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.

PATRICIA C T CHANG
H KAZ SOONG
W. K. Kellogg Eye Center,
University of Michigan,
1000 Wall Street,
Ann Arbor, Michigan 48105,
USA

JAMES M BARNETT
315 East Sherman Boulevard,
Muskegon, Michigan 49444,
USA

- 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, Sanitito 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.

G IMRE
2nd Department of Ophthalmology,
Maria U.39,
H-1085 Budapest,
Hungary

- 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*. S. 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

and Ericson and the recovery rate according to Okun), I and Diotallevi reported on the decrease of aqueous humour formation in diabetic patients.¹ No difference was found between diabetics with and without retinopathy. In contrast, we found that insulin treatment was able to restore aqueous humour formation to normal values in diabetic patients, provided retinopathy had not yet been observed. Our findings suggested that the rate of aqueous flow is influenced by insulin treatment in diabetic patients, at least in the initial stages of the disease.

G AURICCHIO
Eye Clinic,
1st Faculty of Medicine,
University of Naples,
Italy

- 1 Hayashi M, Yablonski ME, Boxrud C, Fong N, Berger C, Iovanovic LJ. Decreased formation of aqueous humour in insulin-dependent diabetic patients. *Br J Ophthalmol* 1989; 73: 621-3.
- 2 Auricchio G, Diotallevi M. Beziehungen zwischen Insulinbehandlung und Kammerwasserproduktion bei Diabetikern. *Graefes Arch Clin Exp Ophthalmol* 1965; 168: 85-9.

SIR,—We appreciate Dr Auricchio's interest in and comments on our article. The suction cup method which Dr Auricchio used to measure aqueous humour formation is a pressure-dependent technique and has several inherent problems similar to those of tonography. We believe that fluorophotometry is a much more accurate method of investigating aqueous humour dynamics. However, we regret that we did not refer to Dr Auricchio's paper, which also found a decrease in aqueous humour formation in diabetic patients.

MIEKO HAYASHI
MICHAEL E YABLONSKI
Department of Ophthalmology,
600 South 42nd Street,
Omaha, NE 68198-5540,
USA

Bacillus-induced endophthalmitis

SIR,—We regret that we incorrectly stated some antibiotic dosages in our article entitled 'Bacillus-induced endophthalmitis: new series of 10 cases and review of the literature' in the *BJO* 1990; 74: 26-9.

In Table II (p 27) the doses of 400 milligrams (mg) gentamicin (patients 5 and 8) are incorrect; the intravitreal dosage of gentamicin was 400 micrograms (μ g). The doses of clindamycin 1 gram (g) in patients 5 and 8 are also incorrect; the dose was 1 milligram (mg). Finally, the vancomycin 1 gram (g) dosage for patient 8 is incorrect; the dosage was 1 milligram (mg).

ANN S BAKER
RAMZI HEMADY
Harvard Medical School,
Massachusetts Eye and Ear Infirmary,
243 Charles Street,
Boston, MA 02114, USA

BOOK REVIEWS

Primary Angle Closure Glaucoma. By R F Lowe and A S M Lim. Pp. 89. No price given. PG Publishing: Singapore. 1989.

This slim volume distils the experiences of two eminent ophthalmologists in the diagnosis and

management of angle closure glaucoma. The first eight chapters were written by Ron Lowe and reflect publications written by him on this subject dating from 1961. The remaining four chapters were written by Arther Lim and describe his operative and laser techniques in the treatment of this condition.

Dr Lowe's contribution covers pathophysiology, clinical types, examination, and management. Each chapter carries many words of wisdom, as is to be expected from an ophthalmologist who has spent much of his professional career studying angle closure glaucoma. Each chapter concludes with references for further reading.

Dr Lim's contribution describes methods for the surgical and laser treatment of angle closure. He covers iridectomy (though the illustrations for this appear to be the same as, but smaller than those which appeared in his book *Peripheral iridectomy*, also published by PG Publications), trabeculectomy, including complications, and concludes with a small section on combined cataract and glaucoma.

The authors do not identify a readership. This reviewer also found it difficult to see whom the book was directed towards. The book is a simple and didactic description of angle closure glaucoma. It would be covered in content by more general ophthalmological texts. The illustrations have substantially been published elsewhere. The text is too simple and without adequate references for the specialist student of glaucoma. If a coffee table existed for ophthalmology textbooks, this one could be on it, but it need not grace the bookshelf.

