LETTERS TO THE EDITOR

Peribulbar anaesthesia

SIR,—Joseph *et al*[1] report a case of globe penetration occurring as a complication of peribulbar anaesthesia. They highlight the potential risks of this procedure which have been previously reported.[2,3] They also advocate the use of short blunt needles and the possible use of subconjunctival administration of the anaesthetic in order to minimise the risk of globe penetration. Although the case presented does indeed demonstrate the possible hazard of peribulbar anaesthesia the authors fail to provide any convincing evidence to support their proposals.

The length of the needle used in the presented case is irrelevant as the penetration occurring at the equator could still have been caused by a shorter needle. Although the use of a shorter needle has been described[4,5] and should reduce the likelihood of posterior entry and exit wounds (as occurred in the series examined by Duker *et al*[2]) and optic nerve injury[6]; penetration at the equator and anterior to this region will not be prevented if the needle tip is misplaced. Even in the emmetropic patient it is usually possible to indent the equatorial retina, for the purpose of funduscopy, by pressure on the skin surface. It is then easy to imagine the length of needle required to traverse the distance between skin surface and equatorial retina being less than 2 cm.

The lack of sharpness of the Atkinson needle in the case presented did not prevent globe penetration. Indeed it is possible that the blunt tip actually compresses the tissues ahead of the tip thus giving a misleading impression of depth of injection.

The equator of the globe, with the eye in the primary position, is the greatest diameter in the coronal plane. Any needle entering the orbital region anteriorly must be directed in such a manner as to avoid encountering the sclera. Only by accurately judging the position of the equator can a needle be inserted in safety. A technique of indentation, via skin or conjunctiva, can be used to judge the limits of the globe and, if uncertain, examination of the retinal indentation just prior to the injection could confirm the identification of the equator.

The use of the subconjunctival route for local anaesthesia has been described[7,8] as a satisfactory method, and could be expected to lead to a reduced risk. Unfortunately even this still carries some risk of penetration as illustrated by Yanoff,[9] and the length of the needle is even less important in this technique as there is less tissue for an errant needle to traverse before reaching the globe.

In summary this case indeed confirms the findings of others[2,3] that peribulbar injections may be dangerous, despite the advantages over the retrobulbar route.[10,11,12] However it is doubtful that a shorter needle would have prevented penetration – in this case of globe penetration. The blunt needle has been shown here to have its limitations; in addition subconjunctival injection does not virtually eliminate risk of penetration as stated by Joseph *et al*. Surely one can only draw the conclusion that all needles in the orbit are potentially hazardous in the wrong hands, and that careful supervision and training in technique have far more relevance than the type of needle used in the administration of local anaesthesia prior to ocular surgery.

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Reply

SIR,—Our purpose in reporting this case was to highlight the risks of peribulbar anaesthesia, a procedure which has been said to have a low risk of globe perforation. Since our report went to press additional cases have been described,[1,2,3] suggesting that perforation of the globe due to peribulbar anaesthesia occurs more frequently than was previously supposed.

The length of a normal globe from anterior cornea to the macula is approximately 2·5 cm, the distance to the equator about half this, and it is of course possible to penetrate the globe posterior to the equator with very short needles indeed. A short needle is recommended for peribulbar injections for a number of reasons. Firstly, a 2 cm needle is more than long enough for the job. Secondly, a needle of this length should make injury to the optic nerve and injection of anaesthetic into the subarachnoid space impossible. In addition the risk of injury to the other nerves and vessels in the orbit should be reduced. Thirdly, a short needle should reduce the risks of posterior perforations of the globe as it is unlikely to penetrate sufficiently deeply for this to occur. Fourthly, a short needle affords greater sensitivity to the position of its tip than a long one as anyone who has used intravenous cannulas will attest. A greater awareness of the position of the needle tip should reduce the risk of globe penetration. A blunt needle is recommended as it is less likely to penetrate the globe than a sharp needle although in the myopic sclera described in our report this was clearly not the case. Blunt needles are also recommended as they afford greater sensitivity to the tissue planes, thereby providing the operator with further information about the position of the tip of the needle.

Mr Hawksworth rightly points out that all sharp needles should be used with care and caution around the eye. We believe that this would have no role in making orbital anaesthetic injections safer.

While clearly the subconjunctival route for injection of anaesthetic is not without some risk it must be safer to introduce a needle under direct vision below the conjunctiva rather than to direct it 'blindly' into the orbit.

