Sir,

I read with interest the article 'Primary orbital Ewing's sarcoma: report of a case and review of the literature' by Dennis S.C. Lam *et al.* (*Eye* 1999;13:38–42), and wish to comment on it. The authors entitled the article 'primary orbital Ewing's sarcoma' in spite of the fact that it is clear from the CT scan that there is involvement of the ethmoid sinuses bilaterally as well as the floor of the anterior cranial fossa and orbit. Since the bones do not appear to be significantly eroded in the roof of the orbit, it is my assumption that this lesion was in fact of primary origin from the nasal sinuses, which has extended bilaterally into the orbits and to the intracranial cavity through the very thin bones of the ethmoids. It would be hard to call it a bilateral primary that had invaded the other way, in view of the fact that the bony structures appear relatively intact. In addition to this, the authors themselves mention that the lesion was biopsied from the nasal sinuses. I would certainly have classified this as a secondary tumour of the orbit arising from the nasal sinuses.

In addition, I would like to correct the statistics that the authors have offered in so far as they mention seven cases reported in the literature. I have reported two in the past, and they should be included in their survey. Both of them occurred in young children and both involved the sinus and orbit. They have gone on to successful cure of their disease through combined chemotherapy and radiotherapy with a follow-up of 17 years and 10 years each.

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Sir,

We are grateful to Professor J. Rootman for his interest and comments on our paper.[1] We failed to include the two cases described in his textbook[2] in our original report as we could not find them through the computerised literature search utilising the database of Medscape Medline. We would have included these cases should we have known about them since they would have enabled us to have a more complete picture of orbital Ewing's sarcoma.

A very important point of our article is the non-surgical approach in the management of orbital Ewing's sarcoma.[1] Both of Professor Rootman's cases, like ours, also had excellent outcome after combined chemotherapy and radiation therapy. Since the publication of our article, Choi *et al.*[3] have reported another case of primary orbital Ewing's sarcoma successfully treated with combined chemotherapy and radiation therapy. This additional information further establishes the effectiveness of combined chemotherapy and radiation therapy in the management of orbital Ewing's sarcoma. This is encouraging, since patients with orbital Ewing's sarcoma can have a good prognosis while avoiding extensive surgery, which often results in significant morbidity including mutilating cosmetic effects.

With regards to the origin of the tumour in our patient, Professor Rootman is of the opinion that it is more likely to have originated from the nasal sinuses as the orbital roof did not appear to be eroded. This is certainly a possibility. However, there have been reported cases of primary Ewing's sarcoma of the orbit in which the orbital roof was intact.[4] Therefore, in our patient, even though there was no orbital roof erosion, the tumour could still have arisen from the orbits, with extension involving the ethmoidal sinuses and anterior cranial fossa.

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Sir,

I read with interest the article by Tanner *et al.* on posterior sub-Tenon's triamcinolone injections in the treatment of uveitis,[1] but I wonder whether sub-Tenon injection is indeed sub-Tenon. Indeed the authors were unable to locate the repository drug with the B-scanner and claim the drug used was 'echolucent'; but they also failed to find echo-free sub-Tenon's space occupied by the drug, or the echo-opaque fascia of Tenon.

The fascia bulbi (capsule of Tenon) posteriorly around the optic nerve is pierced by the ciliary vessels and nerves, and becomes very thin; it can be traced only with difficulty to the dural sheath of the optic nerve.[2] Crossing the episcleral space and attaching the fascial sheath to the sclera are numerous delicate bands of connective tissue.[3]

As illustrated in Fig. 1, where actual dimensions of eye globe and 25G needle (5 8 inch, 16 mm) used have been respected, with a straight needle it is not possible to remain in a spherical sub-Tenon space using the method discussed by Tanner. As the needle is advanced up to its hub while keeping it firmly against the globe, its tip moves at least 7 mm away from the posterior curvature of the eye; alternatively keeping the tip of the needle adjacent to the posterior surface

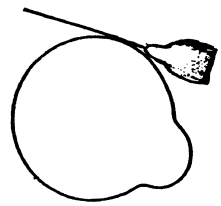

Fig. 1. *A 25G needle ($^5/_8$ inch, 16 mm) on a 24 mm axial-diameter globe.*

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