fact that the affected vessel filled with fluorescein dye at 9.8 s. This was well after the choroidal flush of the angiogram when a cilio-retinal vessel would have filled with dye. This time (9.8 s) is consistent with the early arterial phase of the fluorescein angiogram.[1] A closer inspection of Figure 3a also shows the proximal inferotemporal, superonasal and inferonasal arterial branches containing fluorescein simultaneously with the affected vessel. This indicates that the phase difference of the filling vessel and the other retinal arterial branches was not very significant, as would be the case in cilio-retinal and central retinal vessel filling. This difference is usually in the order of 1–3 s.[1] All four major retinal arterial branches filling with dye simultaneously is more consistent with a common origin from the central retinal artery as opposed to an origin from the posterior ciliary (in the case of cilio-retinal arteries) and central retinal arteries, in which the time difference of dye filling is at least 1–3 s.[1] This was not observed in this case where the time difference was not measurably more than 1 s.

The affected vessel also traces back to the bifurcation of the major retinal vessels (Figure 3a–c) at the centre of the optic disc head. This is readily visible in Figure 3 in spite of the presence of a dilated central retinal vein. Cilio-retinal arteries usually arise from the rim of the optic cup at the lateral border of the optic nerve,[2,3] which was not observed in this patient.

The occluded vessel filled with fluorescein before any other branch, but this phase difference was very small, as mentioned earlier.[4,5] Ordinarily, this should not be so, and in the light of the relatively short phase difference of the dye front, the difference in perfusion pressure between vessels would have to be small. Perhaps the explanation could be anomalous branching of the main central artery with this particular arterial branch arising proximally within the optic nerve head before other branches were given off. This could explain why this vessel filled with dye just before the other main branches.

Central collateral branches of the central retinal artery are known to occur. These vessels arise proximally within the optic nerve and may pass with the central artery towards the lamina cribrosa.[2] It has already been pointed out that this can only be confirmed anatomically. However, there is an anomalous pattern to the arterial branching in this eye at the optic nerve head, and the optic disc in the affected eye was smaller compared to the disc on the other side. A proximal branching origin would also support the theory of vascular compression from optic disc swelling, since the said vessel would have to traverse the lamina cribrosa of the optic nerve, where compression would be likely in a swollen optic disc. Anomalous branching of retinal arteries is a rare but recognised cause of arterial occlusion in the eye.[6] This was also the explanation given as to the possible aetiology in this case report, and the possibility of anomalous or collateral arterial branching should be considered.

The effect of retinal vessel autoregulation may also have contributed to the affected vessel filling slightly ahead of the other vessels. In the presence of hypoxia, as for example in an area of arterial occlusion, metabolic autoregulatory mechanisms in retinal vasculature lead to an accumulation of vasodilatory metabolites.[3] As a consequence, arteriolar tone is reduced and vascular resistance reduces accordingly.[3] This would result in a greater relative blood flow in this particular vessel, allowing it to fill ahead of the other retinal branches. Choroidal blood flow is not autoregulated.[3]

Despite thorough investigations, the exact pathophysiologic mechanism of the vascular insult in this particular case remains unknown.

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Sir,

Orbital cellulitis following corneal gluing under sub-Tenon's local anaesthesia

Regarding the case report by Redmill *et al*,[1] it should not be intuitively surprising that purulent orbital cellulitis resulted from a sub-Tenon's local anaesthetic given in the presence of active corneal ulceration. The authors do not

state whether corneal scrapings were taken or what the culture results of the scrapings were. The fact that they administered topical ofloxacin, however, suggests at least a clinical suspicion of active infection.

My personal experience suggests that sub-Tenon's or other invasive local anaesthetic are not necessary for the procedure of corneal gluing. This can be accomplished quite satisfactorily using topical anaesthesia in every case with which I have dealt. (A highly uncooperative patient would generally be unsuitable for gluing since they also presumably would be uncooperative following the procedure, with a risk of eye rubbing.)

Perhaps, therefore, the main lessons to be drawn from this case report are, firstly, that if invasive local anaesthesia is not necessary, it should not be used, and secondly, that it should be used with extreme caution in the presence of suspected or proven active infection of the external eye.

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Sir,

The article by Shah *et al* (*Eye* **15**; 616–620) does a service to ophthalmologists by drawing our attention to the existence of this distressing visual symptom in the eyes of young adults with advanced glaucoma. I would concur that the simplest explanation would be that of a circulatory 'steal'. It is worth remembering that the phenomenon is not restricted to this age group, and is probably to be found more frequently among the elderly. In this older age group, not only exercise (of such a trivial nature as climbing stairs) but also hot baths have been reported to me as causing the same symptom. Avoidance of the causative action, aspirin, and stricter IOP control appear to be the safest remedies on offer to reduce the frequency of attacks.

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Sir,

The Royal College of Ophthalmologists Cataract Surgery Guidelines: what can patients see with their operated eye during cataract surgery?

I am indebted to Mr Au Eong for pointing out some inaccuracies in the Information for Patients (Appendix 1) section of the Royal College of Ophthalmologists Cataract Surgery Guidelines and for documenting the clinical studies on the subject.[1] It is of course correct to say that patients *may* lose some or all of their vision after receiving local anaesthetic, particularly if given by the retrobulbar or peribulbar route, and during surgery they may experience a number of visual sensations, most usually a variety of colours.

The challenge however when writing patient information is to keep the advice clear and succinct for the majority, and yet not misleading.

Patients *do* need to be reassured that they are not going to 'see the operation' in detail, and warned that they will be dazzled (in the majority of cases, 100% with topical anaesthesia) by an extremely unpleasant bright light at the beginning of the operation. It is these aspects that are most likely to cause concern without prior warning.

Happily nowadays the more invasive forms of local anaesthesia are becoming increasingly rare in the UK, and hence the advice is most relevant for patients undergoing sub-Tenon's or topical anaesthesia.

I agree that there is a place for additional preoperative counselling in order to cover more completely the variety of sensations that the patient may experience so as to allay potential fears. Once suitably reassured, however,