Upsala J Med Sci 97: 251–260

# Visual Impairment of Open Angle Glaucomas at First Presentation and After a Five to Ten Year Follow-up

Lennart Berggren and Ingmar Widengård
*Department of Ophthalmology, University of Uppsala, Sweden*

## ABSTRACT

Of all patients attended to in a clinic during 1986, 441 had open angle glaucoma diagnosed during 1974-1986 on the basis of either a verified visual field defect, a glaucomatous disc, or a repeated intraocular pressure value of at least 35 mm Hg.

At first presentation of recent cases 1984-1986 (N=128) 65 per cent were more than 70 years old. Capsular glaucomas were twice as common as simple glaucomas (low tension cases included).

62 per cent of capsular but only 26 per cent of simple glaucomas had an initial pressure of 35 mm Hg or more ($p<0.001$). One third of both capsular and simple glaucomas had an advanced visual field defect with breakthrough to the periphery in the worse eye already at first presentation. This was more common if the initial pressure was 35 mm Hg or more ($p<0.05$).

Almost half remained unilateral cases, and the rate of severely impaired visual function in the better eye did not exceed 15 per cent.

While generally 30-50 per cent of glaucomatous field defects had progressed in five years, the progression in early detected cases was only three per cent ($p<0.05$). Visual field defects with breakthrough to the periphery already at first presentation progressed more often than circumscribed scotomas ($p<0.02$).

## INTRODUCTION

Retrospective and prospective glaucoma studies often suffer from unavoidable shortcomings. The course of glaucoma might be difficult to establish because cataract, macular degeneration and other age related factors disturb careful examinations (7,9,12,14). It has been shown that 30-40 per cent of optic nerve axons might be lost before a visual field defect can be detected (33,34,35). The evaluation of therapeutic effects can often be questioned, because compliance to medical therapy is unknown and often poor (17,18,29).

We report here the results of an investigation of all open angle glaucomas attended to at an university eye clinic during 1986. Signs of pronounced changes in optic discs and visual fields has been used to classify glaucoma damage. Correlation of initial intraocular pressure, type of

glaucoma and visual field defect has been analysed. Therapeutic outcome is discussed only in terms of visual function.

## SUBJECTS AND METHODS

The Department of Ophthalmology, University Hospital, Uppsala attends to about 60 per cent of the glaucoma out-patients and all in-patients in a district with 250 000 inhabitants. During 1986 1256 patients with a clinical diagnosis of glaucoma or suspected glaucoma were seen. Two thirds of these patients had open angle glaucomas (capsular 475, simple 351, low tension 21), and 177 had ocular hypertension or suspected (untreated) glaucoma. 57 had narrow angle glaucoma, 8 congenital, 6 pigmentary and 151 secondary glaucoma.

### Inclusion criteria and grouping of patients

Visual impairment and intraocular pressures have been analysed only for those patients with verified open angle glaucoma, who had no glaucoma treatment when first seen in the clinic.

Criteria for verified glaucoma were either a glaucomatous visual field defect (stage 1-4 as defined below) detected at at least two examinations, or a glaucomatous disc (stage 3-4), or repeated pressure values of at least 35 mm Hg. The patients were subgrouped according to duration of verified disease.

In the first three subgroups the patients fulfilled our diagnostic criteria at their first presentation:

Recent glaucomas. - First presentation 1984-1986. Duration of disease $1 \pm 1$ years. Number of cases 128.

5-year-glaucomas. - First presentation 1979-1983. Duration of disease $5 \pm 2$ years. Number of cases 174.

10-year-glaucomas. - First presentation 1974 1978. Duration of disease $10 \pm 2$ years. Number of cases 101.

In the following two subgroups the patients did not fulfil our criteria at first presentation. The first group was treated for glaucoma before the verified diagnosis according to our criteria. The second was followed because of ocular hypertension or other risk factors but had no treatment before the verified diagnosis:

Treated before verified diagnosis. - visual field defect developed 1979-1983. Duration of disease $5 \pm 2$ years. Number of cases 20.

Followed before verified diagnosis. - visual field defect developed 1979-1983. Duration of disease $5 \pm 2$ years. Number of cases 18.

Of these 441 patients 293 had capsular, 131 had simple and 17 had low tension glaucoma.

### Examination

Intraocular applanation pressure value - was recorded at first presentation as the mean of a diurnal curve (3-7 values).

