Sir,

I read with great interest the study by Cahill *et al*.[1] on the clinical detection of diabetic retinopathy in white and green light. I was, however, disappointed that the authors did not report the details of the light sources used. I assume that the 'green' light was generated by using the filter that is commonly attached to the slit lamp, direct and indirect ophthalmoscopes and that the 'white' light was generated by tungsten bulbs. It is important to point out that there are nine different combinations of bulb voltage and filters in the Haag-Streit slit lamp, each of which will produce a different 'white', and that the bulb voltage of the direct and indirect ophthalmoscopes is variable by a potentiometer. Considering just the slit lamp, the bulb can be run at 4, 5 or 6 volts. As the bulb voltage increases, the tungsten filament temperature increases and the spectrum of light it produces changes, shifting to shorter wavelengths. The slit lamp housing contains an infrared (IR) filter and a neutral density (ND) filter that will also affect the spectrum of light reaching the patient's eye. The green filter can be used with any bulb voltage, but not the IR or ND filters, but this still gives three different 'green' lights that could be used. Did the authors constrain the use of the bulb voltage, IR and ND filters to a single setting, or were the examiners free to vary these parameters?

Out of interest, I measured the spectral output of a Haag-Streit slit lamp for the different combinations of filters and the results are shown in Fig. 1. The colours produced by such spectra can be represented in CI x,y coordinates, with the luminance as the tristimulus value Y given in Table 1.

