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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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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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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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