We would also like to point out that the peribulbar anaesthetic in our case was administered by an experienced, well trained ophthalmologist who subsequently performed the surgery. We believe the complication of globe perforation could happen to any surgeon and merely reflects the hazards of needles in the orbit. The use of a short blunt peribulbar needle would be expected to reduce the risk of globe penetration but not eliminate it altogether, even in the most skilled hands.

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1 Duker JS, Belmont JB, Benson WE, *et al*. Inadvertent globe perforation during retrobulbar and peribulbar anesthesia. *Ophthalmology* 1991; **98**: 519–26.
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3 Hay A, Flynn HW, Hoffman JI, Riviera AH. Needle penetration of the globe during retrobulbar and peribulbar injection. *Ophthalmology* 1991; **98**: 1017–24.

The continuing challenge of ocular leprosy

SIR,—Always I read with great interest Mr T J ffytche's articles and comments on ocular leprosy and I appreciate his views. However, he writes in his mini review[1] 'Recent work by Hogeweg *et al*[2] on the relationship between the position of facial patches during erythema nodosum leprosum (ENL) reactions, and the subsequent development of lagophthalmos has considerably helped to identify those patients most at risk.' This quotation may cause confusion because this article deals with type I (reversal) reactions. ENL reactions have neither relationship with facial patches nor with lagophthalmos.

Secondly, corticosteroids are generally considered as the treatment of choice in reversal reactions. The use of clofazimine is controversial and thalidomide is considered to be ineffective in reversal reactions.[3,4]

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2 Hogeweg M, Kiran UK, Suneetha S. The significance of facial patches in type I reactions for the development of facial nerve damage in leprosy. *Lepr Rev* 1991; **62**: 143–9.
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Reply

SIR,—I quite agree with Dr Hogeweg's comments concerning the relationship of facial patches with leprosy reactions and lagophthalmos. This occurs exclusively in the type I (reversal) reaction, and not in ENL. This was an error in the text for which I apologise. The

treatment in this acute situation consists of systemic steroids to suppress the reaction. High doses of clofazamine and thalidomide have been tried in cases where steroids are unobtainable or contraindicated but their action is too slow and they are generally ineffective.

One other error in the article, which escaped both our notice, was the statement that paralysis of the maxillary branch of the facial nerve causes lagophthalmos. This should of course have read 'the zygomatic branch', and I am grateful for this opportunity to rectify this.

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Fluphenazine induced welding arc maculopathy?

SIR,—Powers *et al* have presented the case of a 45-year-old male welding trainee who incurred bilateral maculopathy supposedly due to unprotected exposure of less than 2 minutes' duration to a manual metal arc welding unit.[1] They believe that fluphenazine, which had accumulated in his retinal pigment epithelium (due to 10 years' treatment for depression), rendered him particularly susceptible to retinal damage.

The authors further state that 'one of the unusual features of this case is that though the patient was only welding for two minutes he developed a bilateral maculopathy without any evidence of a keratitis'. In the early part of the paper they mention that 'there was no accurate record of the exact duration of exposure'.

Is it not possible then, that: (1) the exposure might have been actually longer than 2 minutes; (2) fluphenazine may have accumulated in corneal tissues but rendered them more resistant to photic damage thus explaining the lack of keratitis in the presence of maculopathy?

This paper inspires one to conduct an experimental study on animals put on long term fluphenazine. Noting any accumulation of the drug in corneal tissues and the thresholds for photic keratopathy seems in order.

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1 Power WJ, Travers SP, Mooney DJ. Welding arc maculopathy and fluphenazine. *Br J Ophthalmol* 1991; 75: 433–5.

Reply

SIR,—In reply to Dr Neki's letter we would like to make the following points:

(1) Although there was not an exact record of the involved exposure time both the patient and the instructor were quite sure that the exposure duration was no longer than 2 minutes.

(2) Dr Neki suggests that fluphenazine may have accumulated in the patient's cornea and may therefore have rendered it more resistant to photic damage. He further suggests that this may then explain the lack of keratitis in the presence of maculopathy. However the literature would suggest otherwise. In a study on the concentration of phenothiazines in the eye of experimental animals Potts[1] has shown that the concentration of prochlorperazine in rabbit cornea is less than 100th that of the concentration in choroid. He found similar results when he looked at the ocular distribution of phenothiazine in hamsters. It would seem unlikely, therefore, that fluphenazine – a phenothiazine derivative – would accumulate in corneal tissues to a significant degree.

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1 Potts AM. The concentration of phenothiazines in the eye of experimental animals. *Invest Ophthalmol* 1962; 1: 522–30.

