

LETTERS TO THE EDITOR

Acute angle-closure glaucoma following botulinum toxin

SIR,—I would like to point out the great significance of the case report published by P Corridan *et al.*¹ They describe a patient who developed an acute angle-closure attack as a rare complication of periocular botulinum toxin (BoTx) for blepharospasm.

As regards the pupillary effects of BoTx, it was Carl Kupfer² who first reported that retrobulbar injection of BoTx in rabbits produces mydriasis, probably by affecting the ciliary ganglion. However, very high doses were used in his study,² and in order to prevent lethal complications anti-BoTx antibodies were injected into all the animals. In contrast to the lethal doses used by Kupfer² we injected non-lethal doses of BoTx into the right orbit of rats (between 1.5 ng–2.5 ng BoTx). All animals developed ipsilateral mydriasis with cholinomimetic supersensitivity, without apparent optic nerve dysfunction.³ The mydriasis disappeared spontaneously over a period of eight weeks, and pupillary supersensitivity remained for longer. No electrophysiological evidence of generalised neuromuscular dysfunction followed the injected doses of 1.5–2.5 ng BoTx, which are close to those used in humans (0.25–6.5 units).⁴ Our conclusion is similar to that of Dr Kupfer's,² and we are also of the opinion that BoTx injected periocularly or retrobulbarly diffuses towards the ciliary ganglion and there impedes the cholinergic innervation of the pupil.

This case report highlights a serious complication of periocular BoTx injection, which, though rare, should be taken into consideration in patients with closable narrow angles who undergo this procedure.

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- 1 Corridan P, Nightingale S, Mashoudi N, Williams AC. Acute angle-closure glaucoma following botulinum toxin injection for blepharospasm. *Br J Ophthalmol* 1990; 74: 309–10.
- 2 Kupfer C. Selective block of synaptic transmission in ciliary ganglion by type A botulinum toxin in rabbits. *Proc Soc Exp Biol Med* 1958; 99: 474–6.
- 3 Levy Y, Kremer I, Shavit S, Korczyn AD. The pupillary effects of retrobulbar injection of botulinum toxin A (Oculinum) in albino rats. *Invest Ophthalmol Vis Sci* (In press).
- 4 Scott AB, Kennedy RA, Strubbs Jr HA. Botulinum A toxin injection as treatment for blepharospasm. *Arch Ophthalmol* 1985; 103: 347–50.

SIR,—In their recent case report of acute angle-closure glaucoma following botulinum toxin injection for blepharospasm¹ the authors recommend that patients at risk of developing angle-closure glaucoma should undergo prophylactic laser iridotomies. Laser iridotomy is the definitive treatment for most cases of angle-closure glaucoma, but it should not be performed without a proper indication. 'Prophylactic iridotomy' is a misleading term, because we tend to perform it in many eyes which will never develop a disease. Furthermore, one must bear in mind the possible complications of laser iridotomy, which include rare disasters such as foveal burns and retinal detachment, and less disastrous but more frequent compli-

cations such as transient or permanent elevation of the intraocular pressure – the latter due to pigment dispersion in the angle. In addition, there is considerable evidence that lens changes, leading to myopia and cataract, are frequent sequelae of laser iridotomy.²

There is an alternative to laser iridotomy in patients about to undergo botulinum toxin injection into the eyelids: why not prevent mydriasis by prescribing pilocarpine for a few days?

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- 1 Corridan P, Nightingale S, Mashoudi N, Williams AC. Acute angle-closure glaucoma following botulinum toxin injection for blepharospasm. *Br J Ophthalmol* 1990; 74: 309–10.
- 2 Hyams S. *Angle-closure glaucoma. A comprehensive review of primary and secondary angle-closure glaucoma.* Amsterdam: Kugler and Ghedini, 1990: 151–63.

SIR,—It is interesting that all the albino rats injected with botulinum toxin intraorbitally developed mydriasis, especially as the dose used by Drs Kremer and Levi was much lower than that used by Kupfer in 1958.

We thank Dr Hyams for his letter but would like to point out that we said in our paper, 'These who are at risk could have prophylactic YAG laser iridotomies.' We did not say they should have iridotomies. It would be inappropriate to be so didactic, as after all there are to date no other reports of angle-closure glaucoma following botulinum toxin injection periocularly in humans. Regarding pilocarpine drops, they would need to be used for up to three months following each injection to coincide with the duration of action of botulinum toxin.

