Firstly, all the discolorations previously published were brown, mostly from USA, Japan, and Europe. In contrast, we report a greenish discoloration, from Asian subcontinent where weather is extreme in nature. It is too hot, humid with bright sunshine in summer and too cold in winter. Socially, the housewives spend much time in kitchens where exposure to heat is immense and inevitable. The exposure to these extreme temperatures may cause change in colour of the IOL. A manufacturing defect cannot be ruled out nor can the inherent problem with the silicone material used, maybe with some impurities.

We yet do not know how much the IOL will further gain greenish colour or deteriorate the vision of the patient. In both the extreme situations, the affected IOL has to be replaced with a newer one. The analysis of explanted IOL (if removed at any time in future) may be helpful in determining the exact cause of this green discoloration. Till then, the question rose by Milauskas in 1991 regarding implantation of silicone IOLs in human beings holds ground and the safety and efficacy of their use need reassessment.

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Sir,

Concurrent *vs* dependent retinal arteriolar occlusion and central vein occlusion

Ozdek *et al*[1] report details of two patients with hyperhomocysteinaemia who presented with nonischaemic occlusion of the central retinal vein (CRV) and putative concurrent occlusion of a branch retinal arteriole. They make reference to an earlier description of seven eyes similarly affected by CRV occlusion and segmental inner retinal infarction[2] in which cilioretinal infarction, secondary to the CRV occlusion,[3,4] was discounted as the mechanism of ischaemia. However, a single vascular luminal obstruction is more likely than two simultaneous (but separate) occlusions, especially in young patients. The possibility of cilioretinal infarction should be seriously entertained, therefore, even if this implies that up to half of the retinal circulation must perforce derive from posterior ciliary branches of the ophthalmic artery. Indeed, in Case 1 of this latest report,[1] the inferotemporal vessel supplying the territory of the infarct appears to have a 'hook' characteristic of a cilioretinal arteriole as it emerges from the disc rim.

In CRV occlusions, the distinction between a (simultaneous) branch arteriolar occlusion and a (consequential) cilioretinal arteriolar occlusion is not merely of academic interest. Recognition of the dependent association will spare the patient from unnecessary investigations, including the search for a source of arteriolar embolism. Moreover, therapeutic lowering of the intraocular pressure (as attempted in Case 1) runs the risk of precipitating retinal haemorrhage by increasing transmural hydrostatic pressure gradients, without any prospect of improving inner retinal perfusion.[5]

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Sir,

Reply to McLeod

We would like to thank to Dr. McLeod for his constructive contributions to our paper describing retinal vascular occlusions in two cases with homocystinaemia.[1] In our paper, we have primarily focused on the finding of homocystinaemia as a cause of vascular occlusions in young patients; however, we have not discussed the probable mechanisms of association of arterial and venous obstructive disease. Three different clinical syndromes have been suggested describing simultaneous arterial and venous obstructions of the retina including; the combination of central retinal artery and vein obstruction; combined occlusion of central retinal vein and cilioretinal artery; and combined branch retinal artery and central retinal vein obstruction.[2] We agree with Dr McLeod in that the arterial obstruction may actually be a relative hypoperfusion of the cilioretinal arteries secondary to increased retinal venous pressure.[3,4] Although this dependent occlusion is the most probable explanation, therapeutic lowering of the intraocular pressure was attempted in the presented case considering the other possibilities like two simultaneous (but separate) occlusions which may be the case in a patient with homocystinaemia.

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Sir,

Diode laser trans-scleral cyclophotocoagulation in the management of glaucoma in patients with long-term intravitreal silicone oil

We read with great interest the article by Sivagnanavel *et al*.[1] We will be most grateful if the authors can help to clarify a few points.

In this series, the authors described a significant number of subjects losing vision after trans-scleral cyclophotocoagulation (TSCPC). They felt that the loss was not quantifiable because of the poor visual acuity. They concluded that TSCPC failed to protect against visual loss in the long term. We may like to be aware that it is the natural course of their disease to lose vision. Hence, they may like to compare with the rate of visual loss in the TSCPC-treated group with a control group having similar glaucoma associated with long-term silicone oil placement, and who were treated with other modalities. It may be of interest for the authors to attempt quantification with Logmar (Logarithm of Minimum Angle of Resolution) chart, or express the percentage of eyes losing 2 or more lines.

If the eight cases of ocular comorbidity were excluded, the success rate can actually be raised to 50%. As the authors rightly pointed out, the prolonged placement of silicone oil before treatment (mean duration of oil before TSCPC was 33.7 months (range 1–113 months, SD = 26.9)), and prolonged duration before successfully bringing down the IOP (53% of the patients took 450 days to reduce the IOP to below 21 mmHg), might be factors contributing to the low overall success rate. We may like to be aware of the fact that the success and failure definitions were slightly different among different studies so that the rates may not be accurately comparable with each other.[2]

The authors did possess evidence in support of the efficacy of the TSCPC: the average number of IOP-lowering medications prior to TSCPC was 2.6 (range 1–5). This was reduced to 1.0 (range 0–3) following TSCPC at final follow-up. Moreover, the authors regarded the procedure to be very safe with few side effects. At the time of writing, as far as we know, there is an absence of strong data in the literature supporting other modalities of treatment in the same