Optic disc appearance - was analysed from stereo-diapositives or from a sketch based on direct, binocular examination. Stages:
0 - normal disc appearance,
1 - asymmetry between eyes or vertical elongation of cup,
2 - total polar notch up or down, or cup/disc=0.8,
3 - total polar notch up and down, or cup/disc=0.9,
4 - total excavation to the edge,
X - evaluation not possible, usually because of cataract.

Visual field. - Computer assisted threshold static perimetry (Competer) was used when possible. Patients with large defects and those otherwise unable to co-operate with the Competer were examined with the Goldmann perimeter using a combined kinetic and static technique. Stages:
0 - normal visual field,
1 - 1-2 paracentral scotomas or a nasal step,
2 - fully developed arcuate scotoma,
3 - breakthrough to the periphery and loss of 1/3-2/3 of the total field,
4 - central vision with or without a temporal rest, or blindness,
X - cataract, macular degeneration or co-operation made examination impossible.

Eye handicap refers to a particular eye, while patient handicap is determined by the condition of the better eye. A visual field defect stage 2 or less was considered as not handicapping.

Worse eye is defined as the eye with the highest stage of visual field defect or, if this does not differ, the eye with the highest stage of optic disc damage. If this could not separate the eyes, the right eye has been chosen.

Progression is defined as passing from one visual field stage to another.

Chi-squares have been used for the calculations of statistical significance.

## RESULTS

Age distribution - all cases. The average patient was old. The age mean $\pm$ SD and median for all cases controlled and treated in 1986 were 76 $\pm$ 8 and 76 years for capsular glaucomas (N=475), 73 $\pm$ 11 and 74 years for simple and low tension glaucomas (N=372).

First presentation. These analyses have been restricted to the subgroup "Recent glaucomas" in order to lessen the influence of mortality.

At first presentation two thirds of the patients were 70 years old or more, and every fifth was 80 or more (table 1). Thus the majority of patients with verified glaucoma were old already at first presentation.

Table 1. Age at first presentation 1984-1986 of open angle glaucoma.

| | All cases N=128 | Capsular glauc. N=85 | Simple + low tension glauc. N=43 |
|---|---|---|---|
| Age years | | | |
| 70 or more | 65% | 67% | 60% |
| 75 or more | 39% | 40% | 37% |
| 80 or more | 19% | 19% | 19% |
| Age mean ± SD | 71.9 ± 8.9 | 72.9 ± 8.0 | 70.0 ± 10.2 |
| Age median | 73 | 74 | 71 |

Capsular glaucomas were twice as common as simple and low tension glaucomas. Intraocular pressure and visual field defect in the worse eye are presented in fig 1.

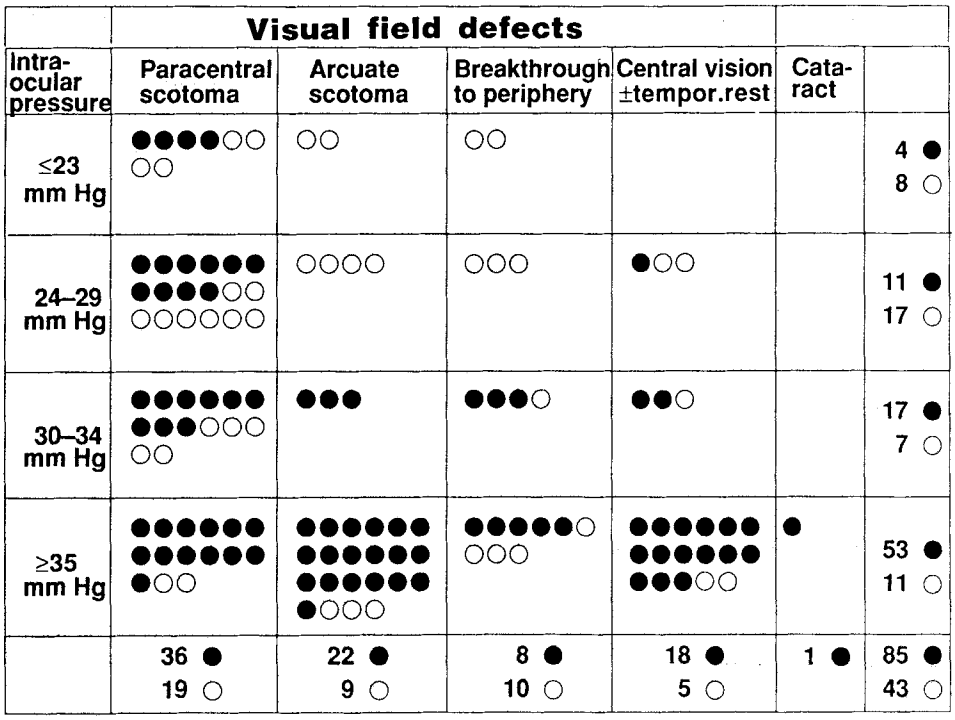


Fig. 1.
Glaucomatous visual field defects and intraocular pressure levels in the worse eye of capsular (●), and simple + low tension (o) glaucomas with a first presentation 1984-1986. In the group ≥ 35 mm Hg/paracentral scotoma 5 capsular glaucomas had high pressure only.