BOOK REVIEWS

Vascular Disorders of the Ocular Fundus. By Rodney Grey. Pp 118. £47·50. Butterworth: London, 1990.

The purpose of this book is to demonstrate in colour the photographic appearances of a variety of retinal conditions not all, it has to be admitted, strictly 'vascular' in origin although, in the retina, vessels of some sort are rarely far away.

The book is a great success. After preliminary general observations on normal anatomy and pathology, methods of examination, principally fluorescence angiography, and the effects of treatment with photocoagulation, the book then covers each condition separately.

A fairly brief but very much to the point description is accompanied by well chosen and beautifully reproduced illustrations. The writing is lucid and therefore a pleasure to read. Afficionados of retinal disease might perhaps question some parts in detail but the average general ophthalmologist, and certainly all postgraduate students, will find the simplicity and clarity of the text most enlightening.

It is traditional to find a few faults. There are a few minor proof reading errors, such as the rather jolly mis-spelling of Dr Dollery's name on pp 6, 12 and 18 and possibly a transposition of two photographs on p 66.

In summary this book is highly recommended.

REDMOND SMITH

Angle-Closure Glaucoma. By Stanley Hyams. Pp 186. Dfl 90.00. Kugler: Amsterdam, 1990.

This small book covers the subject of angle-closure glaucoma in an original and informative manner with special attention paid to the bibliography on which the current understanding of angle-closure glaucoma is based. The major difference between this book and standard texts on the subject is that each chapter is composed of approximately equal portions of text and the author's own abstracts of the papers from which this knowledge has been drawn. This original style of presentation will appeal more to the reader with a specific interest in angle-closure glaucoma than perhaps an ophthalmologist in training who wishes to gain a rapid and easily remembered understanding of the subject. However, this style does allow the reader to cover a very wide range of references quickly without a visit to the library, and makes very interesting reading. It is salutary to find out just how much of our knowledge and accepted teachings are based on small series of patients and case reports – especially with regard to rarer conditions such as malignant glaucoma.

The book is subtitled 'A comprehensive review of primary and secondary angle-closure glaucoma' and certainly the interested reader will find much useful information which does not appear in standard texts, for example the chapters on 'Prevalence' and 'Biometrics.' Most chapter headings are enlivened with relevant quotations from historical writings which show the astuteness and common sense of our predecessors. The author helps to clarify the nomenclature and hence classification of glaucomas associated with narrow and closed angles, and should be applauded for his efforts as accuracy in definition is of such importance. His message on another confused area, that of the role of provocative tests was not quite so clear, however, and the reader is given a rather ambivalent message about their use. In discussing laser iridotomy the author should have made a clear distinction between argon and Nd:YAG laser iridotomy as the reader otherwise gains the impression that the complications attributed to 'laser iridotomy' are equally common with both lasers.

The lack of illustrations, clinical photographs and diagrams, makes this book appear rather stark and this will detract from its appeal, as the modern reader has come to expect the understanding and reinforcement of ideas that good illustrations give to a text's message. However, the readability of this book and its important content make it a useful addition to an ophthalmic library.

PETER K WISHART

OBITUARY

SATISH KUMAR BHARGAVA, *MB, BS, DO, FRCSE, FCOphth*

Satish Bhargava was born in Lahore in 1939 but moved to Delhi with his family in 1947 on Partition. He was one of the first intake of students at the All India Institute of Medical Sciences from which he qualified in 1962. After an internship there he came to England to take up a casualty officer post at Lister Hospital, Hitchin, where he met his future wife.

He started training in ophthalmology at the West of England Eye Infirmary, Exeter in 1963, continued at the Glasgow Eye Infirmary in 1965, and returned to Exeter as Registrar in 1968. He became senior registrar to the professorial unit at Manchester Royal Eye Hospital in 1971 where he became interested in retinal pathophysiology.

On appointment to the consultant staff there in 1975 he developed an electrophysiological diagnostic service and a referral clinic for the management of inherited retinal disorders. Both on his own and in collaboration with the Department of Optometry and Vision Science at the University of Manchester Institute of Science and Technology he was responsible for a steady stream of publications in this field, as well as on colour vision and toxic amblyopia.

In addition to running a very busy unit at MREH, where he was chairman of the medical committee twice (1981–3 and 1987–9), he also developed a close relationship with the Manchester department and School of Orthoptics, of which he was medical director from 1986–9. He frequently examined for the Orthoptic