were the first to describe the use of PHMB in the management of *Acanthamoeba* keratitis, and in one of their cases with early disease an apparent cure was effected after only 1 week of intensive treatment with this drug alone.

In our study single agents were used at an intensity and duration of treatment well below that in current clinical use, and drug levels reached in the cornea may not have been sufficiently high to kill trophozoites and cysts. It seems likely that if a beneficial effect of drug therapy in animal models is to be demonstrated intensive treatment with multiple agents for prolonged periods will be required.

T. J. Ruddell, FRACO, FRCOphth D. L. Easty, FRCS

Department of Ophthalmology University of Bristol Bristol Eye Hospital Lower Maudlin Street Bristol BS1 2LX UK

References

- 1. Elder MJ, Kilvington S, Dart JK. A clinicopathological study of *in vitro* sensitivity testing and *Acanthamoeba* keratitis. Invest Ophthalmol Vis Sci 1994;35:1059–64.
- Larkin DFP, Easty DL. Experimental Acanthamoeba keratitis. I. Preliminary findings. Br J Ophthalmol 1990;74:551–5.
- 3. Neiderkorn JA, Ubelaker JE, McClulley JP, et al. Susceptibility of corneas from various animal species to *in vitro* binding and invasion by *Acanthamoeba castellani*. Invest Ophthalmol Vis Sci 1992;33:104–12.
- He YG, McCulley JP, Alizadeh H, et al. A pig model of Acanthamoeba keratitis: transmission via contaminated contact lenses. Invest Ophthalmol Vis Sci 1992;33: 126–33.
- 5. van Klink F, Alizadeh H, He YG, *et al.* The role of contact lenses, trauma and Langerhans cells in a Chinese hamster model of *Acanthamoeba* keratitis. Invest Ophthalmol Vis Sci 1993;34:1937–44.
- 6. Wright P, Warhurst D, Jones BR. *Acanthamoeba* keratitis successfully treated medically. Br J Ophthalmol 1985;69:773–82.
- 7. Larkin DFP, Kilvington S, Dart JKG. Treatment of *Acanthamoeba* keratitis with polyhexamethylene biguanide. Ophthalmology 1992;99:185–91.

Sir,

Management of Superior Limbic Keratoconjunctivitis with Botulinum Toxin

Theodore's superior limbic keratoconjunctivitis is a disease which was first described in 1963. It is characterised by keratinisation and cellular infiltration of the superior bulbar conjunctiva, together with cellular infiltration of the upper palpebral conjunctiva. The disease is painful and the pain develops as the day progresses, usually reaching its maximum in a working individual in the late afternoon. The disease seldom interferes with sleep and is usually at its best on awakening.

The disease is difficult to treat successfully. Theodore reported treating the upper palpebral conjunctivae with silver nitrate 0.5–1.0% solution. This, in my experience, exacerbates the pain for 2 or 3 days and is followed by some relief which is never prolonged. I have never found the administration of drops to be of any value.

Some invasive treatments have been suggested, among them resection of the superior limbic and bulbar conjunctiva. Donshik *et al.*¹ claimed that all 4 of their patients had immediate and continued relief after this operation. Passons and Wood² stated that 8 of 10 of their patients who underwent this operation were either asymptomatic or much improved. Yet Darrell³ found that 2 cases of superior limbic keratoconjunctivitis of 16 years' duration, in identical twins, never permanently responded to bilateral resection of the superior bulbar conjunctiva.

My experience with superior bulbar conjunctival resection has been mixed. I have performed the operation on 9 patients. I asked my patients to score the improvement in their symptoms by pointing to a scale marked from 0 to 10. Four failed to get relief, with scores of 0. Two had moderate relief, with scores of from 4 to 7. One of these achieved this score after having failed with botulinum toxin injections. Three had marked relief: 1 of these with a score of 8 and lost to long-term follow-up, and two had scores of 10, remaining so for 4 years.

Wright⁴ proposed a mechanical cause for the condition and Ostler⁵ postulated an upper lid in tight apposition to the superior bulbar conjunctiva, together with a lax superior bulbar conjunctiva, as the cause.

Accordingly I assessed the effect of botulinum toxin injections to the orbicularis muscle in patients with superior limbic keratoconjunctivitis. Generally I started with half the recommended dose given for essential blepharospasm. If there was no obvious blepharospasm associated with the superior limbic keratoconjunctivitis I tended to confine my injections to the superior orbital portions of the orbicularis muscles. Some of my patients had obvious blepharospasm and in these cases I injected the inferior orbital portions of the orbicularis as well.

The botulinum toxin used was Dysport (Speywood Pharmaceuticals, Maidenhead, UK). This is dispensed in a 500 unit vial to which is added 2.5 ml saline for injection. The injection sites are illustrated in Fig. 1. The doses indicated are full doses. (It must be emphasised that Botox, from Allergan Pharmaceuticals, Irvine, California, measured in units, is approximately three times more powerful than Dysport.)

Again I scored the success on a scale of 0 to 10. I

144



Fig. 1. Injection sites for the Dysport solution with the standard amounts labelled in millilitres. The direction of insertion of the needle is indicated by the arrowheads. The uppermost curve in the diagram indicates the superior orbital margin. The injections are conveniently performed with a 1 ml diabetic syringe.

treated 21 patients. Four of these, who had 0 ratings for superior limbic resections, had 0 ratings for botulinum toxin injections, as had a further patient lost to long-term follow-up (24% altogether). Outcomes in 3 of the patients (14%) were moderately successful, with scores of 4 to 7. One of these was a patient who failed with superior bulbar conjunctival resections. Outcomes in 13 (62%) of the patients were outstandingly successful, with scores of 10. In 2 of these patients the injections did not have to be repeated, as usual, after a period of time and they still have a score of 10 after 3 years. One of these patients had contemplated suicide on three occasions; she now needs injections to the superior orbital portions of the orbicularis at 6 monthly intervals and has a 10 out of 10 score.

As far as complications of the treatment are concerned, 2 patients developed ptosis. One had a

marked bilateral ptosis and the other a moderate unilateral ptosis. Both had scores of 10 after these resolved in a few weeks. Ptosis can be avoided by injecting above the superior orbital rim and using half doses at the start. One patient developed temporary lower lid entropion on two occasions after the injections. One patient with a successful outcome developed a facial change in which the angles of the mouth dropped. There was no visible facial paralysis but she was distressed. This took several months to resolve, as is usual with this complication. It can be avoided by omitting the injections to the inferior orbicularis.

Despite improvement in the symptoms with this treatment there was no consistent decrease in the signs of this disease, except when they were initially gross.

Ian A. Mackie

99 Harley Street London W1N 1DF UK

References

- Donshik PC, Collin HB, Foster CS, Cavanagh HD, Boruchoff SA, Magee AJ. Conjunctival resection treatment and ultrastructural histopathology of superior limbic keratoconjunctivitis Am J Ophthalmol 1978;85:101–10.
- 2. Passons GA, Wood TO. Conjunctival resection for superior limbic keratoconjunctivitis. Ophthalmology 1984;91:966–8.
- 3. Darrell RW. Superior limbic keratoconjunctivitis in identical twins. Cornea 1992;11:262–3.
- 4. Wright P. Superior limbic keratoconjunctivitis. Trans Ophthalmol Soc UK 1972;92:555–60.
- Ostler HB. Superior limbic keratoconjunctivitis In: Smolin G, Thoft RA, editors. The cornea. Boston/ Toronto: Little Brown, 1987:296–8.