LETTERS TO THE EDITOR

Sir,

I note that in their paper describing the treatment of recurrent erosions of the cornea,[1] Bernauer *et al.* dismiss mechanical debridement of the cornea as a method of treatment of recalcitrant cases. However, they may have proved its efficacy.

Before they can claim that excimer laser treatment has any added benefit, they really need to do a controlled trial to compare debridement followed by laser, with debridement alone.

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Reference

1. Bernauer W, De Cock R, Dart JKG. Phototherapeutic keratectomy in recurrent corneal erosions refractory to other forms of treatment. Eye 1996;10:561–4.

Sir,

Mr Kyle has raised an important issue. Mechanical debridement of the cornea is not dismissed in our paper. It was referred to as superficial keratectomy and two references were given.[1,2] This technique requires removal of all the epithelium to the limbus. We agree that a randomised control trial is required to test the different techniques for the management of refractory recurrent corneal erosion syndrome. Because the natural history of recurrent erosion is benign, with relatively few patients requiring invasive treatment,[3] we believe this would have to be a multicentre trial to recruit enough patients. Until this is done we could not comment on the relative efficacy of superficial keratectomy versus laser treatment for the management of this condition.

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2. Buxton JN, Carstad WH. Superficial epithelial keratectomy. Cornea 1987;6:292–7.
3. Hykin PH, Foss AE, Pavesio C, Dart JKG. The natural history and management of recurrent corneal erosion: a prospective randomised trial. Eye 1994;8:35–40.

Sir,

Karen Goodall and colleagues[1] described three case histories from keratitis patients, the aetiology of the infection being *Acanthamoeba* but the medical picture being reminiscent of adenovirus keratitis. We have reported similar observations.[2] Adenovirus keratitis was the preliminary clinical diagnosis in a contact lens wearer who presented at casualty with a painful red eye, when symptoms had been apparent for some 10 days. There was punctate epithelial keratitis reminiscent of early adenovirus infection. After a further 8 days, various sub-epithelial infiltrates had developed. At this stage, *Acanthamoeba* was isolated from a corneal scrape.

Early recognition in a soft contact lens wearer of unilateral conjunctival inflammation, with photophobia and excessive lacrymation, in the presence of a 'typical' epitheliopathy or pseudo-dendrite, with sub-epithelial opacities in some patients, provides a strong index of suspicion of *Acanthamoeba* keratitis. The clinical diagnosis is reinforced if there is excessive pain and corneal perineuritis is observed using the slit lamp. The observation of Goodall *et al.* in cases of adenoviral corneal infection, that focal sub-epithelial opacities are present later beneath epithelial lesions, and that this feature is unusual before 6–9 days, is a useful parameter for differential diagnosis. A further potentially confusing situation is afforded by the so-called tight fit or over-wear syndrome, which can present in the contact lens wearer with conjunctival inflammation, punctate epithelial keratitis and sub-epithelial infiltrates.[3] The latter triad of signs can be readily confused clinically with both adenovirus infection of the cornea and early *Acanthamoeba* keratitis.

Investigations of contact lens wearing patients with a clinical diagnosis of adenovirus kerato-conjunctivitis should, if possible, include isolation of the virus from the swabs or conjunctival scrapings, using susceptible cell cultures. This is time-consum-

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,