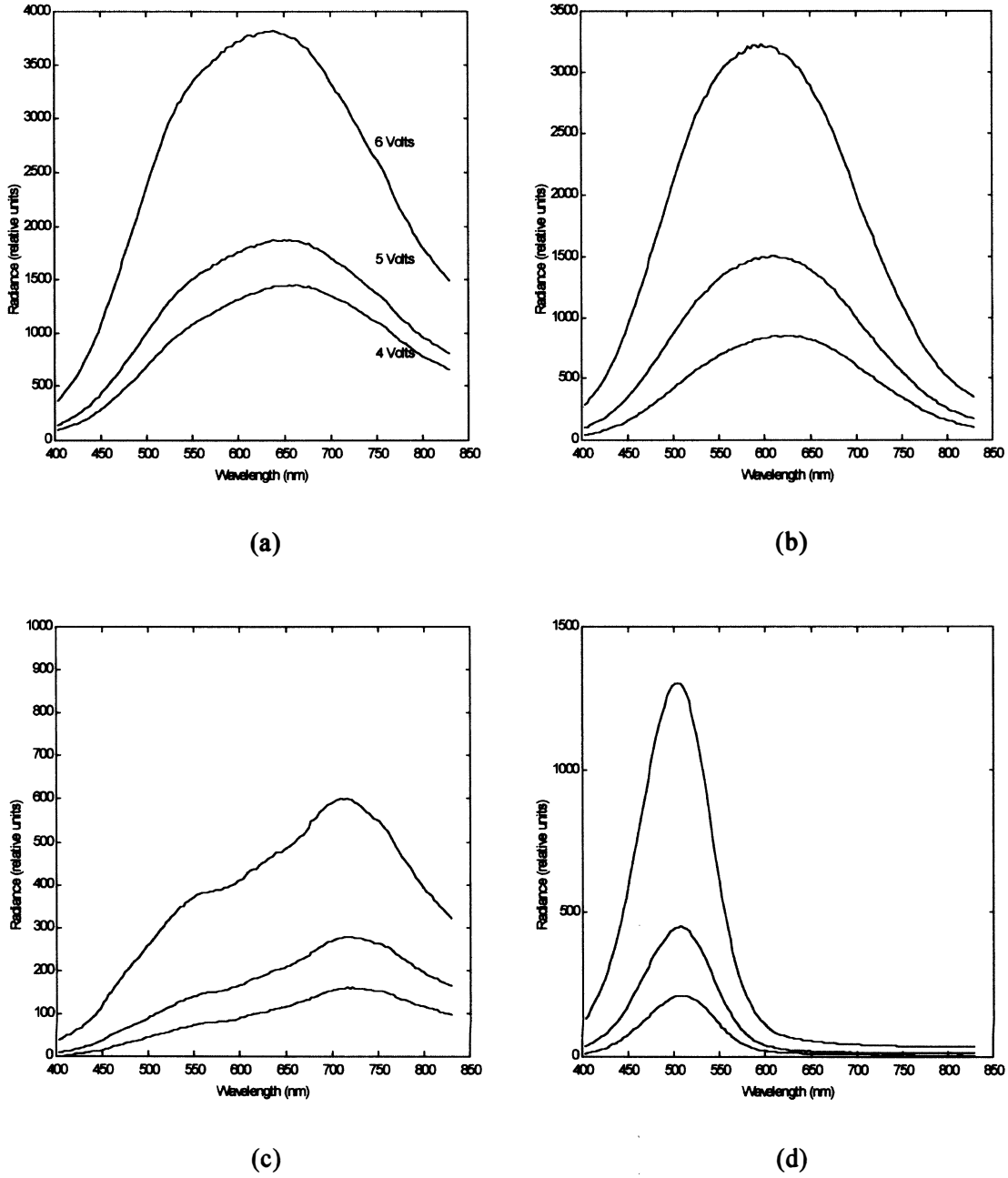

Fig. 1. *Spectral output of the slit lamp (a) with no filter, (b) with the infrared filter, (c) with the neutral density filter and (d) with the green filter, each for the tungsten bulb at 4, 5 and 6 volts.*

Table 1. *x,y chromaticity coordinates and Y luminance values of the spectral sources on a slit lamp*

Source	x	y	Y (relative units)
4V, no filter	0.4383	0.4297	39191
4V, IR filter	0.4394	0.4378	24633
4V, ND filter	0.4568	0.4344	2677
5V, no filter	0.4286	0.4286	53809
5V, IR filter	0.4243	0.4358	46129
5V, ND filter	0.4405	0.4265	5103
6V, no filter	0.4156	0.4251	117470
6V, IR filter	0.4100	0.4304	102850
6V, ND filter	0.4207	0.4208	13150
4V, green filter	0.1955	0.4063	3423
5V, green filter	0.1896	0.3864	7016
6V, green filter	0.1844	0.3656	19670

IR, infrared, ND, neutral density.

The authors made little mention of the mechanism that might lead to an increased sensitivity and specificity of retinopathy detection during clinical examination. 'Red-free' light is thought to facilitate detection by increasing contrast. The task of detection of retinopathy is psychophysical and relies on the luminance and colour contrast detection thresholds of the examiner. These contrasts of retinopathy (e.g. microaneurysm) seen against the normal fundus will be affected by the spectral composition of the illuminating light, the ocular media of the patient and the ocular media of the examiner. I thought it would be interesting to see how the various spectral outputs of the slit lamp might affect the contrast of a microaneurysm seen against the fundus for a standard young observer, using a model.

The model requires two spectra, one for the light exiting the patient's eye after reflection from normal retina, the other for the light reflected by a microaneurysm. These spectra can be estimated using spectral reflectance data for the normal fundus and haemoglobin for the microaneurysm.[2] Passage through the examiner's ocular media can be estimated using a lens absorption curve for a young observer.[3] The S, M and L cone excitations resulting from such illumination can be calculated for both spectra and these transformed into DKL colour space,[4] which gives an estimate of the activation of the post-receptoral pathway in the examiner's visual system, in the luminance (L+M) channel and the red–green and blue–yellow colour opponent channels (L–M and S – (L+M)). These values can be expressed as a contrast and thus the effect of change in spectral illumination investigated.

