The visual prognosis of the fellow eye in unocular chronic open-angle glaucoma

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SUMMARY The risks of damage to the visual field in a previously undamaged eye of patients with unocular chronic open-angle glaucoma was found to be 1 out of 4 after 5 years of follow-up. The prognosis of the second eye was worse if the cup/disc ratio was ≥ 0.5 , a haemorrhage was present, and the pressure was raised.

It is now generally considered that the lower levels of raised intraocular pressure are less likely to result in subsequent damage than had previously been feared. The incidence of visual field loss in patients with raised intraocular pressure alone is only about 0.5% to 1% per year of follow-up (Anderson, 1977). It is greater at higher levels of intraocular pressure and also in older age groups (Armaly, 1972). Factors other than intraocular pressure must make some individuals and some eyes more susceptible to visual field loss (Armaly, 1969a). In order to manage more rationally the fairly large pool of patients with raised intraocular pressures it is important to learn to recognise all risk factors, so that the intraocular pressure may be reduced in those patients who require treatment, and the rest could return for observation only.

The fellow eyes of patients who have glaucomatous damage in 1 eye is of particular interest in this regard, because these patients must have some of the systemic susceptibility factors, and one would expect the incidence of visual field defects in fellow eyes to be considerably higher than among those patients who have bilaterally raised intraocular pressures without damage in either eye.

Nine patients (Harbin *et al.*, 1976) with unocular glaucomatous damage in whom the other eye developed visual defects on follow-up have recently been analysed. They were part of a group of 21 patients with monocular glaucoma who were followed up. Subsequently, presumably the same 9 patients were studied in a subset of 31 patients with monocular glaucoma (Kass *et al.*, 1976). The calculated incidence of glaucomatous damage in these fellow eyes was 43% in the first series and 29% in the second series over 3 to 7 years of observa-

tion. This indicated a much higher incidence of glaucomatous damage in fellow eyes than would have been expected from the population statistics available for patients with bilaterally raised intraocular pressure but no damage (Anderson, 1977).

In the first of those 2 studies asymmetry of intraocular pressure was present in only 43% of the group, so that in less than half of them the intraocular pressure accounted for the asymmetry of the damage. Unfortunately such studies do not measure the asymmetry of the duration of pressure elevations as a cause of asymmetric involvement. In 57% of those unilaterally damaged patients the eye with glaucomatous damage had a significantly larger cup in the damaged eye. Analysis of the level of intraocular pressure during the period of the follow-up revealed no significant differences in pressure levels above 24 mmHg in those fellow eyes which subsequently lost part of the visual field and those which did not (Harbin *et al.*, 1976).

In the second study the initial intraocular pressure level of fellow eyes which subsequently lost part of the visual field was significantly higher than in those who did not suffer loss of visual field, and pressures over 24 mmHg occurred more frequently in fellow eyes which did develop visual field defects (Kass *et al.*, 1976). None of the other factors studied, including the mean cup/disc ratio or the frequency of cup/disc ratio ≥ 0.5 were significantly different between the 2 groups of fellow eyes.

We previously reported on (Drance *et al.*, 1968) 31 patients with apparent unocular visual field defects of the chronic open-angle glaucoma type of whom the majority showed either a marked pressure difference or a perfusion pressure difference between the 2 eyes, or a local vascular accident in 1 eye to account for the asymmetry. In the remainder, in whom no asymmetric features were found, studies by means of more sensitive methods of visual field

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examination showed that they in fact had early damage in the second eye also. A significant difference was found (Armaly, 1969b) in the appearance of the optic discs of the damaged and undamaged eye of patients with unilateral visual field defects, but the cups in the undamaged fellow eyes were significantly larger than in normal eyes. The larger cups were thought to indicate either a greater susceptibility to field loss in eyes with genetically large cups, or that the fellow eye already showed acquired tissue changes of the optic nerve head prior to the development of field defects.

The present study is concerned with 104 patients with unioocular glaucomatous field and disc defects of whom 16 initially undamaged fellow eyes developed field defects when carefully followed.

Patients and method

On reviewing case records of patients with chronic open-angle glaucoma under continuous supervision all unioocular field defects associated with appropriate disc changes were made available for study. Patients were fully examined ophthalmologically, the examination including repeated screening on the Goldmann perimeter and, where necessary, static profile perimetry on the Tübingen perimeter. Stereophotographs were available on almost all of the discs. Neurovascular evaluation and biochemical investigations were available, and x-rays of the pituitary fossa excluded obvious pituitary tumour.

At least 2 normal visual field examinations before the occurrence of a visual field defect were necessary to establish an absence of a field defect. Visual field defects were considered to have developed when nasal steps larger than 5°, or deeper with static cuts than 0.5 log units, or paracentral scotoma deeper than 0.5 of a log unit and reproducible on at least two occasions occurred. Horizontal and vertical cup/disc ratios were determined in both the damaged and undamaged eyes from the stereophotographs. Patients were stratified into 5-year age groups and also by length of follow-up at yearly intervals. The observed risk of developing field defects in previously undamaged fellow eyes was calculated for every year of the follow-up. The cumulative risk of developing a field defect was also calculated.