R HITCHINGS

Synopsis of Ophthalmology. 6th Edn. By Jack J Kanski. Pp 237. £14.95. Butterworth: Sevenoaks, Kent, 1989.

Aids to Ophthalmology. By P T Khaw, D S Hughes, S J Keightley, R F Walters, A R Elkington. Pp. 294. £8.95. Churchill Livingstone: Edinburgh, 1989.

These two books cover similar subject matter so are reviewed together. *Synopsis* is a completely revised 6th edition, the previous edition having been originally published in 1975. *Aids* is a new book from a group of authors from Southampton Eye Hospital. Similarities between the books include their size, both being easily transported paperbacks, and intended audiences, in both cases ophthalmologists in training approaching their postgraduate examinations. They are also similar in use of short note/list presentation rather than prose, and neither contains any illustrations or diagrams.

The subject matter in Kanski's book is a systematic review of the main aspects of clinical ophthalmology, the chapter titles including eyelids, orbit, lacrimal apparatus, etc. Khaw and co-authors cover this ground but add more basic science material including optics and pharmacology, a detailed chapter on visual standards and a dedicated chapter on medical ophthalmology. Neither book can really be faulted for the accuracy of information presented. Interestingly, both books cover the difficult topic of nystagmus, but neither quite reaches the standard of the masterly chapter in Bajandas and Klein's *Neuro-ophthalmology review manual* (Slack, 1988).

The print is much tighter on the pages of the book by Khaw and co-authors and is therefore a much more daunting prospect than the spacious and concise layout of Kanski's book. The quantity of information in *Aids* is also much greater, and it has much more the feel of

a condensed version of a textbook such as Newell's *Ophthalmology: principles and concepts* (Mosby, 1986). By contrast, Kanski sticks to mainstream clinical matters, which are covered in the neat way we have come to enjoy in his other books, including *Clinical ophthalmology* and *The eye in systemic disease*. The effect of these differences would be to make memorising the book by Khaw and co-authors a major challenge unless tackled with advanced examination preparations. Conversely, the moderately well read ophthalmologist in training could skim through *Synopsis* quite quickly and feel that new facts had been learnt on each page.

Both of these are good books and their authors are due great credit for the effort that their production must have involved. *Aids to ophthalmology* is packed with information and represents extremely good value for money. If I had to choose one to take on a desert island I would probably choose *Synopsis* because the delivery of information is so smooth.

Finally, I noticed that Duke-Elder's *System of ophthalmology* did not appear in the suggested reading at the end of *Aids*. I wonder if we could persuade the energetic authors of these two books to update the *System of ophthalmology*; this might once again mean that the definitive ophthalmic textbook came from the United Kingdom.

JOHN BRAZIER

Manual of Retinal Surgery. Edited by Andrew J Packer. Pp. 126. £29.95. Churchill Livingstone: Edinburgh. 1989.

This manual has been collected by a number of well-known American vitreoretinal surgeons. Nine chapters make up its contents, compressing much valuable information into a short book. It forms an admirable way for a junior ophthalmologist to get a quick overview of the principles of an approach to a patient with retinal detachment. As always in a book of this size there is a challenge to the contributors to limit their comments to what they consider to be the most important topics. It was nice to see the chapter by Chang emphasising the need for careful preoperative examination and charting, though it had not occurred to me that the nasal periphery of the retina is easier to examine after indentation of the temporal periphery has softened the eye. There are useful comments on the use of preoperative ultrasound, and the chapter on prophylaxis poses the questions but is not strong enough on providing clear-cut guidance on the author's views as to when treatment should be recommended. The controversial topic of pneumatic retinopathy is included, and there is a welcome, but relatively long contribution on the anaesthesia for surgery. The section on scleral buckling surgery is full of useful practical information, but sadly the principles governing non-drainage retinal detachment surgery are poorly explained and some of the indications for encirclement (the treatment of aphakic retinal detachment) are contentious. It would also have been better if the principles of peroperative injection of air and the operative sequence necessary for its correct use had been clearly elucidated.

The chapter on posterior segment vitrectomy by Abraham is particularly effective. The length of this section reflects the increasing use of vitrectomy in modern vitreoretinal surgery. The author cleverly manages to incorporate a substantial degree of sophistication in the techniques that he describes. These techniques are of course well beyond the capabilities of those for whom this manual is intended, but it