and biological tissues¹ and more research is needed. When a masking agent is used, a fan causing ripples on the surface is undoubtedly undesirable.

Masking agents provide the key to producing a regular surface from an irregular one and must not be dismissed. Gartry *et al* obtained superior results because great care was taken continuously to refill and uniformly to redistribute the masking agent over the ablated area and constantly to monitor the changes in tissue fluorescence.¹ This is a surgical art rather than 'worshipping at the altar of high technology.'

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- 1 Gartry D, Kerr Muir M, Marshall J. Excimer laser treatment of corneal surface pathology: a laboratory and clinical study. *Br J Ophthalmol* 1991; 75: 258-69.
- 2 Sher NA, Bowers RA, Zabel RW, Frantz JM, Eiferman RA, Brown DC, *et al*. Clinical use of the 193-nm excimer laser in the treatment of corneal scars. *Arch Ophthalmol* 1991; 109: 491-8.
- 3 Olson RJ. The altar of high technology and the excimer laser. *Arch Ophthalmol* 1991; 109: 489-90.

Reply

SIR,—Following on the interesting comments made by Dr Liu in his letter we reported¹ that superficial keratectomy with the excimer laser results in a hyperopic shift of 2.85 dioptres on average in our series. This was an unexpected finding since we had assumed that photoablation of an even layer of tissue across the entire cross-section of the beam (4 mm in diameter in most cases) would produce no change in corneal curvature. This is in contrast to the planned change in corneal curvature attempted when a motorised iris diaphragm is used during photorefractive keratectomy.

We put forward four possible mechanisms to explain this phenomenon. (1) The distribution of calcium within the band keratopathy might mask the beam allowing greater ablation of tissue centrally. (2) A centrifugal contraction of the lamellae at the ablated surface. (3) Shielding of the periphery of the beam by debris ejected from the surface. (4) Increased obliquity of incident radiation at the edge of the beam (due to the curvature of the cornea) resulting in slightly less photoablation in the periphery of the ablation zone.

We described the treatment of two main categories of anterior corneal pathology — namely rough surfaces and smooth surfaces. The background and principles of superficial keratectomy with the excimer laser to smooth a rough surface were documented in detail and in particular the importance of masking areas of stroma between surface peaks to avoid reproducing the surface irregularity deeper into the cornea. We found 1% methyl cellulose optimal for this purpose. When treating the second category (for example smooth band keratopathy) since we started with a smooth surface it was unnecessary to apply any masking agent. As we explained in our paper in general the eyes with the best visual potential fell into this latter category and it was in these cases that subjective refraction revealed a hyperopic shift (cases 18, 19, 21, 22 and 23).

Dr Liu suggests that when using overlapping ablation zones more tissue will be removed from the central cornea due to intersection of treated areas. This is not the explanation since we found considerable hyperopic shift in patients treated with a single axial zone (for example cases 22 and 23). In addition, in those patients with good visual potential treated with

multiple zones a 4 mm central zone was usually cleared first followed by continuous areas with minimal overlap. He also states that 'it is conceivable that both regular and irregular astigmatism could be created in the same way.' We agree with this of course since we documented the effect in case 19. As stated in our paper it was this potential for irregular astigmatism when using multiple continuous zones that led us to conclude that where good visual potential exists a single central zone is the most effective treatment method.

Dr Liu's second suggestion to explain the hyperopic shift relates to the use of methyl cellulose and since this was only employed (as stated) in cases with rough corneal surfaces (where the aim was smoothing rather than glare reduction and improvement in acuity) this cannot provide a basis for the effect.

The third mechanism proposed however — that of epithelial hyperplasia — warrants further consideration. We have found that regenerating epithelium accounts for a large part of the regression in refraction occurring in our photorefractive keratectomy series. We have hypothesised that epithelial hyperplasia over the ablated zone accounts for the first part of an asymptotic regression towards a plateau with stromal remodelling providing the basis for subsequent lesser change. It is difficult to envisage that entirely the reverse could occur when treating smooth band keratopathy. We also suggest that any 'relative concavity' due to hyperplasia at the edge of the ablated zone would be minimal since it is necessary only to ablate between 10 and 15 µm of stroma (virtually only Bowman's layer) to clear smooth band keratopathy and this is a shallow ablation compared with the thickness of the overlying regenerated epithelium.

Of the four mechanisms proposed in our paper to explain the hyperopic shift we favour centrifugal contraction of lamellae resulting in a flattening of central cornea. This concept will be clarified further and evidence in its support provided when we report our photorefractive keratectomy series.

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- 1 Gartry D, Kerr Muir M, Marshall J. Excimer laser treatment of corneal surface pathology: a laboratory and clinical study. *Br J Ophthalmol* 1991; 75: 258-69.

