LETTERS TO THE EDITOR

Adverse effects of subconjunctival injections of mydriatic agents

SIR,—The adverse effects of mydriatic agents are well documented. When using these drugs one must carefully consider the relevant medical history. The uveitic patient with early posterior synechiae and concomitant systemic disease is illustrative of this problem. We report a serious complication in a patient treated with mydriatic agents.

A 26-year-old male had a 10-day history of photophobia, fatigue, and fevers for several months. On examination his vision was 20/40 and the pupils were constricted secondarily to 270° posterior synechiae. He had hyperaemic conjunctivae, diffuse 'mutton-fat' keratic precipitates, and 3+ cells. Dilatation with topical and pledget soaked tropicamide 1% and phenylephrine 2.5% was unsuccessfully attempted. The patient returned 24 hours later, and a mixture of two drops (100 µl) each of tropicamide 1%, atropine 1%, and phenylephrine 10% was injected subconjunctivally at the inferior limbus of the right eye. This mixture comprised 7 mg of phenylephrine, 1 mg of atropine, and 1 mg of tropicamide. The patient immediately developed severe headache, dyspnoea, and diaphoresis. His pulse was 110 per minute and blood pressure of 160/110 mmHg.

He was taken to the emergency room, where he was found to be in respiratory failure. Chest x-ray revealed diffuse interstitial infiltrates not present on the previous day's films. The patient was intubated, and Swan-Ganz catheterisation revealed a pulmonary wedge pressure of 8 mm. He was transferred to the intensive care unit for management of non-cardiogenic pulmonary oedema. Subsequent diagnostic testing was consistent with a diagnosis of sarcoidosis. The pulmonary oedema resorbed and the patient was discharged on topical steroids with subsequent resolution of the iritis.

This is the first reported case of non-cardiogenic pulmonary oedema associated with the use of mydriatic drugs. Both atropine and tropicamide are anticholinergic drugs whose actions include relaxation of the circular muscle of the iris and paralysis of the ciliary muscle. The principal adverse cardiopulmonary effect of these drugs is tachycardia. Phenylephrine, a potent, direct-acting, and selective α_1 agonist exerts its main mechanism of action by stimulation of α receptors of the dilator pupillae.¹ Stimulation of these receptors can cause constriction of the systemic, pulmonary, and coronary arteries, leading to severe hypertension, headache, ventricular arrhythmias, myocardial infarction, and cardiac arrest.² It is conceivable that in our patient phenylephrine may have produced severe pulmonary vasoconstriction leading to alveolocapillary damage and consequent non-cardiogenic pulmonary oedema. In addition our patient had pre-existing pulmonary sarcoidosis, which may have increased his susceptibility to lung injury from other insults.

Subconjunctival injections of mydriatic agents to forcibly dilate pupils with synechiae is recommended³ and is commonly used in several eye centres. These injections, especially in hyperaemic conjunctivae, can lead to enhanced systemic absorption and serious complications. We advise extreme caution

when using this method of administering dilating agents.

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- 1 Lamberts DW, Potter ED. *Clinical ophthalmic pharmacology*. Boston: Little, Brown, 1987: 321.
- 2 Fraunfelder FT, Scafidi AF. Possible adverse effects from topical ocular 10% phenylephrine. *Am J Ophthalmol* 1978; 85: 447.
- 3 Duane TD. *Clinical ophthalmology*. Revised ed. Philadelphia: Lippincott, 1988: 4: chapter 43: 3.

Branch retinal artery occlusion in toxoplasma retinochoroiditis

SIR,—Although toxoplasma retinochoroiditis may cause retinal vein occlusion, vascular remodelling, and even retinochoroidal anastomosis,^{1,2} retinal artery obstruction is rare.^{3,5} We have recently studied a patient suffering from such a complication.

An 18-year-old student attended in March 1989, 24 hours after the sudden loss of inferotemporal field in the right eye, which was confirmed by automated perimetry. A branch artery occlusion was found in a corresponding area of focal retinochoroidal inflammation. Some areas of periphlebitis were also seen peripherally. Fluorescein angiography confirmed occlusion of the artery within the area of inflammation (Fig 1). Antibodies to toxoplasma were detected by latex agglutination at serum dilutions of more than 1:1000.

Figure 1 Fluorescein angiogram showing the active retinochoroidal inflammation and the occluded retinal arteriole (arrow).

Figure 2 Fluorescein angiogram showing resolution of the inflammation and reperfusion of the retinal arteriole (arrow).

The patient was treated with clindamycin, 150 mg four times a day, and prednisolone 60 mg daily, reducing over eight weeks. At the end of this period the inflammatory focus had become inactive. Repeat fluorescein angiography demonstrated reperfusion of the artery (Fig 2). However, the field defect persisted.

This case confirms that field loss in toxoplasma retinochoroiditis can result from arterial occlusion. Arterial thrombosis is presumably caused by the intense adjacent inflammation. Corticosteroid and antimicrobial therapy⁴ may assist resolution of the inflammatory focus and should be considered when retinal vessels are involved and therefore at risk of occlusion. If, as in this case, occlusion has already occurred, retinal function may not recover despite resolution of the inflammation and reperfusion of the blood vessel.