We would like to correct an error in our original paper regarding the dose of botulinum toxin used, in the paragraph entitled 'Technique.' The units were µg, not mg as stated. It should read 0.05 µg of freeze-dried botulinum toxin-haemagglutinin complex (0.008 µg neurotoxin).

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Lens biometry and diabetes

SIR,—In a study based on Scheimpflug photo evaluation of anterior segment structures early-onset diabetics have been compared with a control group.¹ Plots and regression lines are presented with biometry data as the dependent variable and age on the abscissa. Diabetics have thicker lenses (for age) than non-diabetics, a trend that is more apparent the longer the duration of the diabetes.

The authors state that this is the first report in which a precise assessment of the effect of 'true' diabetic duration on lens biometry has been possible. However, as part of a series of studies with focus on diabetic myopia we published a similar finding in 1987.² Compared to the present article our set-up was small-scale and with less detail regarding lens biometry. Our material was composed mainly of early-onset diabetics. A few type 2 diabetics with clinically clearly documented diabetes onset were included, our working hypothesis being the duration of the metabolic disorder proper, and not the insulin medication as focused on by Sparrow *et al.*

Whatever the mechanism, the two studies both support the concept of a positive cor-

relation between lens thickness and diabetes duration.

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- 1 Sparrow JM, Bron AJ, Brown NAP, Neil HAW. Biometry of the crystalline lens in early-onset diabetes. *Br J Ophthalmol* 1990; 74: 654–60.
- 2 Fledelius HC, Miyamoto K. Diabetic myopia – is it lens-induced? An oculometric study comprising ultrasound measurements. *Acta Ophthalmol (Kbh)* 1987; 65: 469–73.

SIR,—In our study of lens biometry in early-onset diabetes one of our objectives was to provide a precise statement concerning the effects on the lens of 'true' diabetic duration.¹ Information on disease duration of 'late-onset' diabetes is frequently inaccurate, and for this reason a strict definition of 'early onset' diabetes was applied in our study. Our information on 'true' diabetic duration was therefore reasonably precise.

Among the early-onset diabetics we found that, for all but one of the lens biometric features measured, age and diabetic duration were the most important determinants of lens biometry. In our multiple regression analysis we fixed the age slope in the diabetic subgroup to that of the non-diabetic control group prior to fitting the duration regression term. The slope of our duration term therefore represents the additional effect of diabetic duration over and above the normal aging effect. Our methods thus provide the most precise estimate available of the effect of 'true' diabetic duration on lens biometry.

Fledelius *et al* take issue with our statement that 'this report is the first in which a precise assessment of the effect of "true" diabetic duration on lens biometry has been possible.' In their paper, however, they provide no information regarding the diabetic type of the patients included in their study.² The reader is therefore left ignorant as to whether the 'true' diabetic duration of their patients was known. It is apparent only from their present communication that the patients in their study were mostly early-onset diabetics, with 'a few type 2 diabetics' included.

Fledelius *et al* employed a statistical method which demonstrated an effect of diabetic duration. Their method did not, however, provide 'a precise assessment of the effect of "true" diabetic duration on lens biometry.' In their method they calculated: 'individual lens thickness deviation values (in %), with + or – to signify higher or lower than expected according to non-diabetic regression line values for actual age and sex.' Using these calculated 'percentage deviation values' they then performed a second regression analysis against diabetic duration. Their method is acceptable for determining whether there is a duration effect or not, but it certainly has not provided 'a precise assessment of the effect of diabetic duration.' In fact, the regression coefficient quoted in their paper is a rather meaningless statistic, and does not provide the reader with any sort of useful estimate of the impact per year of diabetic duration on lens thickness. (Their reason for using a 'percentage deviation value' is unclear, as this would have the effect of distorting the magnitude of their duration effect, and implies an *a priori* belief that the impact of diabetic duration varies with the size of the lens.)

Our statement therefore is defensible, firstly, because no information regarding type of diabetes was provided by Fledelius *et al* in their paper,² and, secondly, because the



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