of the globe would shift the shaft of the needle again 7 mm away from the globe at the point of entry. Concerning the 'wide sweeping action [of the needle] pivoting at the point of insertion, maintaining the needle as close to the globe as possible' in order to detect engagement of the globe, this manoeuvre could not possibly allow the needle tip to remain in the sub-Tenon space, for it would not seem plausible that a virtually spherical space would allow such gross rectilinear motion. But it is possible that the repository steroid is kept much closer to the globe than with an orbital floor injection, probably in the anterior intraconal space.

I also wonder as to the mechanism of the levator palpebrae aponeurosis disinsertion in the two cases which developed ptosis after treatment, for neither a lid speculum nor a bridle superior rectus suture were used. However, mild ptosis is a well-documented side-effect of topical and periocular steroid use.[4,5]

Lastly, grouping 10 retrospective patients along with 18 prospective ones may not be statistically acceptable.

Placing a drug in the sub-Tenon space should be done in a safe and predictable manner. Probably the procedure to follow is to use a blunt curved sub-Tenon cannula after opening and identifying this space under appropriate magnification. The drug may then be stored in the newly formed sub-Tenon's pocket for weeks, rather than it dispersing and being taken away by the blood vessels of the orbital fat, whether it is in the anterior intraconal or orbital floor space. The high dose and longer time of the drug directly in contact with the ocular tissues may be responsible for a more intense and longer healing response and possibly fewer anterior segment, orbital and systemic adverse effects.

This predictable technique would allow us not only properly to compare the action of the different injection modes available at present but also reduce the tendency for the more complicated, risky or harmful treatment modalities and injection sites to be used in the treatment of inflammatory conditions in the posterior segment of the eye.

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Sir,

Mr Sajnani raises several issues related to sub-Tenon's steroid injections in his letter relating to our recent paper.[1]

We feel the diagram drawn by Sajnani is misleading as the needle he illustrates is directed posteriorly with no attempt to follow the globe curvature. We emphasise that the point of insertion of the needle should be as far posteriorly as can be easily visualised and the tip of the needle should be maintained as close as possible to the sclera. We feel that even in those cases where the steroid may inadvertently be placed in the anterior intraconal space, transscleral absorption of steroid is probably greater than if the steroid were injected using an orbital floor approach.

It was not our intention to provide a detailed study of the echographic localisation of periocular steroid injections and for further information the reader is referred to Freeman *et al.*[2] as referenced in our original paper. These authors provide echographic evidence of the localisation of steroid injections to the sub-Tenon's space in the majority of cases and were able to identify those injections which passed through the sub-Tenon's space into the intraconal space (1 of 18 injections given via the superotemporal approach using a technique identical to ours).

It is possible to make a surgical incision in the inferotemporal fornix and then inject triamcinolone using a blunt sub-Tenon cannula. However, many patients with uveitis will require multiple injections and we suspect the requirement for repeat incisions will result in increased scarring, making subsequent injections extremely difficult. Furthermore, the use of a larger entry site may result in premature leakage of triamcinolone from the posterior sub-Tenon's space, decreasing its efficacy and potentially causing problems with wound healing. Although any technique which decreases the risk of globe perforation is obviously desirable we can find no reference in the literature to this technique being employed.

The rationale for data presentation and statistical analysis of combined results from the prospective and retrospective series is outlined in the patients and methods section of our original paper.

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Sir,

I read the article on genetic predisposition to ocular melanoma by Houlston and Damato[1] with great interest. A component of their argument that 'host factors play a greater role in the development of ocular melanoma than in cutaneous melanoma' is that there is 'little variation in the incidence of uveal melanoma within and between countries'. This is certainly the case for the age-standardised mortality rates for England and Wales[2] but surprisingly not the case for the United States of America, where there has been a 58% fall over the period 1955–1989.[3] The constant rates for England and Wales but falling mortality rate in the USA is explicable in terms of the underlying incidence rates for cancer of the eye, which show a 25% fall for the period 1973–1989 for the USA but are remaining constant for England and Wales.[3] This observation of falling mortality rates for ocular melanoma has also been extended to Canada.[4] This relatively rapid and large change in rates argues for an important environmental factor at work.