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- 1 Gaynon MW, Boldey ED, Strahlman ER, Fine SL. Retinal neovascularisation and ocular toxoplasmosis. *Am J Ophthalmol* 1984; 98: 585–9.
- 2 Gass JM. *Stereoscopic atlas of macular disease: diagnosis and treatment*. 3rd ed. St Louis: Mosby, 1987; 2: 464–9.
- 3 Owens PL, Goldberg MF, Busse BJ. Prospective observation of vascular anastomoses between the retina and the choroid in recurrent toxoplasmosis. *Am J Ophthalmol* 1979; 88: 402–5.
- 4 Braunstein RA, Gass JDM. Branch artery obstruction caused by acute toxoplasmosis. *Arch Ophthalmol* 1980; 98: 512–3.
- 5 Morgan CM, Gragoudas ES. Retinal artery occlusion associated with recurrent toxoplasmosis. *Arch Ophthalmol* 1987; 105: 130–1.
- 6 Lakhanpal V, Schocket SS, Nirankari VS. Clindamycin in the treatment of toxoplasmic retinochoroiditis. *Am J Ophthalmol* 1983; 95: 605–13.

Corneal penetration by tarantula hairs

SIR,—Tarantulas are large, hairy spiders belonging to the family Theraphosidae. Their popularity as pets parallels the increasing popularity of exotic pets in general. Although their bite is virtually harmless to humans, many species of New World tarantulas possess projectile urticating hairs on the dorsal surface of the abdomen.¹ When threatened, the spiders rapidly stroke their hind legs in a vibratory fashion on the dorsal hairs, catapulting a cloud of barbed hairs at the attacker. Some species may incorporate urticating hairs into their retreats or webs. These hairs, each possessing multiple barbs, are capable of penetrating deeply into skin, and causing intense and prolonged localised urticaria in humans.^{1,2} In addition to dermatological trauma tarantula hairs are capable of embedding into the ocular tissues and producing an inflammatory reaction in the eye.^{3,4} Due to its small calibre, offending hairs could easily be overlooked without a slit-lamp examination of the anterior eye segment.

A 13-year-old boy reported feeling severe foreign body sensation in both eyes within five minutes of stroking his pet tarantula. This was followed by intense pruritus, tearing, conjunctival injection, and eyelid swelling, thus prompting the patient to rub his eyes vigorously with his fingers. The eye rubbing further aggravated his discomfort. The patient was seen that day by his family physician, who treated him with antibiotic eyedrops. When the symptoms and signs showed only minimal resolution after three weeks of treatment, the patient was referred for ophthalmological con-

Figure 1: Straight arrows indicate discrete, mammular, deep corneal stromal infiltrates round partially degraded hairs. Curved arrow indicates keratic precipitate surrounding spider hair protruding posteriorly through Descemet's membrane into anterior chamber. Hairs not visible in photograph, but were better appreciable by actual clinical examination.

sultation. At that time, his visual acuity was 20/40 OD and 20/25 OS. Mild to moderate conjunctival redness and swelling were noted. By slit-lamp biomicroscopy multiple tarantula hairs were seen at all depths of both corneas. Some hairs were also noted to protrude through Descemet's membrane posteriorly into the anterior chamber. Hairs embedded in the corneal stroma were surrounded by focal, whitish, intrastromal infiltrates, while hairs protruding posteriorly into the anterior chamber were encapsulated with dense, 'mutton fat' inflammatory precipitates (Fig 1). A mild iritis was also present. The remainder of the ocular examination showed nothing remarkable.

As the spider hairs appeared deeply embedded in the corneas, we did not attempt to remove them. In order to reduce the existing inflammatory reaction in the anterior eye segment, topical corticosteroids were instilled in both eyes about 10 times each day during waking hours. A rapid decrease in the ocular inflammation was observed within 48 hours, and the corticosteroid eyedrops were tapered slowly over a period of three months. The embedded hairs gradually underwent resorption, and by 10 months none were visible. The visual acuity returned to 20/20 in both eyes, and the patient has remained symptom-free.

The irritative effects of tarantula hairs are thought to be mainly mechanical,¹² though a toxic chemical or hypersensitivity phenomenon has not been ruled out. Cooke *et al.*¹ classified the hairs from Theraphosid spiders into four types. In small mammals, such as mice, a generalised inflammatory reaction from type IV hairs involving the respiratory tract may produce death from asphyxia. It is believed that type III hairs, which are long and thin with many sturdy barbs, are most likely to penetrate skin and cause severe urticaria in humans. *Brachypelma smithi*, a large colourful species native to Mexico and often sold in pet stores, has type III hairs. Eye injury from hairs have included keratitis, conjunctivitis, iritis, and putative chorioretinal scarring.^{3,4} We recommend that extreme caution be exercised during and after handling these spiders.

Patients suspected of having ocular injuries should be examined closely by slit-lamp biomicroscopy.