High intraocular pressures were common, 50 per cent (64/128) had a pressure ≥ 35 mm Hg. This was more pronounced among capsular glaucomas (62 per cent or 53/85) than for simple glaucomas (26 per cent or 11/43). This difference is statistically significant ($p < 0.001$).

One third (41/128) had a handicapping field defect (stage 3 or 4) in the worse eye. Capsular and simple glaucomas did not differ in frequency of handicapping field defects.

The mean age of patients with a visual field defect stage 4 was 5 years higher than the mean age of the whole group (71.9 $\pm$ 8.9 and 76.7 $\pm$ 8.6 respectively).

A handicapping field defect was found in 41 per cent (26/64) of worse eyes with a pressure of 35 mm Hg or more at first presentation. In worse eyes with a lower pressure the rate was only 23 per cent (15/64). The difference is statistically significant ($p < 0.05$).

While 34 per cent of recent glaucomas had a handicapping defect in one eye already at first presentation, the other eye showed a handicapping defect in only 6 per cent (fig 2). 45 per cent of better eyes showed no sign of glaucoma.

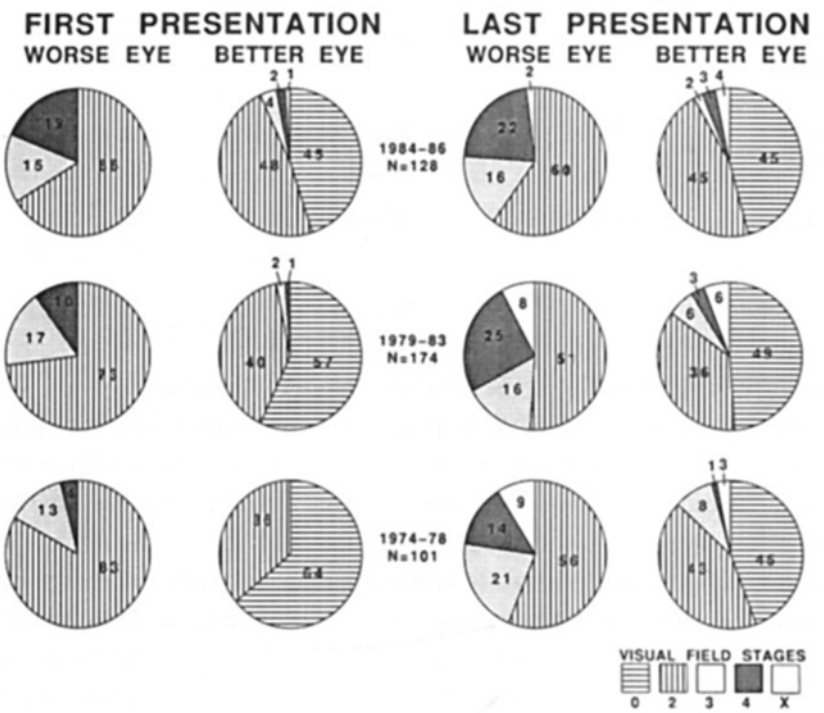


Fig. 2.
Percentage distribution of glaucomatous visual field defect stages 0-1, 2, 3, 4 or X. The diagrams show the condition for worse and better eye at first and last presentation for the subgroups Recent glaucomas, 5-year-glaucomas and 10-year-glaucomas.

Patient handicap. At first presentation a patient handicap (visual field defect 3, 4 or X in better eye) was found in 7 per cent of recent glaucomas (fig 2). At last presentation the rate of patient handicap in the three groups amounted to only 9 - 15 per cent.

Glaucoma progression. - Glaucoma progression in different subgroups is analysed in table 2.

