated than some of the previous studies, no differences in visual field patterns between the two groups were found. However, it was not clear that the two groups of patients were in the same stage of the disease nor was it clear at what rates they reached this stage. Cup/disc ratio was used as an indicator of the stage of the disease; no information was given regarding total visual field loss. Optic disc cupping may not be an appropriate way to compare different groups of patients, since we have no idea of the previous size of the cup in each patient, and the pattern of optic disc cupping may be different in patients with high-tension and low-tension glaucoma.[9]

Difference in IOP is probably not the best way of subdividing patients with open-angle glaucoma. Because of the controversy in this area, we took a new approach to examine the problem. Eyes were assigned solely on the basis of visual field criteria, in an effort to study eyes with a relatively pure type of diffuse visual field loss, contrasted to those with a relatively pure type of localized loss. Pure examples are difficult to find so the number of subjects in this initial study are small. Nevertheless, different patterns appear to emerge between these groups, with respect to IOP and optic nerve cupping. The selection criteria were designed to minimize the problem created by eyes included in the diffuse loss group, because of nonglaucomatous processes such as cataract, miosis, or refractive error. Selection criteria also included standards for patient reliability and placed a limit on the magnitude of SF, further reducing the number of eyes included.

Although considerable overlap was evident between the groups regarding IOP levels, the pressures were consistently higher in the diffuse group as compared to the localized group, whether analyzed by IOP mean, median, high values, or low values. A better way of summarizing IOP history may be to place eyes into IOP categories in a masked fashion. The diffuse group contained more eyes in the high IOP category, while

the localized group contained more eyes in the low IOP category. Still, there was some overlap in the intermediate pressure category.

It is important to emphasize that all eyes included in this study were in an early phase of glaucomatous optic nerve damage and were matched for equal amounts of total visual field loss. There are no "normal" values for optic disc cupping, but there are "normal" values for visual field thresholds. Hence, we believe it is rational to match patients for stage of the disease by using visual field criteria. In eyes matched in this way, it is evident that the pattern of optic disc cupping in patients with localized loss is different from that of patients with diffuse loss. The diffuse loss eyes have round optic nerve cups, which are central and have a disc rim, which is intact and uniform in thickness. By contrast, eyes in the localized loss group have optic discs with significantly more cupping occurring eccentrically with shallow thinning of the disc rim temporally and inferotemporally.

We hypothesize, based on this preliminary information and pending further work, that diffuse loss of visual field sensitivity from glaucoma is largely pressure dependent and may be secondary to diffuse axonal dysfunction (and later death), leading to progressive concentric enlargement of the optic nerve cup. In the localized loss group, visual field loss seems less pressure dependent. Here, loss may occur at low pressures as well as high pressures. The damage to the optic nerve head tends to be less generalized, leading to focalized loss of the disc rim temporally. It is tempting to speculate that pressure-dependent gradual diffuse loss may involve a prolonged phase of ganglion cell dysfunction without death, leading to visual abnormalities with relatively small amounts of progressive cupping. This could explain the presence of psychophysical disturbances preceding detectable morphologic change. On the other hand, the localized loss group may be associated with accelerated ganglion cell death, leading to thinning of the disc rim and dense scotomas in a relatively "early" phase of the disease.

The eyes selected for this study represent examples of relatively pure types of visual field loss. It is apparent that the majority of patients with glaucomatous optic nerve damage have a mixed type of visual field loss, with both diffuse depression and scotomas occurring in the same field. Diffuse loss may precede scotomas in many patients, and at an advanced stage of the disease, patterns of visual field loss may become indistinguishable. This study represents a beginning in terms of identifying possible subsets of patients with glaucoma and quantifying the abnormalities within these subsets.

