ing, however, and may take up to 3 weeks before a result is available.[4] Enzyme immunoassay for adenovirus type 8 can also be used for confirmation,[5] as can immune dot blots.[6,7] Such methods can provide a result within 3–7 days. It is necessary to be aware, however, of the limitations of such techniques. We favour the use of transmission electron microscopy for virus detection, but recognise that this facility may not be available in all eye institutes. Such non-culture methods are useful since they can permit fairly early exclusion of a viral aetiology, and allow for investigation of a differential diagnosis of *Acanthamoeba* keratitis. Confirmation of this infection may be provided in many instances within 1 hour of receipt of a corneal sample, by simply examining the tissue using phase contrast microscopy. Culture on non-nutrient agar prepared in enriched amoebal saline should follow. There is no reliable method for unequivocal diagnosis of 'tight fit' lens syndrome, but symptoms and signs appear to abate rapidly when the contact lenses are removed from the eye.

Adenovirus kerato-conjunctivitis treatment is palliative and it is conventional to use analgesics and NSAIDs. Use of antibiotics, for example chloramphenicol, is not generally required.

Goodall *et al.* reported that the combination of propamidine (as Brolene) and neomycin did not provide a successful outcome in one of their 3 patients with *Acanthamoeba* keratitis. This treatment regimen has now been superseded. Cysts from most *Acanthamoeba* strains tested are resistant to neomycin, at least *in vitro*.[8] Some strains are resistant to propamidine.[9] If a patient has protozoologically confirmed *Acanthamoeba* keratitis, it is our contention that the treatment provided should comprise the combination of chlorhexidine (0.02%) and Brolene; this regimen has been shown to be an effective treatment, and is particularly useful if commenced at an early stage in amoebal infection of the cornea.[10]

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Sir,

We very much enjoyed the mathematical model proposed by Aylward and Lyons for achieving a single intraocular gas bubble during retinal reattachment surgery.[1] As the authors point out, creation of a single bubble greatly facilitates the subsequent fundus view allowing accurate and localised retinopexy. However, the risk of subretinal gas with multiple small bubbles is likely to be influenced, in addition, by other factors.

The greatest risk of subretinal gas relates to its use with pneumatic retinopexy in patients with vitreous detachment and horseshoe tears. This occurs early, and is not apparently related to subsequent gas expansion.[2] Access to the subretinal space is only possible if gas is behind the posterior hyaloid membrane (PHM) and care should be taken to ensure that the gas injection is carried out anterior to the PHM (Fig. 1). If the PHM is inadvertently breached then the gas injection may force it anteriorly, opening the break leading to subretinal gas bubbles (Fig. 2) ('gas bubble squeezes itself through . . . like a baby's head during delivery'[2]). In addition, new breaks can be produced by separating the PHM beyond its arrested insertion.[3] Fortunately,

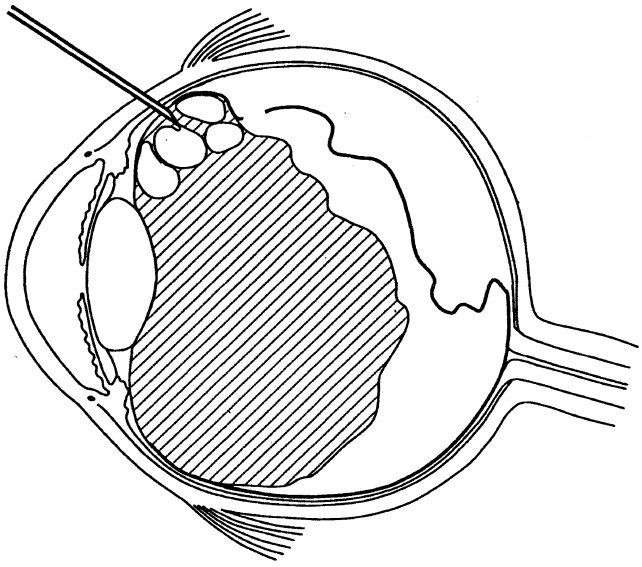

**Fig. 1.**

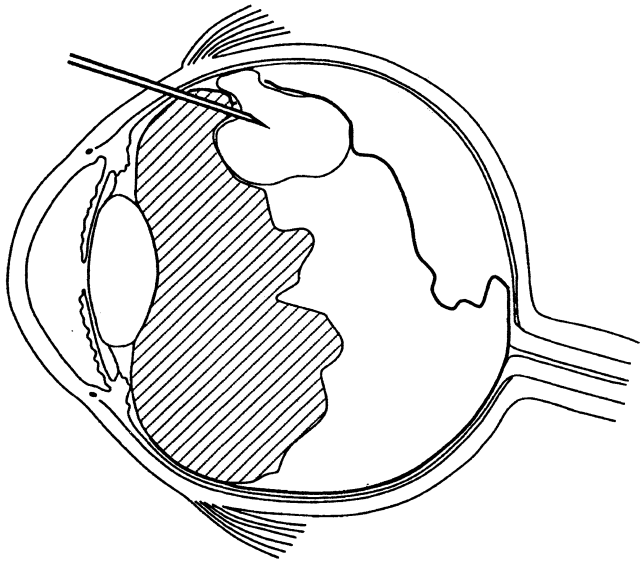

**Fig. 2.**

because the PHM is tough, inelastic and usually highly mobile, penetrating it with a needle tip to gain access to the retrohyaloid/preretinal space is quite difficult, as testified by attempts to sample subretinal fluid in this way prior to vitrectomy.

It follows that patients with an attached PHM and round holes have a negligible risk of subretinal gas. Those at greatest risk are: (1) patients in whom the PHM is shortened, e.g. by incarceration, so that its mobility is lost and perforation made easier; (2) patients who have an incomplete posterior vitreous detachment with a large defect in the PHM allowing gas bubbles through. These patients can still be managed safely with gas provided the retina is first flattened to close the break using the correct surgical D-ACE sequence as described by Gilbert and McLeod,[4] avoiding pneumoretinopexy.

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Sir,

We were very interested to read the paper by Sadiq and Downes[1] on dysversion of lateral eyelashes in children. They described four cases where this new diagnosis was made for recurrent red, sore, watery eyes in young boys. We would like to support this new diagnosis by reporting three further similar cases in young girls and discuss the implications for management.

*Case 1*

A 4-year-old girl attended her general practitioner with a history of recurrent episodes of a sticky, watery left eye. She was noted to have some ingrowing eyelashes at the lateral end of the left upper lid and was referred to the eye clinic for further management. The ophthalmologist confirmed the finding of misdirected lashes at the lateral aspect of the left upper lid which were subsequently treated with electrolysis under general anaesthetic (GA). This resulted in the resolution of symptoms for 3 years until she re-presented with similar problems in the right eye. On examination she was noted to have dysversion of the lateral lashes of the right upper lid, and was treated again with electrolysis under GA. Six months later she remains symptom free.

*Case 2*

A 4-year-old girl was referred to the eye clinic with a history of recurrent red, sticky eyes. There had been a variable response to treatment with topical antibiotics. On examination, several misdirected eyelashes were seen at the lateral aspects of both upper