Table 2. Progression of glaucomatous visual field defects in 5 ± 2 years and 10 ± 2 years.

| Group | Year of ver. diagnosis | Eye | VFD stage | N | Progr % |
|---|---|---|---|---|---|
| 5-year-glauc. | 1979-83 | Worse | 2 | 127 | 30 |
| | | | 3 | 29 | 48 |
| | | Better | 2 | 70 | 27 |
| Treated before verified diagn. | 1979-83 | Worse | 2 | 20 | 5 |
| Followed before verified diagn. | 1979-83 | Worse | 2 | 18 | 0 |
| 10-year-glauc. | 1974-78 | Worse | 2 | 84 | 32 |
| | | | 3 | 13 | 54 |
| | | Better | 2 | 36 | 28 |

In worse eyes with a follow up of 3 years or more (5-year-glaucomas and 10-year-glaucomas grouped together) progression was found in 31 per cent of eyes with a visual field defect stage 2, and in 50 per cent of eyes with stage 3. The difference is statistically significant ($p < 0.02$). There is no difference in progress rate between worse and better eyes with the same stage of visual field defect.

The two groups followed before verified diagnosis differed markedly from the 5-year-glaucomas in spite of the same follow up time for the visual field defect. In the group "Treated before verified diagnosis" only one case progressed from stage 2, and in the group "Followed before verified diagnosis" no one. The differences versus the 5-year-glaucomas are statistically significant (treated group $p < 0.05$, followed group $p < 0.01$).

## DISCUSSION

Epidemiological studies (2,4,13,21,22) have shown that glaucomas with low or normal tensions are common. It may be argued that the increasing prevalence of glaucoma with age (22) would suggest, that population studies using upper age limits of 70 respectively 74 years (4,13) might have excluded a significant proportion of the glaucoma population with high pressures. In our study high initial pressures dominated - among open angle glaucomas,

diagnosed at this hospital in 1984-1986, 50 per cent had a pressure of 35 mm Hg or more, and only 4 per cent were of the low tension type with pressures below 21 mm Hg. However, the high age of our patients at first presentation can not explain this difference. We did not find any correlation of pressures to age.

The predominance (2/3) of capsular glaucomas in our material may partly explain the difference. 62 per cent of capsular glaucomas had initial pressures $\geq$ 35 mm Hg, but only 26 per cent of simple glaucomas, and no patient with capsular glaucoma had initial pressures $\leq$21 mm Hg. Figures of prevalence and incidence based on single pressure measurements should be cautiously interpreted. Several assumed low tension glaucomas can be reclassified as simple glaucomas after repeated pressure measurements (25). The diagnoses in our sample were based on diurnal pressure curves. Finally, glaucomas found in population surveys may differ in type or stage of the disease from those found in patients seeking medical care, usually because of visual disturbances (44).

The pathogenesis of glaucomatous optic nerve damage has been extensively reviewed (1,25,27,36,38). No single theory can explain all features of damage in the different kinds of open angle glaucoma, but the risk of development and progress of glaucomatous optic nerve damage increases with increasing pressures (3,5,16,19, 24,28,31,37,39,42). Correspondingly, we found a significantly higher rate of severe visual field defect in eyes with intraocular pressure of 35 mm Hg or more than in eyes with lower pressures.

In agreement with other studies (8,26,30,40,41) we found that for many patients the first glaucoma presentation was also a late presentation. Over 30 per cent of our patients had a marked visual field loss in the most affected eye, when first seeking care for eye symptoms. Severe bilateral defects were found, but were rare. On the other hand, unilateral glaucoma was common (about 45 per cent). The age at first presentation was high, 65 per cent were more than 70 and 19 per cent more than 80 years old. A combination of old age and unilateral painless disease probably explains several late presentations.

Indirect clinical evidence from retrospective studies of several hundreds of carefully examined cases indicates that prognosis of glaucoma depends on the degree of initial glaucomatous nerve damage, and these studies emphasise the importance of an early diagnosis (6,8,10,20,43). We found that visual field defects, present already at first presentation, progressed faster than defects developing during control or treatment. These latter defects were probably diagnosed within months of observable damage, and at an earlier stage of the disease, than those existing when the patient first sought medical care.

Visual field defects, however, only indirectly and non-uniformly reflect loss of optic nerve axons. Many glaucomas have already lost a majority of their axons by the time of the first presentation (33,34,35). Even a small further loss of axons might hypothetically be noted as a major progression of visual field loss (11). We found a statistically significant lower rate of progress in eyes with only an arcuate scotoma than in eyes with breakthrough of the scotoma to the periphery.