REFERENCES

1. Phelps CD, Hayreh SS, Montague PR: Visual fields in low-tension glaucoma, primary open angle glaucoma, and anterior ischemic optic neuropathy, in EL Greve, A Heijl (eds): *Fifth International Visual Field Symposium*. The Hague, Dr W. Junk Publishers, 1983, pp 113-124.
2. Motolko M, Drance SM, Douglas GR: The visual field defects of low-tension glaucoma: A comparison of the visual defects in low-tension glaucoma with chronic open-angle glaucoma, in EL Greve, A Heijl (eds): *Fifth International Visual Field Symposium*. The Hague, Dr W. Junk Publishers, 1983, pp 107-112.
3. Primrose J: Clinical review of glaucomatous discs, in JS Cant (ed): *The Optic Nerve*. St Louis, CV Mosby, 1972, p 314.
4. Levene RZ: Low-tension glaucoma: A critical review and new material. *Surv Ophthalmol* 1980; 24:621.
5. Anderton S, Hitchings RA: A comparative study of visual fields of patients with low-tension glaucoma and those with chronic simple glaucoma, in EL Greve, A Heijl (eds): *Fifth International Visual Field Symposium*. The Hague, Dr W. Junk Publishers, 1983, pp 97-99.
6. Greve EL, Geijssen C: Comparison of glaucomatous visual field defects in patients with high and with low intraocular pressures, in EL Greve, A Heijl (eds): *Fifth International Visual Field Symposium*. The Hague, Dr W. Junk Publishers, 1983, pp 101-105.
7. Caprioli J, Spaeth GL: Comparison of visual field defects in the low-tension glaucomas with those in the high-tension glaucomas. *Am J Ophthalmol* 1984; 97-730-737.
8. King D, Drance SM, Douglas G, et al: Comparison of visual field defects in normal-tension glaucoma and high-tension glaucoma. *Am J Ophthalmol* 1986; 101:204-207.
9. Caprioli J, Spaeth GL: Comparison of the optic nerve head in high- and low-tension glaucoma. *Arch Ophthalmol* 1985; 103:1145-1149.
10. Drance SM: Low-tension glaucoma. *Arch Ophthalmol* 1985; 103:1131-1133.
11. Flammer J, Drance SM, Augustiny L, et al: Quantification of glaucomatous visual field defects with automated perimetry. *Invest Ophthalmol Vis Sci* 1985; 26:176-181.

DISCUSSION

Dr Allan E. Kolker. In 1984, Caprioli and Spaeth evaluated patterns of visual loss in patients with glaucoma, to determine if those with low-tension glaucoma (LTG) had differences in field loss, from those with classic open-angle glaucoma associated with elevated intraocular pressure—ie, high-tension glaucoma (HTG). By selecting patients with substantial differences in intraocular pressure, they noted that cases of LTG had scotomas closer to fixation, of greater depth, and of steep slope, compared with those having HTG. They concluded that these differences in patterns of field loss, implied a different pathogenesis of optic nerve damage, in eyes with HTG and LTG. They postulated that LTG might result from an ischemic optic neuropathy, whereas classic HTG might be the result of mechanical compression of the lamina cribosa by the elevated intraocular pressure.

In the present study the authors correctly point out that separation into LTG and HTG by an arbitrary level of intraocular pressure, is unreasonable, if not impossible. They therefore approach the topic from the opposite side. Using highly sophisticated techniques, available only with computerized automated static perimetry, they separate glaucoma patients by different patterns of visual field loss and then analyze the levels of intraocular pressure at which these patterns of optic nerve damage developed. This is a unique twist in getting at the

question of different mechanisms of damage, if any, between subjects having glaucomatous visual field loss at relatively low or high intraocular pressures. The authors are to be congratulated on their approach.

The separation into types of field loss was made using indices of visual damage. The mean defect (MD) value corresponds to a diffuse depression of retinal sensitivity, whereas the corrected loss variance (CLV) is a measure of nonuniformity of visual thresholds and corresponds to localized defects. Using cases of glaucoma having relatively pure types of field loss, they were able to show that patients having diffuse visual field loss had consistently higher intraocular pressures than those with localized loss. The diffuse loss patients also demonstrated generalized enlargement of the optic cup, compared to the localized loss cases, who had an eccentric cup with localized thinning of the disc rim tissue.

Several basic questions arise from the study: (1) Is the pattern of visual damage different in cases of LTG and HTG? The present data suggests that this may be the case. (2) Do the different patterns of visual field loss indicate different mechanisms of damage—ie, vascular *vs* mechanical? (3) If different mechanisms of damage exist, how do the various measures of visual function, associated with glaucoma, correlate with the type of damage? For example, which psychophysical tests correlate more with diffuse or localized damage? Are bundle or sector defects in the retinal nerve fiber layer seen more frequently in LTG than in HTG? (4) Do individual patients with glaucoma often have more than one mechanism of optic nerve damage and how does this influence therapy or prognosis?

