glaucoma and one hopes that appropriate histological investigations will clarify these issues in the future.

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Paecilomyces keratitis

EDITOR,—The successful treatment of a *Paecilomyces* keratitis described by Mizunoya and Watanabe¹ raises a number of important issues. *Paecilomyces* as a cause of deep keratitis has been reported in a series² of cases to which now a further two cases have been added. As described in this article, medical treatment is rarely successful and the only cases in which the patients were able to maintain an eye was when surgical extirpation of the corneal infection was undertaken. The lesion as depicted in Figure 1C, would have lent itself to a large eccentric corneal patch graft despite the authors' concerns about approaching the limbus at the 6 o'clock position. Although there was an apparent beneficial effect of pulling a conjunctival flap over a perforated cornea, the predictable result of secondary glaucoma from loss of the angle resulted. This sequela is far less acceptable than a possible corneal graft rejection from an eccentric graft. I believe the authors were exceptionally lucky to maintain an eye with some vision. The suggestion that a conjunctival flap is an appropriate method of treatment in a perforated cornea may lead readers to emulate this treatment which will result in many disasters of continuing infection and secondary glaucoma. The use of large corneoscleral grafts³ despite their many associated problems, would be a preferable method of treatment, both in eradicating the disease and maintaining a relatively normal angle anatomy, than covering an active infection and a large perforation with conjunctiva.

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Reply

EDITOR,—We appreciate the comment of Hirst concerning our case report. We agree

that treating corneal defect by sclerokeratoplasty improved by Cobo *et al*¹ will prevent secondary angle closure glaucoma. However, the sclerokeratoplasty has the following problems. Firstly, because in this method a 1 to 2 mm peripheral corneal lip is left in the recipient's eye, when the lesion is close to the limbus as our case, fungi may remain in the lip. Secondly, a very high incidence of graft rejection necessitates repeated keratoplasty, which is difficult to do in Japan where donor corneal grafts are few in number. We regret that in our case anterior segment reconstruction² at the time of secondary keratoplasty was not enough. If it had been done thoroughly, trabeculectomy might have been unnecessary.

Keratomycosis with perforation is a very severe condition, and there is no reliable treatment to salvage an eye. We would select primary keratoplasty, sclerokeratoplasty or the method we reported according to the position of the perforation, the size of it, and the activity of the lesion.

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Could colour vision tests predict or find retinopathy in diabetic schoolchildren?

EDITOR,—Only a few studies concerning colour vision of diabetic children are available.¹⁻³ In them, colour vision has been found to be normal, and a follow up study of diabetic children and young adults showed that colour vision (examined with the Farnsworth-Munsell 100 hue test) deteriorated with the development of retinopathy, but not before.⁴ Distinguishing retinopathic eyes from those without retinopathy has not been studied with colour vision tests in this age group.

We have observed colour vision of diabetic schoolchildren with healthy eyes during 6 years, from 1987 to 1993. Answers for two questions were sought: (1) Would the results of colour vision tests in diabetic children without retinopathy in 1987 predict the development of retinopathy during the next 6 years? (2) Would the results of colour vision tests in 1993 distinguish the eyes with retinopathy from those without retinopathy?

(1) *The follow up study 1987-1993.* In 1987 colour vision of 54 diabetic schoolchildren with healthy eyes was studied (29 girls, 25 boys; aged 9-19 years (mean 14 (2 SD) years); duration of diabetes from 1 month to 15 years, mean 6 (4) years. Colour vision was examined with the Farnsworth panel D 15, Lanthony desaturated panel, and Nagel I anomaloscope. The panel D 15 was correctly interpreted by all of the children. In the desaturated panel, seven children did not pass the test showing 1-3 tritan confusion lines. In the Nagel anomaloscope examination, normal anomalous quotients (AQ, from 0.7 to 1.2) and normal matching ranges (MR, from 0 to 5) were observed in all children. Fifty six eyes had an MR completely on the red side of the Rayleigh equation. (The predictive value of the red side MR for the appearance of retinopathy has been suggested.)

In 1993, 23 of the children (35 eyes) had retinopathy, which in 33 eyes was background retinopathy. None of these eyes had macular involvement. Only one patient had proliferative retinopathy with macular oedema in both eyes. Of those seven children who did not pass the desaturated panel test in 1987, only two developed background retinopathy in one eye. However, all these seven children passed the desaturated panel in 1993. The patient with proliferative retinopathy passed the test normally in 1987. Of the 56 eyes with a red side MR in 1987, 16 (29%) had developed background retinopathy, 40 eyes still had intact retinas. Of the 52 eyes with a green side or mixed red-green side MR, 19 (17 background and two proliferative, 37%) had developed retinopathy, 33 eyes had intact retinas.

(2) *The cross sectional study 1993.* In addition to the above mentioned colour vision tests, the blue equation of another anomaloscope: colour vision meter (CVM) 712 was used. Fifty three of the patients passed both the panel D 15 test and the desaturated panel test. Only the 23-year-old man with proliferative retinopathy and macular oedema in both eyes could not correctly interpret the panel D 15 test; he made four tritan confusion lines in both eyes. The desaturated panel was impossible for him to work. In the Nagel anomaloscope and CVM, there were no significant differences in the results of AQs or MRs between the retinopathy and non-retinopathy group. The Nagel anomaloscope results, AQs and MRs, were all within normal limits. In the CVM, four diabetic patients (four eyes) had an abnormal AQ, >1.4. One of the eyes had no retinopathy, two eyes had background retinopathy, and one eye had proliferative retinopathy. All the other 31 eyes with retinopathy (one of them with proliferative retinopathy) had AQs within normal limits. Ten of the retinopathic eyes showed an abnormal MR in the CVM, from 11 to 26, mean 18 (5). Also 10 non-retinopathic eyes had an abnormal MR, from 11 to 21, mean 15 (3). No significant difference was found between these means.

Conclusion. The 6 year follow up study in colour vision of diabetic schoolchildren showed that no predictive signs for retinopathy could be found with the panel D 15, desaturated panel or Nagel I anomaloscope test results. In the cross sectional study, it was observed that the eyes with and without retinopathy in diabetic schoolchildren and young adults could not be distinguished from each other with the results of four colour vision tests: panel D 15, desaturated panel, Nagel I anomaloscope, and colour vision meter 712 (blue equation). Only the two eyes with advanced proliferative retinopathy with macular oedema could be found with the panel D 15 test.

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