employed for this type of problem. Finally, keratometry measures central corneal curvature in orthogonal meridians and assumes the whole cornea adopts this regular spherocylinder. All incisions have a paracentral flattening effect which is initially tempered by sutures. Removing them hastens the swing towards against-the-wound astigmatism which the coupling effect compounds. We have presented work that establishes how incisions have differing effects on vectored cylindrical outcome depending on their orientation.⁴ This is why selective suture removal is important. Removal of the closest aligned suture to the steep meridian will not necessarily reduce the magnitude of that cylinder but rather swing the axis of the vector in the opposite direction.

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Sir,

We thank Mr Horgan for his comments on our recent publication.¹ Several points are made in his letter, which are dealt with individually.

1. Automated keratometry was performed using the Canon IOL estimator, and this instrument was programmed to calculate the mean of five separate readings.
2. Sutures that appeared tight were removed. Twenty-two patients had one suture removed, 10 patients had two sutures removed and 2 patients had three sutures removed.
3. The topical corticosteroid–antibiotic preparation had been discontinued for at least 2 weeks prior to suture removal and all patients received a 5-day course of prophylactic chloramphenicol drops following suture removal.
4. We did not specifically design our study to address the issue of timing of suture removal but we found no statistically significant effect on the rate of astigmatic decay (see Results, last paragraph).
5. The mean change in cylindrical power and axis are clearly tabulated and show that the mean

cylindrical power does indeed decrease the most in the first 5 minutes following suture removal. We also state clearly in the results that 'the greater the initial astigmatism, the greater the change induced by suture removal' – not in the first 5 minutes but total change.

6. The mean change in astigmatism at 2 weeks is 1.29 dioptres; this value is clearly tabulated. The reason there is an upward trend in Fig. 1 is that the magnitude of change in astigmatism between 15 and 30 minutes is less than the change seen between 30 minutes and 2 weeks. Again this is clearly tabulated and demonstrated in Fig. 1.
7. We did not use vector analysis because the main aim of our paper was to assess in a simple clinical manner whether the resulting astigmatism 30 minutes following suture removal differs significantly from the residual astigmatism 2 weeks later. The most important readings, therefore, were those comparing astigmatic change between 30 minutes and 2 weeks after suture removal. The mean change (11.77) and range of change in axis (10.84–12.76) shown in the table for this time period of observation would not have influenced the results greatly if vector analysis had been performed. Secondly, it was felt that although the role of vector analysis in small incision and refractive surgery has been well defined, this is not the case in large sutured incisions.

Our paper dealt with what was at the time a common surgical problem in a clinical and practical way. It is, we believe, possible to approximately estimate the residual astigmatism 2 weeks following suture removal from the keratometry findings 30 minutes after suture removal.

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Reference

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Sir,

I am grateful to Martin Leyland¹ for highlighting my concern over the use of CS aerosols by the police.^{2,3} I have personally seen three police officers who developed significant ocular morbidity secondary to acquired dry eye states following a 'demonstration' of the CS aerosols – although these are more appropriately described as 'squirt cans' because they emit a pressurised stream of the solution of CS in a similar manner to windscreen de-icer cans, and not the mist

one tends to associate with the term aerosol. I understand that because of the problems that arose in his officers, their Chief Constable decided not to advocate the use of CS in his force. I have also seen many casualties, both in Inner London and in the West Country, following exposure to CS; I am not aware of any of these developing long-term sequelae although quite a few were 'lost to follow-up'. Many of these patients had non-ocular symptomatology, often of a respiratory nature, as well as ophthalmological involvement, and this may well reflect why such patients are not brought to a dedicated eye casualty department because of primary triage by the ambulance service. In addition the police surgeon would be the first person called to attend any individual with medical problems in police custody. I believe that the Poisons' Information Centre will be monitoring patients exposed to CS⁴ and one awaits their findings.

I would once again emphasise that CS is a safe agent when used for riot control purposes,⁵ where it is effective in producing intolerable symptoms at atmospheric concentrations of 0.0026%. However, the aerosols utilised by the police squirt a 5% solution of CS dissolved in methylisobutyl ketone, a solvent used in the organic chemical industry where safety regulations advise that protective clothing, gloves and eye/face protection are worn by all workers handling the chemical.⁶ Yet police officers are spraying CS dissolved in methylisobutyl ketone [4-methyl-2-pentanone] directly at members of the public – an action that may well lead to serious sequelae.

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Sir,

I welcome Mr Gray's comments on his experience with CS gas injury, and apologise for incorrectly referencing his previous correspondence on the

subject. It is clear from the nature of both the agent itself and the vehicle used for its delivery that ocular injury is theoretically possible. My point is that we have not in practice seen any cases in this hospital since the adoption of CS gas by the Metropolitan Police.

I am not aware of any published data describing ocular injuries resulting from the CS aerosol available to the police. Such information would clearly be helpful in the management of any future casualties.

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Sir,

Congratulations to Bray *et al.* (*Eye* 1996;10:714–8) upon bringing the subject of amblyopia to general attention once again. After years of neglect in the morass of developmental trends and the shift of responsibility to parents, as advised by the Hall report and so much beloved by the cost-cutting administrators, this is a breath of fresh air.

My experience over 30 years of paediatric ophthalmology evolved along similar lines. It was satisfying initially to pick up amblyopes from whatever cause at 3 and 5 years of age and to carry out the treatments with the expense involved.

However, in due time, I found by organising screening clinics during the sensitive period of visual development, with orthoptists at post-natal and infant welfare clinics, it was possible to pick up younger children with early visual defect, before gross amblyopia had developed. This meant that there was early referral, with only slight visual suppression, which entailed only a short period of occlusion and treatment. This was cost-effective for hospital attendances and for the families; indeed, in my region of South West Thames, full-blown amblyopia was virtually eliminated at that time.

Perhaps the pendulum may once again turn towards really early assessment, and referral, and hence lead to prevention rather than cure. I would urge the authors and all concerned with paediatric management to direct their energies towards this goal and fulfil the aspirations of the giants of yesteryear such as Mary Sheridan and Ronnie MacKeith.

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