The results of this model are shown in Fig. 2. The contrast of a microaneurysm seen against the fundus is increased in the luminance and blue–yellow channels of the observer in green light, but little effect is seen in the red–green channels in comparison with white light. The different white lights used do give different values of contrast in all three channels, but the change is small and probably not significant. In terms of the spatial detection of retinopathy the blue–yellow channel may not contribute greatly as the S cone matrix is relatively sparse in comparison with the M and L cone matrix. The luminance pathways are known to have a greater spatial resolution than the colour opponent pathways as the latter

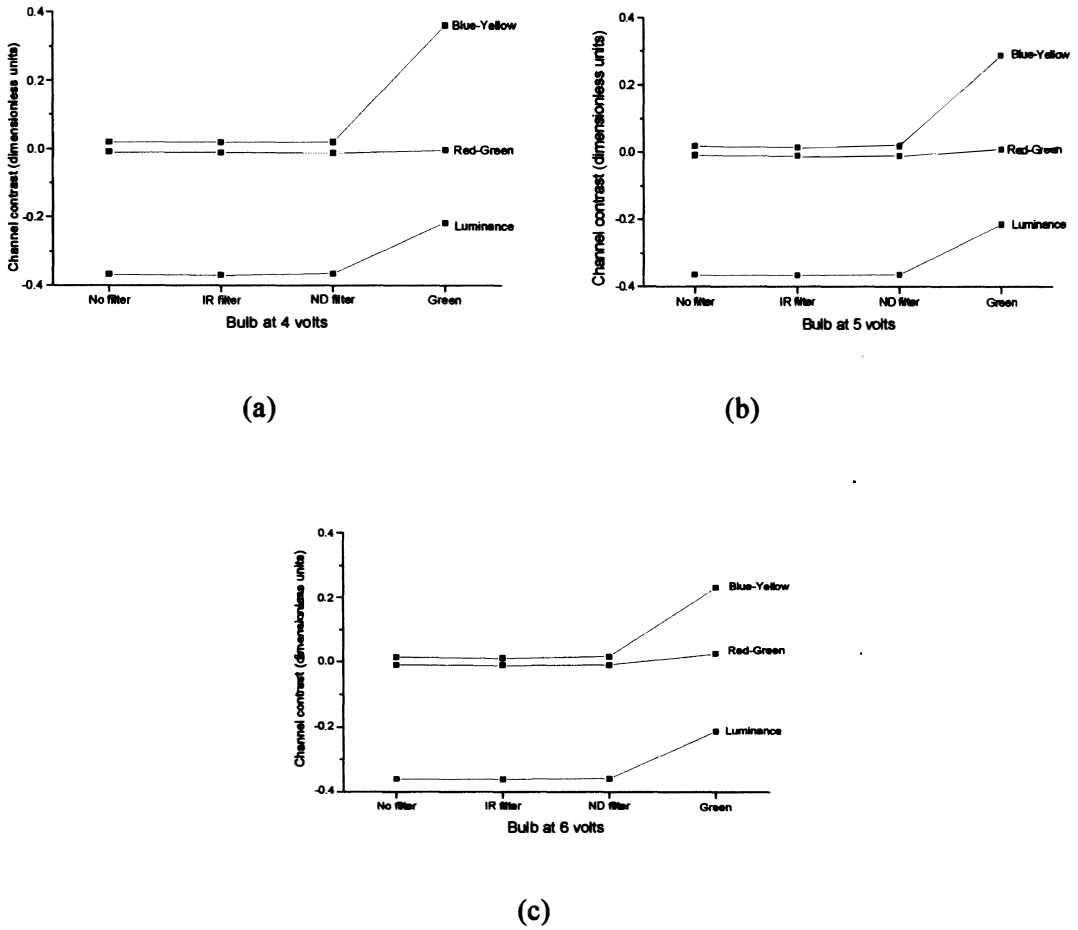

Fig. 2. *The contrast of a haemoglobin-filled microaneurysm seen against the fundus in the luminance, red–green and blue–yellow channels of a young observer: (a), (b), (c) for 4, 5 and 6 volts respectively.*

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.

References

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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.