Houlston and Damato's paper provides further good evidence for a sub-group of uveal melanomas having a genetic basis, but it is a small sub-group. Amalgamating three large series, numbering 9000 patients, only 2% have a family history of a relative with uveal melanoma, and then it is usually only one other family member,[5–7] suggesting that while genetic factors are important they account for only a minority of cases of uveal melanoma.

The nature of the environmental factor(s) causing uveal melanoma is obscure and it almost certainly is not

ultraviolet light.[2] It is certainly worthwhile to identify high-risk groups and offer screening, but it would also be helpful to identify the relevant environmental factor(s) as well.

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Sir,

The purpose of our article[1] was to highlight the increasing evidence that some cases of uveal melanoma arise as a result of a genetic predisposition. We do not think that we overstated the role of constitutional gene mutations in the development of uveal melanoma. Although as Foss points out only 2% of cases are familial as defined by having a relative affected with uveal melanoma, indirect evidence suggests that a greater number, around 7% of cases, are likely to be caused by mutations in genes with pleiotropic effects such as *BRCA2* and those causing the atypical naevus syndrome.

To assess the contribution of germline mutations to the development of uveal melanoma we have made a systematic collection of family histories, blood samples and tumour material from over 400 patients attending the Ocular Oncology Service in Liverpool. Using this resource we are currently investigating the contribution of mutations in *BRCA2* and *CDKN2A* to uveal melonoma by screening both these genes in this series of blood samples from these patients.

Clearly the identification of genetic factors does not detract from the potential role of specific environmental factors in the aetiology of uveal melanoma, and in this respect we concur with the view of Foss.

Reference

1. Houlston R, Damato B. Genetic predisposition to ocular melanoma. Eye 1999;13:43–6.

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Sir,

I have read with interest the paper by Zambarakji *et al.*,[1] and subsequent correspondence,[2] concerning capsulorhexis phymosis following phacoemulsification. My own experience illustrates another possible parameter in the estimation of capsulorhexis phymosis.

A healthy 70-year-old man underwent phacoemulsification via a 5.0 mm capsulorhexis. The procedure was uncomplicated apart from a small (2 clock-hours) zonular dehiscence. A 23.0 D acrylic intraocular lens (Acrygel, Corneal Laboratoire) was implanted into the capsular bag.

Post-operative acuity was 6/9, and further changes were as shown in Table 1 (periods of subnormal best corrected visual acuity were assumed to be due to cystoid macular oedema, not proven by fluorescein angiography, but which improved with the use of oral acetazolamide and topical betamethasone). At this point capsulorhexis phymosis was noted, and nine radial relaxing incisions were made in the anterior capsular ring with a YAG laser. The posterior capsule was left intact. The refraction and best corrected acuities following YAG laser are shown in Table 2.

Table 1. *Post-operative refraction and best corrected acuity*

Post-operative time (weeks)	Refraction	Best corrected acuity
4	+2.00/—	6/18
8	0.00/–1.50 × 90°	6/9
20	+7.00/–2.00 × 150°	6/18

The only logical explanation for this remarkable variation in post-operative refraction is posterior displacement, or posterior bowing, of the flexible intraocular lens caused by capsulorhexis phymosis and relieved by YAG laser relaxing incisions. Previous literature has alluded to the fact that capsulorhexis phymosis can alter refraction,[3] and Shammas[4] has measured such changes in refraction. In one case, he reports +1.25 D, and in another, +0.75 D of induced hyperopia.

My experience indicates that accurate refraction may help in monitoring some cases of capsulorhexis phymosis, especially if a foldable intraocular lens is used.

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Table 2. *Post-YAG laser refraction and best corrected acuity*

Post-YAG laser time (weeks)	Refraction	Best corrected acuity
2	+4.00/–1.50 × 180°	6/18
7	+1.75/–0.25 × 180°	6/6
26	+1.00/—	6/6