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- 1 Cooke JAL, Miller FH, Grover RW, Duffy JL. Urticaria caused by tarantula hairs. *Am J Trop Med Hyg* 1973; 22: 130-3.
- 2 Ratcliffe BC. A case of tarantula-induced papular dermatitis. *J Med Entomol* 1977; 13: 745-7.
- 3 Stulting RD, Hooper RJ, Cavanaugh HD. Ocular injury caused by tarantula hairs. *Am J Ophthalmol* 1983; 96: 118-9.
- 4 Hered RW, Spaulding AG, Sanitato JJ, Wander AH. Ophthalmia nodosa caused by tarantula hairs. *Ophthalmology* 1988; 95: 166-9.

Lactic acid and neovascularisation

SIR,—My opinion was that the *British Journal of Ophthalmology* is one of the best ophthalmological journals all over the world, but reading your editorial in the June issue¹ I am disappointed. In your editorial you cite my work² and then write: 'Later attempts to confirm this by demonstrating excess lactic acid in the vitreous of kittens and rats whose retinas has been rendered ischaemic were unsuccessful, and lactic acid seems to have been dropped.' This was the work of Gerke *et al.*,³ and I am sorry that you have not read my answer to it.⁴ I reported that in cases of rubeosis iridis the lactic acid concentration of the aqueous humour is significantly increased, and stated that 'the findings of Gerke *et al.* (1976) prove that at the beginning of the proliferative phase of experimental fibroplasia the lactate content of the inner layers of the retina, i.e., of the least part of the investigated specimens, did not rise to a degree which could have significantly increased the lactate concentration of the whole vitreous-retina-choroid specimen'.

Since then I have summarised my work on ocular neovascularisations several times,⁵⁻⁸ and I know of some work of others confirming my findings. For example, Deem *et al.*⁹ and Cunha-Vaz¹⁰ have found that after a single injection of lactic acid into the vitreous there was an intensive endothelial proliferation or thymidine uptake – that is, the first signs of neovascularisation – in the retinal venules of experimental animals. I see that you have not met with these publications either.

I have not dropped the lactic acid and I am still working in it, and my results prove the following: (1) The lactic acid concentration of vascularising tissues is increased. (2) Increasing the lactic acid concentration of avascular tissues leads to vascularisation. (3) In cases of avascular swelling the lactic acid concentration is decreased. (4) L-lactate induces more intensive corneal vascularisation than D-lactate foreign to the organism. (5) Lactic acid in 10^{-3} M/l concentration stimulates the thymidine uptake and the proliferation of cultivated vascular endothelial cells in vitro.

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- 1 Smith R. Neovascularisation again. *Br J Ophthalmol* 1990; 74: 323.
- 2 Imre G. Studies on the mechanism of retinal neovascularisation: role of lactic acid. *Br J Ophthalmol* 1964; 48: 75-82.
- 3 Gerke E, Spitznas M, Brodde OE. The role of lactic acid in retinal neovascularization. *Graefes Arch Clin Exp Ophthalmol* 1976; 200: 79-84.
- 4 Imre G. The role of lactic acid in neovascularizations. *Graefes Arch Clin Exp Ophthalmol* 1977; 201: 263-6.
- 5 Imre G, Bögi J. Corneal vascularization. *Int Congr Symp Ser* 1981; 40: 233-5.
- 6 Imre G. The role of increased lactic acid concentrations in neovascularisations. *Acta Morphol Hung* 1984; 32: 97-103.
- 7 Imre G. Pathogenesis of proliferative retinopathy and rubeosis iridis of diabetics. In: Henkes H, ed. *Proceedings of the VIIth Congress of the European Society of Ophthalmology*. Helsinki: Yliopistopaino, 1985, 510-1.
- 8 Imre G. The role of lactic acid in the mechanism of neovascularization. *Doc Ophthalmol Proc Ser* 1987; 50: 511-6.
- 9 Deem CW, Futterman S, Kalina RE. Induction of endothelial cell proliferation in rat retinal venules by chemical and indirect physical trauma. *Invest Ophthalmol Vis Sci* 1974; 13: 580-5.
- 10 Cunha-Vaz J. Physiopathogenesis of retinitis proliferans and new vessel formation. In: François J, ed. *Blutkreislauf in der Uvea und im Sehnerven*. 5. Kong Europ Ges Ophthalmol Hamburg 1976. Stuttgart: Enke, 1978: 247-53.

** Many thanks to Professor Imre for his kind words about the *BJO*. As to the question of lactic acid and its role in neovascularisation, I am delighted to hear further details of this and also congratulate him for his determination in pursuing his researches. I am sorry he was disappointed that the editorial coverage was not complete, but the main function of this sort of editorial is to stimulate interest, and this seems to have been successful. I would be the first to admit that this type of editorial can never give complete coverage of a difficult subject like this. – ED, *BJO*.

Aqueous humour in insulin-dependent diabetic patients

SIR,—I read with much interest the article by Hayashi M, *et al.*¹ which reported on the decrease of aqueous humour formation in insulin-dependent diabetic patients, as measured by means of fluorophotometry. It is interesting to note that in 1965, by means of other methods (the suction cup of Rosengren