THE 5-YEAR PROGNOSIS FOR VISION IN DIABETES

by

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DIABETIC retinopathy is a common cause of blindness. The introduction of photocoagulation treatment has stimulated interest in this condition and several well designed and properly controlled studies have shown that either xenon arc or argon laser photocoagulation therapy can arrest or delay the progression of diabetic retinopathy.1-7 This treatment is now available at many centres in the UK, and the time has come to assess its impact on the overall expectation for vision in diabetics. This paper reports the natural history of visual acuity in a selected group of diabetic patients observed for at least five years. Photocoagulation therapy was performed when indicated, as were other specific ophthalmic procedures.

PATIENTS AND METHODS

The combined Diabetic/Eye Clinic at the Royal Victoria Hospital, Belfast, was established in 1972 as a referral centre for diabetic patients with ophthalmic disease in Northern Ireland. Patients have been referred to this clinic in several ways. The majority were routine referrals from the Diabetic Clinic at the Royal Victoria Hospital, for detailed fundus examination, photographic documentation and fluorescein angiography. Only those patients who had diabetes for more than 10 years are included in this particular study. Patients from the RVH or other hospital diabetic clinics and occasionally from family doctors were also referred for specific ophthalmic reasons, e.g., loss of vision. Consultant ophthalmologists in other hospitals have also referred patients for specialist advice and treatment—these tended to be only the more severe cases.

Visual functions were recorded and detailed ophthalmological examination undertaken after dilatation of the pupils (phenylephrine 10 per cent, cyclopentolate 1 per cent) at each visit. Fluorescein angiography was performed if necessary. The ocular fundi were photographically documented (standard fields). The retinal appearances were classified as "no retinopathy", "background retinopathy" (microaneurysms, haemorrhages and exudates without macular involvement) and "severe retinopathy" (proliferative vasculopathy or exudative maculopathy). The best corrected visual acuity using a Snellen chart was recorded at each visit by trained nursing personnel under medical supervision. Argon laser photocoagulation treatment was given for proliferative vasculopathy, either directly to new vessel formations if remote from the macula or optic disc, or indirectly by more generalised retinal ablation, especially in optic disc proliferation. In exudative maculopathy direct treatment was given to microvascular abnormalities associated with exudative lesions at the posterior pole, identified by fluorescein angiography or seen at the centre of circinate patterns.
Data on blood glucose control during the entire diabetic history of each patient was not available, as many had come from other hospitals. For those for whom complete or nearly complete documentation of two hour postprandial venous blood glucose values were available from the onset of diabetes, it was possible to calculate a mean and standard deviation of blood glucose for each patient. This was possible in view of the relatively large number of observations: it has been the practice in several clinics in Northern Ireland to measure postprandial venous blood glucose in diabetic patients for many years.

RESULTS

Between 1972 and 1979 we examined 1157 patients at the Diabetic Eye Clinic. Fifty-two of these patients died before 1979 and 79 are no longer attending the Clinic, 1026 patients remain under regular review. The life expectancy of these

### TABLE I

**Diabetic Eye Clinic, Royal Victoria Hospital, Belfast**

*Patients attending November 1972—August 1979*

|                   | Still Attending | Not Attending | Died | Photocoagulation treatment |
|-------------------|----------------|---------------|------|---------------------------|
| Severe Retinopathy| 145            | 28            | 28   | 142                       |
| Background Retinopathy | 453        | 18            | 15   | 126                       |
| No Retinopathy    | 401            | 33            | 6    | —                         |
| Others/unclassified | 27           | 0             | 3    | 4                         |
| **Total**         | **1026**       | **79**        | **52** | **272**                 |

### TABLE II

*Classification on Ophthalmic Assessment: Patients observed over 5 years*

| Classification of Diabetes | Mean Age at Classification (±SD) | Mean Duration of Diabetes (±SD) | Therapy Diet/OHA % | Therapy Insulin % |
|----------------------------|---------------------------------|--------------------------------|-------------------|------------------|
| Severe retinopathy (40 patients) | 47 ± 15                         | 18 ± 7                         | 83                | 17               |
| Background retinopathy (33 patients) | 55 ± 14                        | 16 ± 12                        | 76                | 24               |
| No retinopathy (32 patients)      | 42 ± 17                         | 12 ± 10                        | 81                | 19               |

OHA—Oral hypoglycaemic agent.
patients has been analysed separately. Two hundred and seventy-two patients have received photocoagulation treatments between November 1972 and August 1979 (Table I). We have now continued observation of 105 of these patients for at least five years. The ophthalmological classification in this subgroup at the initial examination showed no retinopathy in 32, simple background retinopathy in 33 and severe retinopathy in 40 (Table II). The mean age and mean duration of diabetes at classification were not significantly different. The great majority of patients were on insulin treatment. Blood glucose data was available for 23 of the no retinopathy group, 21 of the background retinopathy group and 27 of the severe retinopathy group, who had attended the RVH diabetic clinic.

