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Progression of diabetic retinopathy following cataract surgery: can it be prevented?

Diabetic patients have an increased risk of developing cataract. This risk is related to age, severity of retinopathy, duration of the disease and, possibly, systemic hypertension.^{1,2} Harding *et al* in 1992, using data from the Oxford Region in the UK, demonstrated that diabetes mellitus is associated with a fivefold increase in the risk of developing cataract. They further estimated that 11% of all cataracts in the UK were in diabetic patients.³ The Royal College of Ophthalmologists' audit of cataract surgery in 1991 revealed that 4% of all patients undergoing cataract surgery had diabetic retinopathy. Approximately 50 patients with diabetic retinopathy have cataract surgery each week in the UK.⁴ Cataract is very common after laser photocoagulation for proliferative diabetic retinopathy. Blankenship, in 1989, reviewed all the survivors of the original diabetic retinopathy study photocoagulation trial in 1976 and found that 13 out of 51 eyes had had cataract surgery in the 15 years after photocoagulation.⁵ In addition, posterior vitrectomy for vitreous haemorrhage and tractional retinal detachment is frequently followed by cataract.

Cataract surgery in diabetic patients, therefore, forms a significant part of every eye department's workload. It is obviously important that such patients are managed appropriately to minimise visual loss from progression of diabetic retinopathy.

Cataract surgery in diabetics with little or no retinopathy has the same good prognosis as cataract surgery in non-diabetics.⁶⁻⁸ However, in the presence of significant diabetic retinopathy the results can be disappointing.⁹⁻¹¹ The paper by Henricsson *et al* in this issue of the *BJO* (p 789) demonstrates, for the first time, that it is possible, in the presence of diabetic retinopathy, to predict outcome. In this small, but carefully documented prospective study, 89% of eyes achieved 6/12 (0.5) or better vision and maintained it for at least 18 months. One third of the eyes had non-proliferative diabetic retinopathy and almost a quarter had treated proliferative diabetic retinopathy.

Severe visual loss following cataract surgery in diabetics may be due to worsening macular oedema, continuing anterior and posterior segment proliferation, posterior capsule opacification, or unrelated events, such as retinal vein occlusion. Risk factors associated with worsening retinopathy after cataract surgery include pre-existing severe treated or untreated retinopathy, poor glycaemic control, increasing age, and planned or unplanned posterior capsule disruption.

Non-proliferative diabetic retinopathy can rapidly progress to severe diffuse macular oedema in the months following uncomplicated cataract extraction. Jaffe and Burton¹² and later Schatz *et al*¹³ emphasised that the retinopathy progressed rapidly in the operated eye compared with the fellow, control phakic eye. None of the patients documented in these reports had received any preoperative

photocoagulation. Pollack *et al*,⁹ as well as Cunliffe *et al*,⁶ showed that macular oedema and neovascularisation can worsen after cataract extraction even with photocoagulation. Fortunately, only a small minority of diabetics with non-proliferative diabetic retinopathy develop severe persistent macular oedema and poor vision after cataract surgery. Pollack *et al*¹⁴ also demonstrated that 81% of eyes with pre-existing background retinopathy developed clinical cystoid macular oedema after uncomplicated cataract surgery compared with only 32% of eyes without background retinopathy. Menchini *et al*¹⁵ compared the incidence of cystoid macular oedema following cataract extraction in diabetics with no diabetic retinopathy with normal non-diabetic controls using fluorescein angiography. They found that the incidence of fluorescein angiographic macular oedema was similar at 30 days in both groups, but that 24.5% of the pseudophakic diabetic eyes still had macular oedema at 1 year compared with none of the non-diabetic pseudophakic controls. This suggests that the blood-retinal barrier is significantly impaired, even in diabetics with no retinopathy and that cataract surgery worsens this impairment.

Preoperative and early postoperative photocoagulation for macular oedema appears to reduce but not to eliminate the risk of visual loss. For this reason, careful preoperative assessment and regular follow up, if necessary using fluorescein angiography, are essential. Fluorescein angiography is particularly required to differentiate between pseudophakic cystoid macular oedema and worsening diabetic macular oedema which may require photocoagulation.⁹

Many patients, including those with diabetic retinopathy, may have very high expectations from cataract surgery. For this reason, patients with diabetic retinopathy and cataract need to be advised preoperatively that retinopathy and vision may worsen after cataract extraction.^{11,13}

Neovascular glaucoma and rapidly progressive proliferative diabetic retinopathy can occur after extracapsular cataract surgery in treated and untreated proliferative diabetic retinopathy.^{6,11,16,17} Pollack *et al*⁹ described the rapid development of severe retinal ischaemia confirmed by fluorescein angiography in the 3 months following uncomplicated extracapsular cataract surgery. The visual results of extracapsular cataract surgery in treated proliferative diabetic retinopathy with maculopathy are frequently poor. However, good results have been reported in well treated proliferative diabetic retinopathy without maculopathy.^{5,16}

Adequate panretinal laser photocoagulation is therefore essential if there is severe peripheral retinal ischaemia or early retinal neovascularisation. This photocoagulation should be applied preoperatively. If this is not possible it can be done peroperatively using the laser indirect

ophthalmoscope or in the early postoperative period. Otherwise, a severe fibrinous uveitis with adhesions may prevent photocoagulation. Eyes with severe untreated retinopathy always have abnormally permeable iris vessels so a severe fibrinous uveitis may follow even minimal iris trauma in uncomplicated surgery.¹⁸

Age can be a useful predictor of outcome following cataract surgery in diabetics. Benson *et al*¹¹ reported that 58% of patients under 63 achieved 6/12 or better but only 38% of their patients over 64 years of age achieved this level.

Posterior capsular opacification is significantly more common in diabetics with retinopathy than in non-diabetics. Ionides *et al* stressed the importance of early capsulotomy to ensure an adequate retinal view.¹⁹

The visual results reported following cataract surgery in diabetics by Henricsson *et al*, on p 789, are significantly better than the results from equivalent series in the past: 89% of their patients achieved 6/12 or better. This compares favourably with 67% in Cunliffe *et al*'s series,⁶ 67% in Hykin *et al*'s series,¹⁶ and 48% in Benson *et al*'s report.¹¹

It is of particular interest that only one operated eye lost vision after surgery and this was due to a retinal vein occlusion. In two patients the visual acuity was worse in the operated eye compared with the phakic control fellow eye. Overall, however, there was no difference in the rate of progression of the retinopathy between the operated and unoperated eyes.

Most previous reports have stressed that retinopathy may progress rapidly in the pseudophakic eye compared with the control fellow phakic eye. Henricsson *et al* found that any progression of retinopathy appeared to be related to higher levels of haemoglobin A_{1c} pre- and postoperatively and not to cataract surgery. Most patients in their study had reasonably good control of blood sugar with no patient having a haemoglobin A_{1c} greater than 9.1%. Good glycaemic control and its beneficial effect on progression of retinopathy after cataract surgery has not been reported in the past.

In summary, the good results achieved in this paper appear to be due to the following factors: the cataract extraction was performed when indicated as part of a long term comprehensive diabetic care programme; there was careful long term control of blood sugar; there was appro-

priate pre- and postoperative photocoagulation; and the decision to operate was based on detailed knowledge of the visual acuity and retinopathy over several years, thus reducing the likelihood of cataract surgery on eyes with poor visual potential.

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