Finally, and this is my only real criticism of the paper, the study was done by selecting patients with open-angle glaucoma, separating them by their visual field findings, and then retrospectively reviewing what their intraocular pressure patterns had been. The initial selection is potentially biased, because patients with low pressures and only diffuse visual field depression might not have been picked up and included in the population of patients for this study. The findings of the study could therefore be a result, solely, of this selection bias.

Were all patients on glaucoma therapy at the time of analysis of intraocular pressure, and did the amount of such therapy correlate with the level of pressure? Could the authors detect differences in the rate of progression of field loss between cases with diffuse damage, compared with those having localized damage?

I want to thank the authors for allowing me to review this fascinating and provactive paper.

Dr Robert Drews. One question. Doctor Caprioli talks about things like mean defect and loss variance and demonstrated these to us on gray scale printouts. The gray scale output of the Octopus perimeter represents data which has already been massaged by the computer within the perimeter, filling in the areas between the test spots using statistical means. I wonder whether the calculations of loss variance were based on the gray scale output or on the raw data?

Dr Alfred Sommer. This is a fascinating paper and I look forward to having the opportunity to read it in all its glorious detail. I'm therefore hesitant about making

general comments at this time. I would however, like to inject some specific remarks about automated visual field testing and interpretation, because a number of other manuscripts have come my way based on the Octopus computer. One problem is continued reference to built-in values for "age-matched normal controls." I think many in the audience recognize the Octopus contains no such thing. The "normal" values represent results of repeated testing of a small number of young, healthy Swiss citizens with good visual acuity to begin with. The age "correction" simply assumes a fixed degree of change for every 10 years of age—not a collection of normal controls at these ages. I understand that the people involved with the Octopus computer are now collecting a sample of normal control of various age, just as the people involved with the Humphrey and other instruments are doing. These however are not yet available.

A second factor, unique to the Octopus computer, is the very low level of background illumination under which testing is done. A recent paper by Radius demonstrated that very small reductions in illumination of the test target, as one might get in early or subclinical cataract among people with still normal vision, can degrade threshold measurements considerably. It is not yet known whether testing with other instruments, with higher background illumination would suffer the same fate. As this is likely to effect diffuse more than localized depression, we need to be particularly cautious in attributing generalized depression to a pathologic process.

Dr Thomas Duane. I find this to be a very interesting paper. I think the authors have outlined the critical factors that have to do with field change. However, there are some aspects of the field change that I don't think were covered and I wonder what their opinion is regarding these. The clinical critical factor, particularly in low-tension glaucoma, is the perfusion that results from the heart to the eye levels. Theoretically, if you took the vision or tested the visual field or did any other subjective study, it very well could be different if one tested in a supine as opposed to an erect position. We wouldn't expect there to be any difference between an erect standing position and an erect sitting position because the distance from the heart to the eye is the same. I would suspect that if you placed the patient (horizontally) suprine, and tested for these subtle arcuate scotomata and localized them that we would find them more in the erect position than in the horizontal.

Dr Jonathan D. Wirtschafter. I am concerned that the size of the group that constituted the diffuse visual field loss was reduced by the selection of a subgroup of patients with small root mean square (RMS) values. We know that in areas where the retina is abnormal, you would, in fact, get a high RMS value. It follows that many patients with diffuse abnormalities of the tested points and with high RMS values would have been selected out of the diffuse visual field loss group. Thus the subgroup of patients with high RMS values might have had very different intraocular pressures than the subgroup with low RMS values. The use of RMS values as a selection criteria raises a specific question that we might address

to the authors: Was any effort made to select RMS values in areas of the visual field where the patient threshold was near the age corrected normal or was the diffuse RMS value of the entire visual field used? This could have a very important effect on the size and composition of the groups. I thank the authors for this interesting presentation.

DR JOSEPH CAPRIOLI. I thank the discussants for their thoughtful and most useful comments. Doctor Kolker has indicated that different patterns of visual field loss may be linked to different mechanisms of glaucomatous optic nerve damage. It is certainly attractive to speculate that localized loss is a result of an ischemic event or infarct, and that diffuse depression is the result of mechanical compression or posterior deformation of the lamina cribrosa. While our preliminary results are compatible with such an hypothesis, they cannot be considered proof. The data are consistent, however, with the occurrence of more than one type of optic nerve damage in glaucoma. The scattergram of mean defect (MD) versus loss variance (LV) indicates that most of the eyes meeting the screening criteria display a mixed type of visual field loss. We selected and analyzed only the relatively pure types of localized and diffuse visual field loss. The fact that most patients have mixed patterns of loss is consistent with the notion that there is commonly more than one cause of optic nerve damage in glaucoma.

