Sir,

We read with interest Mr Abu El-Asrar's article on giant retinal tears after pars plana vitrectomy.[1] He stated that although patients 1 and 3 underwent pars plana lensectomy besides vitrectomy, complete removal of the vitreous base could not be performed initially. To accomplish maximum removal of basal vitreous gel, a lensectomy must be performed in most phakic eyes as trauma to the lens is very likely when working in the anterior vitreous. On the other hand, tractional bands are formed between peripheral iris structure and vitreous gel due to the anterior chamber reactions in penetrating injuries. Exposure of these structures is only possible if deep scleral depression is done. It is obvious that incomplete removal of peripheral vitreous base creates much higher spontaneous giant retinal tear incidence than Freeman's suggestion.[2]

As giant retinal tears are always associated with posterior vitreous detachment, retinal pigment epithelial cells and other components of the fibrocellular proliferation cascade can gain easy access to the retinal surface and form epiretinal membranes, leading to proliferative vitreoretinopathy (PVR).[3] We think 360° cryotherapy was very extensive for patients 2 and 4 and it could cause haemorrhage, submacular pigmentary clumping and increase PVR. We suggest two or three rows of contiguous laser photocoagulation to the margins in the treatment of giant retinal tears, as described previously.[4,5]

Both silicone oil and gas tamponades offer high rates of retinal reattachment in the management of giant retinal tears. However, in general, giant breaks of 180° or less can usually be managed with gas tamponade, while breaks larger than 270° are probably best managed with silicone oil.[5] Gas tamponade would be appropriate for patients 2 and 4.

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Cosar Batman, MD ✉
Osman Cekic, MD

Division of Vitreoretinal Surgery
SSK Ankara Eye Hospital
Ulucanlar
Ankara
Turkey

Sir,

I thank Drs Batman and Cekic for their interest regarding my report on 'Giant retinal tears after pars plana vitrectomy'.[1] It is undoubtedly very important that excision of the vitreous gel in the periphery is performed. However, young patients frequently have incomplete posterior vitreous separation interfering with complete removal of the vitreous gel, and detachment of the residual vitreous is noted in the post-operative period as seen in the patients included in this report. In addition, patient 3 underwent diagnostic and therapeutic vitrectomy to treat post-traumatic endophthalmitis. Drs Batman and Cekic would appreciate that trying to make total vitrectomy in endophthalmitis is hazardous in the presence of inflamed retina. Vitrectomy in these cases is confined to the central portion of the vitreous to avoid placing any stress on the retina.

Cryotherapy was applied to the edge of the retinal flap when the retina was flat using perfluorocarbon liquid (PFCL). This minimises dispersion of retinal pigment epithelial cells into the subretinal space. A potentially harmful effect of cryotherapy is dispersion of viable retinal pigment epithelial cells.[2] Therefore, a meticulous lavage of the vitreous fluids is performed around the PFCL bubble to remove the dispersed retinal pigment epithelial cells and debris after cryotherapy. There is vast clinical experience indicating that cryotherapy is effective and safe in treating most eyes with retinal detachment. Therefore, many surgeons continue to use cryotherapy in the management of giant retinal tears.[3] Currently, I frequently use the indirect ophthalmoscope to deliver three or four rows of laser photocoagulation to posterior retinal flap and posterior to ora serrata in the fundus periphery not involved in the giant tear. The outcome of the two treatment modalities is similar.

The third issue raised by Drs Batman and Cekic concerns my use of silicone oil as an internal tamponade in patients 1 and 4. Some form of internal tamponade is necessary to keep the retina in position while chorioretinal scarring takes place. A long-acting gas or silicone oil tamponade is used depending on the surgeon's choice and experience. I prefer, like others,[3-6] temporary tamponade with silicone oil. There are several advantages of using silicone oil compared with gas in the management of giant tears: during PFCL–silicone oil exchange, slippage of the posterior edge of the giant tear is less likely than with fluid–air exchange; silicone oil provides excellent clarity of the media throughout the course of the exchange; use of silicone oil avoids maintaining a prolonged head-down position; finally, the excellent clarity of the media provided by silicone oil allows laser photocoagulation augmentation in the post-operative period if needed. The complications related to silicone oil are avoided by earlier removal of silicone oil, at about 6–8 weeks after the operation.