Figure 1. *Initial visual acuity on vertical axis: Visual acuity at 5 years on horizontal axis. Each circle or triangle represents one eye. Open circles indicate eyes photocoagulated. Numbers indicate numbers of eyes. Diagonal line represents no change in visual acuity over 5 years.*

CF = counting fingers
PL = perception of light
HM = hand movements
NPL = no perception of light
Figure 2. Initial visual acuity on vertical axis: Visual acuity at 5 years on horizontal axis. Each circle or triangle represents one eye. Open circles indicate eyes photocoagulated. Numbers indicate numbers of eyes. Diagonal line represents no change in visual acuity over 5 years.

CF = counting fingers  
HM = hand movements  
PL = perception of light  
NPL = no perception of light

**TABLE III**

5 Year Prognosis for Vision: Individual Eyes  
(a change of 2 lines in the Snellen chart was necessary for change in visual assessment)

| Visual Assessment | Severe Retinopathy | Background Retinopathy | No Retinopathy |
|-------------------|--------------------|------------------------|----------------|
| Same              | 25 (31%)           | 43 (65%)               | 55 (86%)       |
| Better            | 10 (13%)           | 5 (8%)                 | 4 (6%)         |
| Worse             | 45 (56%)           | 18 (27%)               | 5 (8%)         |
|                   | 80                 | 66                     | 64             |
The visual acuity at the initial assessment compared to that at five years later for the three subgroups is plotted in Figures 1, 2 and 3 and analysed in Table III. Considering individual eyes there were 64 with no retinopathy, 66 with background retinopathy and 80 with severe retinopathy (Table III). Visual acuity deteriorated over the five years in five eyes (8 per cent) that were initially classified as no retinopathy due to the development of cataracts. In the background retinopathy group visual acuity deteriorated in 18 (27 per cent) of the eyes—in the majority due to the development of macular exudates, only a few eyes developing new vessels. Forty-three (65 per cent) of eyes initially classified as background retinopathy remained unchanged over five years. In the severe retinopathy group visual acuity

Figure 3. Initial visual acuity on vertical axis: Visual acuity at 5 years on horizontal axis. Each circle or triangle represents one eye. Open circles indicate eyes photocoagulated. Numbers indicate numbers of eyes. Diagonal line represents no change in visual acuity over 5 years.

CF = counting fingers
PL = perception of light
HM = hand movements
NPL = no perception of light

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TABLE IV

**Risk Factors for Visual Deterioration in Severe Retinopathy**

| Condition                                      | Age (±SD) | Duration of Diabetes (±SD) | Therapy | OHA | BP Mean |
|------------------------------------------------|-----------|---------------------------|---------|-----|---------|
| Poor visual acuity (<6/60) at year 0          | 53 (±13.2)| 17.5 (±9.8)               | 58      | 42  | 153/90  |
| Deterioration in visual acuity to <6/60 in 5 years | 44.4 (±15.2)| 16.4 (±7.9)             | 88      | 12  | 139/87  |
| No change in visual acuity (>6/60) over 5 years | 44.4 (±14.5)| 18.7 (±3.1)             | 86      | 14  | 141/79  |

OHA—Oral hypoglycaemic agent.

TABLE V

**Relation of 2-3 hour postprandial venous blood glucose levels in the long term to the presence of diabetic retinopathy**

| Condition               | No Retinopathy | Background | Severe |
|-------------------------|----------------|------------|--------|
| Number                  | 33             | 21         | 27     |
| Age at onset            | 32             | 28         | 35     |
| Present age             | 47             | 46         | 54     |
| Duration of diabetes    | 15             | 19         | 20     |
| Mean total number of recorded blood glucose per patient | 70 | 102 | 69 |
| Average number of blood glucose per year of follow up | 6 | 6 | 4 |
| Mean recorded blood glucose (±SD) since first attendance mmol/1 | 9.4 ± 4.8 | 10.1 ± 5.4 | 10.1 ± 4.8 |

