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.

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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.

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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?

Reference

1. Choong YF, Irfan S, Menage MJ. Acute angle closure glaucoma: an evaluation of a protocol for acute treatment. Eye 1999;13:613–6.

Elizabeth Patsoura ✉
J.M. Butcher
Ophthalmology Department
Countess of Chester Hospital
Liverpool Road
Chester CH2 1UL, UK

Sir,

The maximum effect of oral Diamox occurs at 2 h but the intravenous Diamox is much faster. If no reduction in intraocular pressure has occurred an hour and a half after the intravenous Diamox then it is not unreasonable to consider further treatment at that stage. After all this condition is extremely painful with potential to damage the optic nerve, and a rapid reduction in intraocular pressure from the very high levels exhibited by some patients is desirable.

Mr M.J. Ménage, MRCP, FRCS, FRCOphth ✉
Department of Ophthalmology
The General Infirmary at Leeds
Great George Street
Leeds
West Yorkshire LS1 3EX, UK
Tel: +44 (0)113 243 2799
Fax: +44 (0)113 392 6336

Sir,

We read with interest the editorial[1] and article[2] on the immediate management of acute angle-closure glaucoma (AACG). We would, however, like to propose an alternative: argon laser peripheral iridoplasty (ALPI).[3,4] ALPI was traditionally employed only when medications failed to control intraocular pressure (IOP),[5] but we found it effective and safe as an initial treatment.

Immediate ALPI may be more effective than conventional medications in lowering IOP. Employing the same criteria of 'satisfactory IOP control' proposed by Choong *et al*.[2] we achieved satisfactory IOP control in 83.3% of AACG patients, at just 15 min after ALPI.[3,4] At 120 min after ALPI we achieved satisfactory IOP contol in 100% of patients. We have, however, excluded patients presenting more than 48 h from the onset of symptoms.

We have also documented, using ultrasound biomicroscopy, re-opening of the closed drainage angle immediately following ALPI.[3] By opening up the angle promptly, we would expect a lower chance of a patient developing peripheral anterior synechiae, and subsequently chronic angle-closure glaucoma.

Immediate ALPI in the management of AACG appears to be a safe procedure. It will certainly spare patients the systemic side effects of acetazolamide, glycerol or mannitol. We have followed up the 18 reported cases for 2 years by now, and so far we have not come across any patient with corneal decompensation or scarring, iris atrophy or necrosis, or other complications that could have arisen from the ALPI. We have also shown, in another study, that halving the quantity of ALPI still yields IOP-lowering results comparable to the conventional 360° treatment.[6] By applying ALPI to only 180° of the peripheral iris, the risk of complication from ALPI is further lowered.

After ALPI, all patients would still require a peripheral iridotomy to break the pupillary block, and to avoid recurrent attack of acute glaucoma. Most commonly, the peripheral iridotomy has to be delayed until the cornea recovers from the acute attack. With the use of immediate ALPI, all corneas clear within 2 h,[3,4] allowing a very early peripheral iridotomy to be performed.

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Dennis S.C. Lam
Department of Ophthalmology & Visual Sciences
The Chinese University of Hong Kong
Hong Kong Eye Hospital
Kowloon, Hong Kong

Jimmy S.M. Lai
Clement C.Y. Tham
Department of Ophthalmology & Visual Sciences
The Chinese University of Hong Kong
Prince of Wales Hospital
Shatin, Hong Kong

Professor Dennis S.C. Lam ✉
Department of Ophthalmology & Visual Sciences
The Chinese University of Hong Kong
Hong Kong Eye Hospital
147K Argyle Street, Kowloon
Hong Kong
Tel: +852 2762 3157
Fax: +852 2711 0464
e-mail: dennislam@cuhk.edu.hk