80-90 per cent of the open angle glaucoma patients still controlled at our clinic after 8-12 years' known duration of the disease had only a small or no visual impairment in the least affected eye. Even the worse eye had a seriously impaired visual function only in 20 per cent, and in over half of the cases the visual damage in this eye was small.

The percentage of progression from not handicapping to handicapping defects or from handicapping to advanced defects did not increase with increasing follow up times. This finding is probably an artefact, explained by the old age of the patients and the retrospective design of the study. The same could be said about the seemingly higher frequency of handicapping visual field defect in the group with medium duration as compared to the group with long duration, in this case the explanation is supported by the finding of a higher mean age at first presentation in the group with visual field defect stage 4.

ACKNOWLEDGEMENTS

This work was supported by grants from the Glaucoma Research Fund, Uppsala, Sweden.

REFERENCES

1. Anderson, D.R.: The optic nerve. In Adler´s Physiology of the Eye. 8th ed. C.V. Mosby, St. Louis, 1987.
2. Armaly, M.F.: On the distribution of applanation pressure and arcuate scotoma. In Paterson, G., Miller, S.J.H. & Paterson, G.D. (eds.): Drug mechanisms in glaucoma, Little, Brown & Co., Boston, 1966.
3. Becker, B. & Morton, W.R.: Topical epinephrine in glaucoma suspects. Am J Ophthalmol 62:272-277, 1966.
4. Bengtsson, B.: The prevalence of glaucoma. Br J Ophthalmol 65:46-49, 1981.
5. Cartwright, M.J. & Anderson, D.R.: Correlation of asymmetric damage with asymmetric intraocular pressure in normal-tension glaucoma (low-tension glaucoma). Arch Ophthalmol 106:898-900, 1988.
6. Chandler, P.A.: Long-term results in glaucoma therapy. Am J Ophthalmol 49:221-246, 1960.
7. Gloor, B.P., Dimitrakos, S.A. & Rabineau, P.A.: Long-term follow-up of glaucomatous fields by computerized (OCTOPUS) perimetry. In Krieglstein (ed.): Glaucoma Update III, p. 123-138, Springer-Verlag, Berlin-Heidelberg, 1987.
8. Grant, W.M. & Burke, J.F.: Why do some people go blind from glaucoma? Ophthalmology 89:991-998, 1982.
9. Haas, A., Flammer, J. & Schneider, U.: Influence of age on the visual fields of normal subjects. Am J Ophthalmol 101:199-203, 1986.
10. Harbin, T.S., Podos, S.M., Kolker, A.E. & Becker, B.: Visual field progression in open-angle glaucoma patients presenting with monocular field loss. Trans Am Acad Ophthalmol & Otolaryngol 81:253-257, 1976.
11. Hart, W.M.Jr. & Becker, B.: The onset and evolution of glaucomatous visual field defect. Ophthalmology 89:268-279, 1982.

12. Heijl, A., Lindgren, G. & Olsson, J.: Normal variability of statistic perimetric threshold values across the central visual field. Arch Ophthalmol 105:1544-1549, 1987.

13. Hollows, F.C. & Graham, P.A.: Intraocular pressure, glaucoma and glaucoma suspects in a defined population. Br J Ophthalmol 50:570-586, 1966.

14. Jaffe, G.J., Alvarado, J.A. & Juster, R.P.: Age-related changes of the normal visual field. Arch Ophthalmol 104:1021-1025, 1986.

15. Jay, J.L. & Murray, S.B.: Early trabeculectomy versus conventional management in primary open angle glaucoma. Br J Ophthalmol 72:881-889, 1988.

16. Kass, M.A., Kolker, A.E. & Becker, B.: Prognostic factors in glaucomatous visual field loss. Arch Ophthalmol 94:1274-1276, 1976.

17. Kass, M.A., Meltzer, D.W., Gordon, M., Cooper, D. & Goldberg, J.J.: Compliance with topical pilocarpine treatment. Am J Ophthalmol 101:515-523, 1986.

18. Kass, M.A., Gordon, M., Morley, R.E., Meltzer, D.W. & Goldberg, J.J.: Compliance with topical timolol treatment. Am J Ophthalmol 103:188-193, 1987.

19. Kolker, A.E.: Visual prognosis in advanced glaucoma: A comparison of medical and surgical therapy. Trans Am Ophthalmol Soc 75:539-555, 1977.

