recognised limitations including the small sample size, the limited post surgical follow-up and the non masking of researchers. Nevertheless, our results have encouraged a randomised controlled trial, which is currently ongoing at Moorfields Eye Hospital and will give more solid evidence regarding the role of posturing in Macular hole surgery.

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Sir,

The use of intracameral phenylephrine in the management of intraoperative floppy-iris syndrome with doxazosin

We read with interest the findings of Dhingra *et al*.[1] Further reports on the problems of intraoperative floppy-iris syndrome (IFIS), associated with doxazosin have been described. The most recent ($n = 31$) finding is that 37% of patients taking doxazosin had features of IFIS.[2] However, larger series indicate that the problem is uncommon and less severe than for the α_{1A}-antagonist tamsulosin.[3–5] An observational study by Cheung *et al*[3] of 1689 patients undergoing cataract surgery found that 9 out of 42 patients using doxazosin had incomplete IFIS. Nguyen *et al*[4] found that out of 375 patients with IFIS taking an α_1-antagonist, six were using doxazosin. This was also reflected by the experience of Chadha *et al*[5] who found that 1 patient out of 48 using doxazosin developed incomplete IFIS.

However, we also recently encountered two patients using doxazosin with resultant moderate IFIS during cataract surgery. The first patient was a 76-year-old Caucasian male using 8 mg doxazosin po od for hypertension. The second was a 72-year-old Asian male taking 4 mg of doxazosin po od for benign prostatic hypertrophy. In both cases, there was incomplete pupil dilatation (5 mm for first patient and 6 mm for the second) and iris undulation was noted after initial wound construction. We successfully utilised dilute intracameral phenylephrine (as described by Gurbaxani and Packard[6]) to prevent iris prolapse and maintain pupil size during phacoemulsification. Surgery was completed successfully without complication.

Both patients were noted to be taking an α_1-antagonist preoperatively and the possibility of IFIS was anticipated. Undoubtedly, this influenced the decision to utilise intracameral phenylephrine early as a means of preventing intraoperative complications. Atthough our subjective grading of moderate IFIS (like Dhingra *et al*[1]) appears more severe than the experience of larger series, we agree that the possibility of well-described intraoperative complications should be anticipated. Among the numerous management options available, we found that the use of intracameral phenylephrine offered a quick and effective method for preventing further problems.

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Sir,

Pseudodendritic keratitis in children

We were interested to read Jain *et al*'s[1] finding of pseudodendritic keratitis associated with meibomitis in young healthy male subjects. Recently, two patients who demonstrated the typical morphological features described were treated at the West of England Eye Unit, Royal Devon & Exeter Hospital, UK. The first was a

6-year-old Caucasian male with a 2-day history of a red right eye associated with photophobia. He had complained of similar symptoms intermittently over the previous year. On examination, there was marked conjunctival infection, evidence of meibomian gland dysfunction, inferior marginal stromal infiltrates, widespread punctate epithelial erosions, and a horizontally orientated pseudodendrite in the inferior cornea. The second patient was an 8-year-old Caucasian female with a 4-month history of recurrent redness and irritation in the left eye, which had not responded to topical antibiotics. She had meibomian gland dysfunction, an inferotemporal marginal infiltrate, and a non-staining, horizontally orientated pseudodendrite inferiorly. Both patients responded rapidly to topical 0.5% prednisolone and oral erythromycin.[2]

In contrast to the series by Jain *et al*[1], both had unilateral disease and one patient was female. Mechanical rubbing of the inflamed eyelids on the corneal surface was suggested as a contributing factor to the aetiology of these lesions and the use of a bandage contact lens was recommended as treatment. The fact that our patients responded rapidly to topical steroids indicates an inflammatory process underlying the pathogenesis. Enhanced cell-mediated immunity at the corneal limbus to staphylococcal cell wall antigens, expressed as delayed hypersensitivity (type IV), has been hypothesised as the aetiology of marginal keratitis.[3]However, in rabbits subconjunctivally and topically immunized with staphylococcal antigens, corneal IgG antibodies have consistently been found[4] and a precipitation line of antigen–antibody complex has been demonstrated in the stroma of rabbits injected with antigen and antibody from opposite sides of the cornea.[5] Pseudodendritic keratitis in young patients with blepharitis may therefore represent an immune complex deposition or type III hypersensitivity reaction to staphylococcal antigen.

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Sir,

Reply to Tinley *et al*

We read with interest the observations of Tinley *et al*. We would agree with the authors that inflammation is one of the major contributing factors for such lesions in cases of meibomian gland dysfunction (MGD). In MGD, alterations in lipid secretions and abnormal keratinization of the meibomian gland duct orifices have a profound effect on the quality of tear film and on the ocular surface.[1] Inflammatory mediators accumulate in the tear film and the induced inflammation damages the ocular surface.[1]

Jain *et al*[2] have recently reported similar findings in a case of ocular rosacea, which after negative virologic studies, was successfully treated with topical steroids. However, in the current series, we noted resolution of the refractory lesions (3/5) after putting a bandage contact lens.[3] None of the cases were treated with the topical steroids. Increased ocular surface inflammation and mechanical rubbing of the inflamed eyelids on the corneal surface may have been the probable contributing causes for pseudodendritic keratitis. If only inflammation and antigen–antibody reactions were the contributing causes, then our cases would not have shown a resolution only with bandage contact lens. Also, at times, the variability in the presentation of the pseudodendrites may lead to a clinical suspicion of herpetic keratitis. In such cases, it is wise to initiate therapy with topical steroids after ruling out a true dendrite in spite of associated MGD. We would also emphasize that in cases of MGD besides other treatment modalities the underlying dysfunction of meibomian glands should be addressed with appropriate therapy.

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