Treatable blindness in temporal arteritis

SIR,—I read with interest the recently reported case.¹ The author reported dramatic visual recovery in the second episode of anterior ischaemic optic neuropathy (AION) when pulsed IV steroid therapy (methylprednisolone, 500 mg daily for 2 days) was used, and recommends this treatment for visual loss due to AION caused by giant cell arteritis (GCA) despite its attendant mortality and morbidity.² Clearly this recommendation can only be accepted if a clear benefit of this treatment can be demonstrated over the more conventional steroid treatment of 80-100 mg prednisolone orally.³ I would suggest there is no evidence of such benefit particularly as, during the first episode of AION this patient suffered, there was an equally impressive recovery of vision on 60 mg of prednisolone orally daily. It seems reasonable to suppose that this may have again occurred had conventional therapy been used.

As the author points out when there is severe

visual loss from AION secondary to GCA marked improvement is rare although several other cases of substantial visual improvement using 80 mg prednisolone orally daily are reported.^{3,4}

Pulsed IV steroids are recommended by the standard reference textbook of ocular pharmacology which states 'with acute visual loss of less than 36 hours duration significant visual improvement may be obtained with 'pulsed' steroid therapy using 1 gm of methylprednisolone intravenously every 12 hours for 5 days.'

Only two case reports are cited as evidence to support this statement, and both are discussed by Mr Diamond. However neither is particularly persuasive.

In the case reported by Model⁶ vision had at no stage been formally tested. In addition prior treatment had been inadequate and of short duration (a single intravenous dose of 100 mg of hydrocortisone and no more than 20 mg of methylprednisolone orally (10 mg four times daily for 12 h). It is therefore uncertain what the degree of alteration in vision was and whether pulsed IV steroid was essential to accomplish it.

In the other case⁷ vision in the eye for which pulsed IV steroids were given was 20/70 before the patient was referred for a second opinion. When assessed immediately prior to pulsed IV steroids, vision was 20/40 ('slowly'), with an inferior altitudinal scotoma to 14e on Goldmann perimetry and ability to recognise only seven of 15 colour vision plates. Following pulsed IV steroids the field loss recovered and visual acuity improved to 20/25. No details are given of how quickly she read or of colour appreciation. The authors claim to have 'restored vision' seems to overstate the case. More accurately they seemed to have arrested moderate fluctuations of vision. It is worth emphasising that, judging from the recorded changes in visual acuity, the majority of improvement (20/70 to 20/40) had occurred before pulsed IV treatment was used (visual acuity improved from 20/40 to 20/25 following this treatment) and it may be argued that recovery was inevitable in any event.

Recovery of vision on AION due to GCA is by no means invariable with pulsed IV steroids and progressive visual loss during therapy has been reported.⁸

Although appropriateness of corticosteroids in the treatment of GCA with starting doses of 80-100 mg orally is well established,³ it is apparent that the evidence to support the use of pulsed IV treatment in order to restore visual function lost from AION due to GCA is deficient. In view of the potential for adverse effects (in the age group most susceptible to adverse effects of steroid) formal critical evaluation of any therapeutic efficacy is required before recommendations for use of this treatment can be accepted.

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- 1 Diamond JP. Treatable blindness in temporal arteritis. *Br J Ophthalmol* 1991; 75: 432.
- 2 Thompson JF, Chalmers DH, Wood RFM. Sudden death following high dose intravenous methylprednisolone. *Transplantation* 1983; 36: 594-6.
- 3 Graham E, Holland A, Avery A, Russell RWR. Prognosis in giant cell arteritis. *Br J Med* 1981; 282: 269-71.
- 4 Clearkin LG, Caballero JC. Recovery of visual loss in giant cell arteritis (in press).
- 5 Breen LA, Burde RM. Temporal arteritis. In: Fraunfelder FT, Roy FH eds. *Current ocular therapy*. Philadelphia: W B Saunders 1985: 116-7.

- 6 Model DG. Reversal of blindness in temporal arteritis with methylprednisolone. *Lancet* 1978; i: 340.
- 7 Rosenfield SI, Kosmorsky GS, Klingele TG, Burde RM, Cohn EM. Treatment of temporal arteritis with ocular involvement. *Am J Med* 1986; 80: 143-5.
- 8 Slain ML, Margolis AJ. Progressive anterior ischaemic optic neuropathy due to giant cell arteritis despite high dose intravenous corticosteroids. *Arch Ophthalmol* 1988; 106: 1167.

Reply

SIR, —I agree that the possible adverse effects of this drug must be carefully weighed against the potential benefits and moreover that the efficacy of the drug requires a full critical evaluation.

However until this evidence is forthcoming I consider that methylprednisolone does have a role in treatment of certain cases of temporal arteritis.

While methylprednisolone is a commonly used drug the number of serious side effects reported is low. Conversely there is a significant morbidity and mortality consequent upon bilateral visual loss in the elderly.

Thus I consider use of methylprednisolone is justified when blindness is threatened; for example, in patients who develop second-eye symptoms when already taking a significant dose of oral prednisolone or for patients who delay presentation until after the second eye has failed.

Total blindness is a catastrophic complication of temporal arteritis. The potential prevention of this outcome would seem to justify utilisation of a relatively safe if unproved therapy.

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BOOK REVIEWS

Converting to Phacoemulsification: A Manual for the Surgeon in Transition. 2nd edn. By Paul S Koch. Pp 84. £24.00. Wiley: Chichester, West Sussex, 1988.