deteriorated in 45 eyes (56 per cent) over five years, remained unchanged in 25 (31 per cent) and improved in 10 (13 per cent). Extensive neovascularization and its sequelae was responsible for deterioration in the majority of the severe retinopathy group. A change of two lines on the Snellen chart in the five years was necessary for this analysis. Seven eyes (11 per cent) received argon laser photocoagulation therapy in the “background” group, 38 (48 per cent) in the “severe” group.
Age, and duration of diabetes, or the need for insulin therapy were not different in those patients whose vision deteriorated (Table IV). The mean blood pressure in those patients whose vision deteriorated was not significantly different from those whose vision did not change. For the patients for whom documentation of outpatient blood glucose values was possible, the mean ± standard deviation of the recorded 2 hour postprandial blood glucose values are shown in Table V. The mean ages and duration of diabetes for these subgroups are somewhat different from those of the complete data. An average of between 69 and 102 blood glucose values were available for these three subgroups, obtained at routine outpatient reviews—this gave an average of four to six values annually. The mean recorded blood glucose from the time of first diagnosis for those in the no retinopathy group was 9.4 mmol/l, for the background retinopathy group 10.0 mmol/l and for the severe retinopathy group 10.0 mmol/l.

DISCUSSION

The prognosis for vision in diabetics with established severe retinopathy is poor, but has improved since the introduction of photocoagulation treatment. Several trials have demonstrated the effectiveness of this therapy in modifying the progress of proliferative retinopathy and exudative maculopathy. There was no difference in antidiabetic therapy or duration of diabetes in our patients whose vision deteriorated compared with those whose vision remained unchanged or improved.

In the absence of a fully prospective study of the development of retinopathy in relation to blood glucose control, it is relevant to observe even the partial documentation which is available in our hospital records. The tradition of measuring outpatient 2-3 hour postprandial venous blood glucose values under supervision has continued for many years in Northern Ireland, and we feel that the incomplete data presented in Table V at least merits inspection. None of the three groups achieved what might be called good control of their observed blood glucose values, but equally, those with the severe retinopathy did not have the highest mean blood glucose value. The studies of Job et al and Ashikaga et al have not resolved the question of whether or not diabetic control affects the development or progress of retinopathy.

Coagulation abnormalities secondary to the abnormal metabolic state in diabetes may be a factor in diabetic retinopathy. The elevated levels of glycosylated haemoglobins found in diabetics with 'bad' control may be associated with tissue hypoxia. In an associated study in Belfast Elder et al did not confirm the presence of a hypercoagulable state in diabetic patients with retinopathy. Rather than a specific or primary increase in one of the many coagulation factors, they found a generalised increase in both coagulation and fibrinolytic elements of the blood clotting mechanism, and felt that these changes were more likely to be secondary to the presence of microvascular disease.

Smoking may be a risk factor for proliferative retinopathy, but we did not investigate this in detail. A greater prevalence of visual deterioration was found among non-smokers in this study, but there may be many reasons for this unexpected finding. The assessment of smoking habits was by simple verbal
questioning at non-standardised times, and it is recognised that as vision deteriorates, many smokers tend to discontinue the habit.

If it is accepted that early detection and treatment of diabetic retinopathy can arrest deterioration of visual acuity, consideration must be given to the staffing of specialised diabetic ophthalmic clinics. From the data at the RVH diabetic clinic of 2200 patients it is estimated that there are about 30 patients with 'severe' retinopathy per 1000 patients attending. Donovan\(^18\) had found a much higher prevalence of 11.6 per cent 'severe' retinopathy in patients attending a diabetic clinic at a district general hospital. The relatively inefficient fundus examination possible through undilated pupils at a diabetic clinic may pick up early retinal disease, but our policy is to obtain detailed ophthalmological examinations in all insulin-requiring diabetics of 10 years duration or where patients present at any time with symptoms of disturbed vision. Only detailed examination of this type can allow accurate assessment of clinical severity.

**SUMMARY AND CONCLUSIONS**

A group of 105 diabetic patients, 32 of whom had no detectable retinopathy, 33 simple background retinopathy and 40 of whom had severe retinopathy were observed over five years with specialist ophthalmic supervision. In 146 eyes with retinopathy 68 (46.6 per cent) remained unchanged, 61 (41.8 per cent) deteriorated and 17 (11.6 per cent) improved during the five years; those graded as severe retinopathy at initial assessment deteriorated most. Age and duration of diabetes, or the need for insulin therapy were not different in those patients whose vision deteriorated. Hypertension was rare. Assessment of available recorded blood glucose values in some of these patients suggested that ideal control of the blood glucose had not been achieved in any of the three groups classified by severity of the retinopathy: neither did those with the most severe retinopathy have the highest mean recorded blood glucose values.

The five year prognosis for vision in this diabetic population was relatively good. Only a small proportion of diabetics develop severe retinopathy. Vision in this group may deteriorate in spite of photocoagulation treatment.

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