In order to compare differences in intraocular pressure behaviour and optic nerve appearances of the 16 patients who developed field defects in the fellow eyes each of them was matched by randomly selected a control within the same age group and length of follow-up who did not develop a field defect in the fellow eye. The number of pressure observations were different in spite of the same duration of observation, and the means of the 2

groups were weighted for the number of observations and compared for statistical significance using a 2-sample *t* test.

Results

A total of 104 patients with unioocular field defects of the glaucomatous type were followed up for longer than 6 months and up to 5 years and were available for study. Sixteen patients developed a visual field defect in the fellow eye over the period of observation. The observed risk of developing field defects during each year of follow-up is seen in Table 1. The Table also shows the cumulative risk during this period of time. It can be seen that the risk of developing a field defect after 5 years is 1 out of 4.

The cumulative risk is also shown graphically in Fig. 1. It can be seen that cumulative risk is almost linear except for the third year of observation, when no fresh cases occurred. With the very small sample size this type of variation is not surprising and is almost certainly due to chance alone. It is of interest that during the fourth year the observed rate was almost twice as high as would have been expected from the previous years and the subsequent fifth year. The median age of the 16 patients who lost visual field and their 16 matched controls was 65 years and the median length of follow-up was 3 years.

Table 1 *The risk of developing a field defect in the fellow eye in unioocular chronic simple glaucoma*

<i>No. of months of follow-up (months)</i>	<i>No. of eyes followed-up</i>	<i>Observed risk of developing field defect (%)</i>	<i>Cumulative risk of developing field defect (%)</i>
0-12	104	3.8	3.8
13-24	88	6.8	10.7
25-36	62	0	10.7
37-48	45	11.1	21.8
49-60	26	3.8	25.6

Fig 1 *Cumulative risk of developing a field defect in fellow eye expressed in percentage terms*

Twenty-nine of the 32 patients had stereophotographs available for study, and in them 13 of 14 (93%) fellow eyes which developed a field defect had a cup/disc ratio both horizontally and vertically ≥ 0.5 , whereas of those who did not develop a field defect only 7 of 15 (47%) had a cup/disc ratio ≥ 0.5 . This difference is statistically significant ($\chi^2 = 5.22$ with Yates' correction, $0.05 > P > 0.02$). Thirteen of 20 (65%) fellow eyes with either a horizontal or vertical cup/disc ratio ≥ 0.5 developed field defects.

A horizontal asymmetry of the cup/disc ratio ≥ 0.2 between the eyes with a field defect and without a field defect was found in 2 of 15 (13.3%) of patients who developed a field defect in the fellow eye and in 6 of 15 (40%) of patients who did not develop a defect. This difference was not statistically significant at the 5% level of confidence. Similar findings were obtained with vertical cup/disc ratio asymmetry.

The mean intraocular pressure of the fellow eyes which developed a visual field defect was 19.89 mmHg (SD 4.76), whereas the mean intraocular pressure of those fellow eyes that did not develop a visual field defect was 18.73 mmHg (SD 4.19). This difference was statistically significant by a 2-sample *t* test on the weighted patient means ($P < 0.02$).

Of the 104 patients 12 were seen to have a haemorrhage on the disc of the fellow eye. Six (50%) developed a visual field defect in that fellow eye, whereas only 10 (11%) of those that did not have a haemorrhage developed a visual field defect ($\chi^2 = 12.5$, $P < 0.001$).

Discussion

This study confirms that the risk of the development of a visual field defect in the undamaged fellow eye of patients with glaucomatous damage in the first eye is considerably higher than would be expected from the incidence of bilateral glaucomatous damage patients with raised intraocular pressure. Over a 5-year period the risk of developing a visual field defect in the second eye of our patients was 1 in 4, whereas utilising the available incidence figures for the development of a visual field defect of 0.5 to 1% per year of patients with bilaterally raised intraocular pressure one would have expected the risk of developing a visual field defect over a 5-year period to be only 1 out of 20. It is therefore important that the second eye of a patient with chronic open-angle glaucoma in the first eye should be considered as at much greater risk of becoming damaged, and it should therefore be treated energetically to try to reduce that risk.

A large cup, presumably an acquired enlargement, was significantly more common in those second eyes that did develop a visual field defect. The large cup

in these circumstances is likely to denote an acquired tissue change in the second eye, and for this reason asymmetry between the 2 discs became a much less predictive factor.

This study further suggests that the intraocular pressure of the fellow eyes that did develop a visual field defect was very slightly higher than the intraocular pressure of those fellow eyes that did not develop a visual field defect. This difference was statistically significant, but it is biologically a very small pressure difference and so was probably not the major factor in determining whether field loss would occur or not. This study again indicates that the presence of a disc haemorrhage is associated with a significantly higher risk of developing subsequent visual field defects than the absence of an observed haemorrhage.

It would be legitimate therefore to conclude that the risk of developing a visual field defect in the fellow eye of a patient with chronic open-angle glaucoma is considerably higher than in a person who has bilaterally slightly raised intraocular pressure. A large cup in the second eye of such a person would suggest an already acquired tissue change. The presence of a haemorrhage on the disc of the fellow eye further increases that risk. Raised intraocular pressure is obviously likely to be yet another factor in determining the prognosis for visual field damage of that second eye. The management of a second eye in a patient with chronic open-angle glaucoma should therefore be fairly energetically pursued.

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