20. Kronfeld, P.C. & McGarry, H.S.: Five-year follow-up of glaucomas. JAMA 136:957-965, 1948.

21. Leibowitz, H.M., Kreuger, D.E., Maunder, L.R., Milton, R.C., Kini, M.M., Kahn, H.A., Nickerson, J., Pool, J., Colton, T.L., Ganley, J.P., Loewenstein, J.I. & Dawber, T.R.: The Framingham Eye Study Monograph. Surv Ophthalmol 24(suppl):335-610, 1980.

22. Leske, M.C.: The epidemiology of open-angle glaucoma: A review. Am J Epidemiol 118:166-191, 1983.

23. Lindblom, B. & Thorburn, W.: Prevalence of visual field defects due to capsular and simple glaucoma in Hälsingland, Sweden. Acta Ophthalmol (Copenh) 60:353-361, 1982.

24. Lundberg, L., Wettrell, K. & Linnér, E.: Ocular hypertension. A prospective twenty-year follow-up study. Acta Ophthalmol (Copenh) 65:705-708, 1988.

25. Maumenee, A.E.: Causes of optic nerve damage in glaucoma. Ophthalmology 90:741-752, 1983.

26. Miller, S.J.H. & Karseras, A.G.: Blind registration and glaucoma simplex. Br J Ophthalmol 58:455-461, 1974.

27. Mincler, D.S. & Spaeth, G.L.: Optic nerve damage in glaucoma. Surv Ophthalmol 26:128-148, 1981.

28. Nastri, G., Pascotto, F., Bifani, M. & Sbordone, G.: The role of hypertension in the genesis of glaucoma. Glaucoma 9:109-117, 1987.

29. Norell, S.E. & Granström, P.A.: Self-medication with pilocarpine among out-patients in a glaucoma clinic. Br J Ophthalmol 64:137-141, 1980.

30. Olivius, E. & Thorburn, W.: Prognosis of glaucoma simplex and glaucoma capsulare. Acta Ophthalmol (Copenh) 56:921-934, 1976.

31. Pohjanpelto, P. & Palva, J.: Ocular hypertension and glaucomatous optic nerve damage. Acta Ophthalmol (Copenh) 52:194-200, 1974.

32. Pohjanpelto, P.: Influence of exfoliation syndrome on prognosis in ocular hypertension ≥25 mm Hg. Acta Ophthalmol (Copenh) 64:39-44, 1986.

33. Quigley, H.A., Addicks, E.M., Green, W.R. & Maumenee, A.E.: Optic nerve damage in human glaucoma. II. The site of injury and susceptibility to damage. Arch Ophthalmol 99:635-649, 1981.

34. Quigley, H.A., Addicks, E.M. & Green, W.R.: Optic nerve damage in human glaucoma. III. Quantitative correlation of nerve fibre loss and visual field defect in glaucoma, ischemic neuropathy, papilledema, and toxic neuropathy. Arch Ophthalmol 100:135-146, 1982.

35. Quigley, H.A., Hohman, R.M., Addicks, E.M., Massof, R.W. & Green, W.R.: Morphological changes in the lamina cribrosa correlated with neural loss in open-angle glaucoma. Am J Ophthalmol 95:673-691, 1983.

36. Quigley, H.A.: Reappraisal of the mechanisms of glaucomatous optic nerve damage. Eye 1:318-322, 1987.

37. Schappert-Kimmijser, J.: A five year follow-up of subjects with intraocular pressure of 22-30 mm Hg. Ophthalmologica 162:289-295, 1971.

38. Shields, M.B.: The optic nerve head. In Shields Textbook of Glaucoma, 2nd ed Williams & Wilkins, Baltimore, 1987.

39. Shin, D.H., Kolker, A.E., Kass, M.A., Kabach, M.B., and Becker, B.: Long-term epinephrine therapy of ocular hypertension. Arch Ophthalmol 94:2059-2060, 1976.

40. Spaeth, G.L.: Visual loss in a glaucoma clinic. I. Sociological considerations. Invest Ophthalmol 9:73-82, 1970.

41. Thorburn, W.: The outcome of visual function in capsular glaucoma. Acta Ophthalmol (Copenh) Suppl 184:132-137, 1988.

42. Werner, E.B., Drance, S.M. & Schultzer, M.: Trabeculectomy and the progression of glaucomatous visual field loss. Arch Ophthalmol 95:1374-1377, 1977.

43. Wilson, J., Walker, A.M., Dueker, D.K. & Cricks, R.P.: Risk factors for rate of progression of glaucomatous visual field loss. Arch Ophthalmol 100:737-741, 1982.

Address for reprints:

Department of Ophthalmology
University of Uppsala
S-751 85 UPPSALA, Sweden