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Ahmed M. Abu El-Asrar ✉

Department of Ophthalmology
King Abdulaziz University Hospital
PO Box 245
Riyadh 11411
Saudi Arabia

Sir,

We would like to respond to Mr Leyland's letter (Eye 1997;11:428-9) concerning the experience of other ophthalmic units with CS gas ocular injury. Over the past two years we have seen a number of cases of CS (*O*-chlorobenzylidene malononitrile) and 'Mace' (chloroacetophenone) gas ocular injuries following illegal use in assaults.

In our experience the symptoms are transient and rapidly reversible, rarely persisting more than 24 h, and are followed by complete recovery, as described previously.[1,2] Patients usually experience intense blepharospasm and lacrimation with conjunctival injection and occasionally corneal punctate epithelial erosions. Treatment consists of aerating the ocular surface and we achieve this by placing the patient in the open space of the hospital grounds with no attendants or individuals downwind. Irrigation leads to reactivation and vaporisation of the CS gas exacerbating symptoms and placing staff at risk. The CS or Mace agent is delivered as an aerosol from a powder form. Particulate matter directed at close range can lead to powder infiltration and mechanical injury.[2,3] We have not experienced this complication, which can have serious ocular consequences including conjunctival cicatrisation, corneal ulceration, scarring and vascularisation. This is particularly associated with chloroacetophenone (Mace), which is caustic,[2] and so a slit lamp examination is mandatory following such injuries. It is suggested after aeration to irrigate with cold isotonic saline and remove particulate matter from the conjunctiva with cotton pledget and corneal particles with a needle tip at the slit lamp.[2] Careful handling of contaminated clothing is required as exposure to water can vaporise CS gas placing the patient or staff at risk of further injury.

Non-ocular injury can produce serious pulmonary sequelae[1,4] and in our experience this outweighs the morbidity induced by the ocular effects, which have been short-lived with complete recovery. Further experience in dealing with these injuries may reveal more serious complications.

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C.F. Willoughby, FRCOphth ✉
B. Ilango, FRCS
A. Hughes, RGN OND

St Paul's Eye Unit
Royal Liverpool University Hospital
Prescot Street
Liverpool L7 8XP
UK

Sir,

I was very interested to read the recent article by Rundle *et al.* on familial deafness associated with iris degeneration and glaucoma.[1] A similar association was recently described in an American pedigree where 11 out of 27 members were affected.[2] Linkage was found to chromosome 13q14 with a peak lod score of 4.64 for marker D13S1253 at $\theta = 0$. The critical disease interval encompasses a 26 cM region. In both families the mode of inheritance is dominant and the extent of iris hypoplasia looks very similar. There were many other non-ocular features noted in the American family including premature loss of teeth, congenital hip malformation and cryptorchism. It would be very interesting to know whether the family described by Rundle *et al.* has any of these non-ocular features and whether linkage can be demonstrated to this region of chromosome 13.

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A.J. Churchill, FRCOphth ✉

Department of Molecular Medicine
Clinical Sciences Building
St James's University Hospital
Leeds LS7 9TF
UK

Sir,

We thank Miss Churchill for her comments. One of us (A.J.L.) saw the poster[1] that she describes, at ARVO, and felt that the iris morphology was very similar to that shown by our patients. None of our patients, however, showed dental abnormalities, hip dislocation nor cryptorchism.

Finally, we have not checked for linkage to the 13q14 region but it would appear to be a possible candidate region, and one well worth excluding.

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Paul Rundle, FRCOphth
Andrew J. Lotery, FRCOphth
Desmond B. Archer, FRCS, FRCOphth
F. Gerard McGinnity, MD, FRCOphth

Department of Ophthalmology
Royal Group of Hospitals
Belfast
Northern Ireland

P. Rundle ✉
Eye and Ear Clinic
Royal Victoria Hospital
Grosvenor Road
Belfast 12 6BA
Northern Ireland
