necessarily requires the excitation of different cone systems.[5] This model indicates that it is probably luminance channel contrast that is increased using the green filter on the slit lamp to aid detection of diabetic retinopathy rather than a colour channel contrast change.

Altering the spectral illuminant to maximise our ability to detect abnormality in the diseased retina may well be able to play a part in management in the future. One can envisage a series of different filters, each designed to highlight specific changes in the retina, that could allow earlier detection of many disease processes.

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Sir,

We welcome the opportunity to reply to Mr Davies' letter to clear up the methodology questions that were raised. The filter available on the Haig-Streit slit lamp generated the green light, no filter was used for examination with white light and in all cases the slit lamp bulb was run at 4 volts. As stated the bulb voltages of both the direct and indirect ophthalmoscopes were controlled with a potentiometer and, while it was difficult to standardise, an attempt was made to use the minimum amount of light necessary to illuminate the fundus when using these instruments. We read with great interest his discourse on the physical mechanism underlying our findings; we agree with his theory and congratulate him on an elegant and erudite explanation.

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Sir,

We read with interest the case report on lightning-induced cataract by Cazabon and Dabbs,[1] who report their case to be possibly the first in the United Kingdom. However, we reported a case[2] in 1998 which was, to our knowledge, the first case of lightning-induced cataract reported in the UK and the very first in the world literature reporting a cataract caused by telephone-mediated lightning injury.

Cazabon and Dabbs describe a patient who developed cataract following a direct lightning strike. Our patient, a 9-year-old boy, developed a posterior subcapsular cataract in his right eye following a lightning strike whilst using a telephone in his home during a thunderstorm. The lightning strike damaged the telephone box and caused superficial facial burns. The cataract was similar to that described by these authors. An uneventful cataract extraction has resulted in a visual acuity of 6/5.

Lightning can traverse the telephone user in two ways.[3] The first method is by the current generated in the communication line as it is struck by lightning. The second method is by an interesting phenomenon called 'earth potential rise' or EPR. The earth is thought to be at zero potential continuously, although the potential can rise when struck by lightning. The telephone is held at zero potential by its connection to the remote earth. When the earth potential rises during a strike, the potential difference between the telephone and the earth makes the current flow through the user to the remote earth, harming the telephone user. Hence the advice: do not use a telephone during a thunderstorm.

These two cases illustrate the dangers of lightning by direct and indirect strikes.

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Sir,

For our case report, lightning-induced cataract, a literature search was carried out using PubMed. The keywords used were 'lightning-induced cataract' and were matched to three reports,[1–3] all found outside the UK. However, we acknowledge the case report telephone-mediated lightning-induced cataract by Dinakaran *et al.*,[4] and also found it very interesting. We would therefore like to apologise if our information was in any way misleading, although it was not intended to be.

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Sir,

We read with interest the report by Venugopalan *et al.*[1] of a girl with Proteus Syndrome associated with ocular anomalies. Ocular complications are frequently reported in patients with Proteus Syndrome.[2–5] However, in a review of the literature, Bouzas *et al.* found that only two out of over 50 patients with Proteus Syndrome had a comprehensive ocular examination.[6] Reported findings were periorbital exostosis, epibulbar tumour, retinal vascular tortuosity, 'enlarged eye', posterior segment hamartoma, heterochromia iridis, retinal coloboma, glaucoma, retinal detachment, cataract, lid hamartoma, strabismus, nystagmus and ptosis. The recent report by Venugopalan *et al.* provides an interesting addition to the ophthalmic literature on Proteus Syndrome, but their assertion that eye changes are unreported in this condition is clearly unsubstantiated by literature review.

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Sir,

We thank Sheard and Snead for their interest and comments on our paper. We note their observation that there have been previous studies on ocular involvement in Proteus Syndrome; we were genuinely unaware of these studies while preparing our manuscript. We apologise for the error. However, we would like to state that these previous publications only serve to substantiate our statement recommending a comprehensive examination of all patients with Proteus Syndrome, including a complete ophthalmic evaluation.

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Sir,

We read the article by King *et al*.[1] with interest. We have recently undertaken a review of blindness and partial sight registrations in the Bristol area. Data on age and cause of loss of sight from registrations for the period 1 August 1990 to 31 July 1993 were examined by retrieving all BD8 forms for individuals living in the Bristol area and registered at the Bristol Eye Hospital (population served approximately 850 000). During this 3 year period, 1468 individuals were registered. Of these, 890 forms (61%) were examined. Of those not examined 102 (7%) had died, 213 (14.5%) had not been seen for over 6 years and their files had been destroyed, 183 (14.3%) were being seen in other hospitals (e.g. Weston-Super-Mare) and 56 (3.8%) were being seen privately. Age-specific rates were calculated using the 1991 population census figures for Avon available from the Office of National Statistics. Results were compared with a similar review of registrations undertaken in Avon for the period 1984–1986.[2]

Analysis of the causes of registration for the 890 available forms demonstrated that age-related macular degeneration is by far the most common primary cause of sight loss, increasing since 1984–1986 in terms of the total proportion of blindness/partial sight. However, glaucoma remains the second most common single cause, and the overall proportion of sight loss from this cause has not declined. This reflects the experience of King *et al*.,[1] who have demonstrated that despite ongoing care and surveillance within the hospital eye service 35% of the 258 patients followed up from 1982 achieved eligibility for registration as blind or partially sighted, although only 18% were actually registered.

The proportion of cases registered blind or partially sighted due to glaucoma appears to have changed little since 1984–1986, when Grey *et al*.[2] demonstrated that glaucoma was responsible for 13% of registrations in Avon. These findings are consistent with a comprehensive study of blindness in the UK[3] from 1950 to 1990 which found that registrations due to age-related macular degeneration were increasing whilst those for all causes, cataract, glaucoma and optic atrophy have decreased. From these national data, it was notable that no appreciable decline in standardised registration rates for blindness was observed between 1980 and 1990 for men and women for glaucoma, which is consistent with the results from our study.

Despite advances in therapy, glaucoma remains a significant cause of blindness within the community. The Office of National Statistics is due shortly to publish the registration data for the previous 3 years, which we await with interest to see whether shifts have occurred.

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Table 1. *Causes of registration of partial sight and blindness in Avon 1984–1986 and 1990–1993 (number and %)*

	1984–1986 (n = 890)			1990–1993 (n = 1692 eyes)[a]		
Cause	*Partial sight*	*Blind*	*Total*	*Partial sight*	*Blind*	*Total*
ARMD	285 (51.9)	187 (55.0)	472 (53.0)	565 (49.0)	234 (43.0)	799 (47.0)
Glaucoma	93 (16.9)	28 (8.2)	121 (13.6)	176 (15.0)	40 (7.0)	216 (13.0)
Diabetic retinopathy	33 (6)	32 (9.4)	65 (7.3)	71 (6.0)	51 (9.0)	122 (7.0)
Cataract	12 (2.2)	5 (1.5)	17 (1.9)	24 (2.0)	30 (5.0)	54 (3.0)
Other	127 (23.1)	88 (25.9)	215 (24.2)	310 (27.0)	191 (35.0)	501 (30.0)
Total	550 (100.0)	340 (100.0)	890 (100.0)	1146 (100.0)	546 (100.0)	1692 (100.0)

[a]Of the total of 1468 BD8 forms analysed.
ARMD, age-related macular degeneration.