Doctor Kolker raised the issue of bias. Bias is certainly an important consideration in any clinical study of this type. Doctor Kolker was concerned about slanting the population toward high-tension glaucoma (which would be more easily diagnosed), and away from low-tension glaucoma (which might not be diagnosed if no visual field test were performed). It is difficult, in fact impossible, to determine exactly what effect bias might had had on our results. However, it would not change the finding that two distinct types or patterns of field loss exist. Randomized, prospective longitudinal studies currently planned may help answer this important question. In the meantime, we would ask each of you to examine your patient populations in an effort to identify such patterns of visual field loss.

Doctor Kolker also asked about the role of medications in this study. The majority of the measured pressures were recorded while patients were under treatment. The mean number of medications per patient was similar in both groups of subjects, as was pupil size.

Doctor Kolker's final remark concerned rates of progression of optic nerve damage. We addressed this excellent point in the manuscript. It is very difficult to match glaucoma patients for extent of optic nerve damage. One way to do this is to match optic nerve cupping. We don't believe this to be the best way, since we don't know in each eye what the initial cup size was, since there is a large variability of "normal" cup sizes. We believe a better way is to use the visual fields. It is possible to construct a definition of a normal visual field and to match visual field loss in glaucoma patients to indicate similar extents of disease. It is, of course, important to eliminate the nonspecific causes of visual field loss when doing this. In the cross sectional study presented here, we did not match patients for rate of progression. However, in our longitudinal studies, it should be possible

to do that.

Doctor Drews asked about the role of the gray scale display of the visual fields in our studies. I simply presented slides of gray scales to give the audience a clear, graphical idea of the appearance of the fields. Gray scales were not used and are never used in the actual data analysis.

One of Doctor Sommer's concerns was that normal values for visual thresholds stored by the Octopus computer were generated by a small group of normal Swiss subjects, and may not apply to normals in a clinical population. In a very recent issue of the American Journal of Ophthalmology, Haas et al published a study analyzing a large number of normal patients using the Octopus computer (*Am J Ophthalmol* 1986; 101:199-203). Their results for normal threshold values compared favorably to the stored thresholds in the Octopus perimeter. Therefore, I have no misgivings about using the stored values in constructing a normal visual field. Doctor Sommer also raised the issue of how the "low" level background illumination used by the Octopus computer might affect the results. The background illumination of the Octopus is 4 apostilbs compared to 31.5 apostilbs on the Goldmann or Humphrey perimeters. Recent studies by Heuer et al (*Invest Ophthalmol Vis Sci* [Suppl] 1985; 26:40) and by Klewin and Radius (*Arch Ophthalmol* 1986; 104:395-397) have indicated a nonlinearity of Weber's Law at low level background illumination, such as with the Octopus perimeter. These studies imply that even an early cataract or mild miosis can produce diffuse depression of the visual field. Instead, Rose de Vries' Law $(\Delta L/L)^{1/2}$ holds at both 4 and 31.5 apostilbs (*Doc Ophthalmol* 1979; 47:89-138). Our study specifically excludes patients with levels of cataract sufficient to produce visual acuities worse than 20/30, and excludes patients with pupil sizes < 3.0 mm. It has long been recognized that media opacity and/or pupillary miosis will affect measurements of the visual field, but also that Weber's law is not linear at background illumination levels of < 100 apostilbs. The same argument would therefore also apply to the Humphrey and Goldmann perimeters.

Doctor Duane brought up the issue of perfusion of the optic nerve head. This is absolutely correct. We must develop methods to assess perfusion of the nerve head in the diagnosis and treatment of this disease.

Doctor Wirtschafter appropriately cautioned against the use of root mean square (RMS) values to segregate patients. Using program 32 on the Octopus perimeter, only ten points are tested twice. This is the reason we used loss variance rather than corrected loss variance as the appropriate statistic, and set an upper limit for RMS as an exclusion criteria, one component of which is short-term fluctuation.

I thank all the discussants, especially Doctor Kolker, for their efforts. We would urge all of you to categorize field loss in your glaucomatous patients to see if multiple patterns emerge.