operatively. If this mechanism is correct, there are implications for wound closure in ophthalmic surgery. Over recent years it has become more common to leave conjunctiva to grow back over a scleral wound or blow it back with an injection of subconjunctival antibiotics rather than suture it carefully. An alternative pathogenesis is that local ischaemia caused by suture or cautery[2] could have caused conjunctival necrosis resulting in the appearance of conjunctival retraction and dellen formation. This may have encouraged tear- or blood-derived leucocytes to produce a pool of immune mediators such as matrix metalloproteases (MMP 8 and 9) released from leucocytes causing necrotising scleritis.

The early presentation of these cases at 1, 3 and 4 weeks post-operatively supports the influence of such peri-operative surgical factors. In our own experience, and those of others, the majority of such cases of SINS present months if not years after surgery, suggesting other aetiologies such as molecular mimicry/cross-reactivity between ocular antigens and remote tissue or microbial antigens.[2] The patients described in this paper may represent a subgroup of SINS with an earlier presentation and a good response to local surgical treatment.[1] However, we believe that this group should be placed in the context of later-onset SINS where early systemic immunotherapy with prednisolone and cyclophosphamide remains the mainstay of treatment.[2] Systemic treatment not only rapidly alters the course of ocular disease but may save life when the scleritis is part of a systemic vasculitis.

References

1. Mansour AM, Bashshur Z. Surgically induced scleral necrosis. Eye 1999;13:723–4.
2. O'Donoghue E, Lightman S, Tuft S, Watson P. Surgically induced necrotising sclerokeratitis (SINS): precipitating factors and response to treatment. Br J Ophthalmol 1992;76:17–21.

Andrew Ramsay ✉
John Dart
Moorfields Eye Hospital
City Road
London EC1V 2PD, UK

Sir,

The comments of Ramsay and Dart are very well taken. They differentiate early-onset scleral melt due to conjunctival retraction from the late-onset vasculitic scleritis. It is of note that the entity described in our article is rare. More common causes of scleral melt include forceps-induced scleromalacia due to non-gentle holding of the eyeball (especially in beginning trainees) and keratoconjunctivitis sicca. Causes of scleral melt in a teaching university setting are, in order of decreasing frequency: (1) trauma, (2) keratoconjunctivitis sicca, (3) vasculitis, (4) conjunctival retraction – dellen complex.

A.M. Mansour ✉
Z.F. Bashshur
American University of Beirut Medical Center
PO Box 113
6044 Beirut, Lebanon
Tel: 354911 350000
Fax: 961 1 345325

Sir,

We read with interest the article in the August 1999 issue of *Eye*, 'Peroperative retinoscopy as a predictor of final post-operative refraction'.[1] It has close parallels with a study we published in the *British Journal of Ophthalmology* in July 1998.[2] We feel that 'immediate post-operative' is a more accurate description than 'per-operative': retinoscopy was undertaken immediately after the operation in both studies.

We also found that following phacoemulsification surgery, immediate post-operative objective refraction can be performed satisfactorily in most cases. Whereas Tappin and Ferguson compared the accuracy in prediction of post-operative refraction by immediate post-operative retinoscopy and biometry and found the former to be significantly better, we looked at the refractive change from the immediate post-operative period to the final refraction. For the particular implant type used in the study (Chiron C10UB) we found a statistically significant refractive change with a mean hypermetropic shift of 1.11 D which can then be taken into account if immediate implant exchange is considered.

References

1. Tappin MJ, Ferguson VMG. Peroperative retinoscopy as a predictor of final post-operative refraction. Eye 1999;13:559–61.
2. Tu KL, Gaskell A. Immediate postoperative objective refraction as an indication to final refraction in phacoemulsification surgery. Br J Ophthalmol 1998;82:842.

Kyaw Lin Tu ✉
Ophthalmology Department
Ward A4
Stepping Hill Hospital
Stockport SK2 7JE, UK

Alan Gaskell
Ayr Hospital
Ayr KA6 6DX, UK

Sir,

We thank Tu *et al.* for their comments on our paper.

We agree that in some respects the two papers are similar; however, we attempted to compare per-operative retinoscopy with the refraction predicted by pre-operative biometry. Retinoscopy was thus used as a predictor of immediate post-operative refraction, rather than for recording the change in post-operative refraction during the first 6 weeks after surgery. We think that per-operative rather than post-operative refraction is a more accurate term owing to the fact that the retinoscopy was performed while the patient was still draped and a sterile surgical field was maintained. If the retinoscopy indicated a very different refraction to that expected from the biometry then an implant exchange could be carried out immediately.

We note that Tu *et al.* found a hyperopic shift of 1.1 D from the time of surgery (in which a plate haptic implant was used) until 6 weeks post-operatively. In our study with flexible haptics there was also a change in refraction during the 6 week period following surgery; there was a mean error of +0.55 D. We think this was due either to a systematic error in the retinoscopy or to a change in the position of the lens.

Michael J. Tappin ✉
Veronica M.G. Ferguson
Moorfields Eye Hospital
City Road
London EC1V 2PD, UK

Sir,

We much appreciated Choong *et al.*'s clinical study,[1] introducing a new protocol for the management of acute angle closure glaucoma. Nevertheless there is an apparent discrepancy as the authors suggest commencing stage 2 treatment (osmotic agents) one and a half hours after the administration of the stage 1 treatment (includes oral Diamox), while declaring that the maximum effect of the latter is exhibited in 2 h.

As the protocol is based on theoretical considerations rather than good randomised controlled trials of glaucoma treatment, would it be best to treat according to the known effects of the drugs concerned?