I was particularly delighted to review this book because when I was in the early stages of converting to phacoemulsification for the management of cataract patients this was a book I purchased and consumed both with pleasure and benefit. It is a slim volume designed specifically to encourage and assist the surgeon undergoing the process of converting to phacoemulsification. This process can be uncomfortable and painful from time to time and encouragement and assistance are both very welcome. The author's style is distinctly chatty but is always very much to the point.

In its 12 short chapters it covers every aspect of the procedure and the important points are illustrated by simple line drawings. No important technical point is omitted. I should, however, point out that in my experience no textbook can replace the value of an instruc-

tional course when it comes to phacoemulsification. This is a subtle surgical procedure requiring skill and care and it is much less comfortable to discover its pitfalls by unguided surgical experience rather than to have been forewarned and forearmed against them. The greatest strength of this little book is its encouraging tone and the author's unwavering belief that what he has to say is in both the surgeon's and the patient's best interests. If it has a weakness it is that it does not take account of some of the more recent developments in cataract management, particularly capsulorhexis and phacoemulsification within the capsular bag. These are, however, techniques which a converting cataract surgeon does need to acquire quite early. I hope that future editions of this volume will cover this omission.

A D MCG STEELE

Sodium Hyaluronate in Anterior and Posterior Segment Surgery. By R G Michels, W J Stark, M Stirpe. Pp 131. US\$ 35. Liviana Press: Via L Dottesio, 1-35138 Padova, Italy, 1989.

This book is dedicated to the use of sodium hyaluronate in all aspects of intraocular viscosurgery. It is edited by a team of well known American and Italian ophthalmologists, and the manufacturers of the product appear to have played a prominent part in the production of the book.

The text is relatively short but clear. The introductory section deals with the chemistry, physics, and physiological properties of sodium hyaluronate. This is followed by four chapters about the clinical applications of this undoubtedly useful substance. Having discussed its use in surgery of the anterior segment and glaucoma, the editors devote the largest section of the text (and the largest volumes of sodium hyaluronate) to surgery of the posterior segment. There are many colour paintings which make the procedures look deceptively simple and free of complications.

All in all this is a beautifully illustrated book which is easy to read, but unless there is a sudden change in the price of sodium hyaluronate it is likely to be limited value to most readers in Britain.

Z GREGOR

Noninvasive Diagnostic Techniques in Ophthalmology. Edited by Barry R Masters. Pp 650. Dm 298.00. Springer-Verlag: Heidelberg, 1990.

This book is an excellent production comprising more than 600 pages and 32 chapters on up-to-date methods of investigation in our specialty. The range of topics covered is wide, varying from magnetic resonance imaging through holographic contour analysis of the cornea to scanning laser and image analysis of infrared choroidal angiography.

Many of the contributors are scientists and not clinical ophthalmologists but their chapters are full of interest though several admit the clinical application of their expertise awaits analysis. Most chapters end with some paragraphs on the present clinical application of the method described and also look into the future regarding potential developments. The references provided are full and frequent sugges-

tions for further reading are included. Many of these references are in non-ophthalmic journals and monographs and the reader cannot fail to recognise the tremendous contributions being made to the advancement of knowledge by scientific workers in our field.

The whole book is fascinating and of immense value to those in the clinical field who may have access to the expensive research instrumentation and the expert knowledge of the relatively few people who know how to manipulate the software and hardware required.

I have the feeling though that, rather like encyclopaedias, the information will rapidly become dated as new devices are developed. I wonder if the editor might consider producing an update at periodic intervals to permit this current publication to remain as the authoritative textbook of noninvasive techniques as cleverly defined in the foreword.

I will enjoy having access to this beautifully presented book with its fascinating photographs and hopefully the possibility will arise of the clinical application becoming available to an increasing number of workers as time and finance become available.

WILLIAM M DOIG

All titles reviewed here are available from the BMJ Bookshop, PO Box 295, London WC1H 9TE. Prices include postage in the UK and for members of the British Forces Overseas, but overseas customers should add 15% to the value of the order for postage and packing. Payment can be made by cheque in sterling drawn on a UK bank, or by credit card (Mastercard, Visa, or American Express), stating card number, expiry date, and full name.

NOTES

International Society of Ocular Trauma

The Society will hold its 2nd International Symposium on Ocular Trauma on 2-5 April 1992 in Geneva, Switzerland. Further details from: The Secretariat, 2nd International Symposium on Ocular Trauma, c/o Kuoni Travel Ltd, 7 Rue de Berne, CH-1211 Geneva 1, Switzerland (tel: 41 22 732 0888; fax 41 22 731 5078; tlx: 412831).

Welsh Cataract Congress 1992

The Welsh Cataract Congress will be held on 10-12 September 1992. Further information from: Eula Mae Childs, Cullen Eye Institute, Baylor College of Medicine, 6501 Fannin (NC200), Houston, TX 77030, USA. Tel: 713-798-5941.

Biomedical Optics '92

The International Society for Optical Engineering will hold a symposium under this title on 19-24 January 1992 at Los Angeles Airport Hilton Hotel. Details from the Society at PO Box 10, Bellingham, Washington 98227-0010, USA.