

MAILBOX

Comparative study of intraoperative mitomycin C and β irradiation in pterygium surgery

EDITOR,—We read with interest the study that compared intraoperative mitomycin C with β irradiation in primary pterygium surgery.¹ The authors rightly commented that long term complications of β irradiation, such as scleral necrosis, may arise more than 10 years after the irradiation.² Longer follow ups are necessary to reveal such complications.

We performed primary pterygium excision with intraoperative β irradiation in one eye of six patients between 1988 and 1990. A dose of 1000 rad of β irradiation was applied to the scleral bed intraoperatively and 1 week later. The patients were recently reviewed in our clinic for recurrence and complications. We also performed ultrasound biomicroscopic examination (UBM) for both eyes in each patient, looking for corneal and scleral thinning. Corneal thickness was arbitrarily measured 0.5 mm anterior to the scleral spur at the 12, 3, 6, and 9 o'clock positions of each eye, while the scleral thickness was measured 2 mm posterior to the scleral spur at the same positions.

Mean follow up was 138.0 months. Mean age at time of surgery was 37.5 years (range 32–45 years). All six eyes were right eyes with nasal pterygia in male patients. No recurrence was found, using the same definition. There was neither significant deterioration in visual acuity nor increase in intraocular pressure in any eye. There were no signs of inflammation.

There were no significant differences in the scleral and corneal thickness between the treated nasal position of the operated eye (mean scleral 0.617 (SD 0.112) mm; mean corneal 0.656 (0.076) mm) and the control nasal position of the fellow eye (mean scleral 0.611 (0.030) mm; mean corneal 0.645 (0.044) mm).

Furthermore, there were no significant differences in the mean scleral and corneal thickness between the operated eye (scleral 0.590 (0.077) mm; corneal 0.635 (0.067) mm) and the fellow eye (scleral 0.590 (0.059) mm; corneal 0.624 (0.054) mm). The mean scleral and corneal thicknesses were calculated by averaging the scleral or corneal thickness at the four measured positions in each eye.

It appears that β irradiation is safe, even in the long term. We believe these additional data could supplement the findings by Amano *et al.*

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Visual field defects after vitrectomy with fluid-air exchange

EDITOR,—The paper by Cullinane and Cleary¹ presents an excellent prospective study of peripheral visual field loss in patients undergoing macular hole surgery. The authors compared vitrectomy with complete posterior cortical vitreous peeling to limited vitrectomy with removal of cortical vitreous off the macula, but not off the optic nerve head or the peripheral retina. The authors showed a statistically significant decrease in peripheral visual field defects with the limited vitrectomy technique (0%, 0/22 patients) compared with the complete vitrectomy group (22%, 18/82 patients).

The authors postulated that this difference is due to the avoidance of traction on the optic nerve head during peeling of the posterior hyaloid, thus limiting damage to the peripapillary nerve fibre layer, which they believed would be most severe nasally because of firmer vitreopapillary attachments nasally. This explanation does not take into account the variable position of visual field defects found in other studies based on the position of the infusion cannula. If the infusion cannula is superiorly located, visual field defects occur superiorly, implicating inferior retinal damage.² If the infusion cannula is inferonasal, visual field defects occur inferonasally and not inferotemporally.³ The inferotemporal location of field defects noted in most studies is based on the conventional placement of the infusion cannula inferotemporally in three port vitrectomy, which results in infused air directed towards the superonasal mid-peripheral retina.

Animal studies show damage to the inner limiting membrane, nerve fibre layer, and ganglion cells of the retina in the path of the pressurised air flow from the infusion cannula.^{4,5} This inner retinal damage could be caused by desiccation of the retina² or by direct mechanical damage by the pressurised air flow.^{4,5} However, humidification of air did not prevent inner retinal damage in animal models,^{4,5} and the sharp demarcation between damaged and undamaged retina on electron microscopic studies supports the theory of direct mechanical damage to the inner retina.⁴ In addition, decreasing the infusion air pressure also decreased the risk of inner retinal damage.⁵ What I think this work by Cullinane and Cleary shows is that leaving the peripheral vitreous in place is another way of protecting the peripheral retina from mechanical damage by pressurised air flow. However, I would be concerned about the potential risk of increased postoperative retinal detachment, which was 10% in the limited vitrectomy group and 4% in the complete vitrectomy group, but was not statistically significant because of small sample size. However, this increased risk of retinal detachment was also a concern in a previous study utilising similar surgical techniques (Brian Conway, Western Association for Vitreoretinal Education Meeting, Maui, Hawaii, 1996).

Because of the studies on retinal damage by pressurised air infusion and the significance of high infusion air pressure, it would be important to know the usual infusion air pressure utilised during fluid-air exchange by the authors, and if the infusion air pressure varied

at any point during the period of the study or between the two vitrectomy groups. Currently, in order to minimise retinal damage induced by pressurised air infusion during vitrectomy for any surgical indication requiring fluid-air exchange, I would recommend simply using a low infusion air pressure.

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Sclerotomy complications following pars plana vitrectomy

EDITOR,—The work of West and Gregor again points out the importance of sclerotomy complications following pars plana vitrectomy.¹ They demonstrate that, even in the hands of a skilful and experienced surgeon, vitreous haemorrhage after vitrectomy for diabetic retinopathy is common and requires vitreous cavity washout (VCWO) in 12% of cases. In their series, over half of the eyes had detectable fibrovascular ingrowth (FVI) as the cause of the haemorrhage.

Interestingly, in this case series of 159 eyes, no occurrences of anterior hyaloidal fibrovascular proliferation (AHFP) were noted. Definition of the relation between these two entities has been controversial, to say the least.

Part of the controversy is due to a misunderstanding of the nature and pathogenesis of FVI. As McLeod points out in his editorial, FVI is a term that has been used inappropriately, suggesting that episcleral tissue grows into the eye through the sclerotomy incision.² While episcleral tissue, scleral fibroblasts, and ciliary epithelium all contribute, the majority of the fibroproliferative healing of a sclerotomy originates from the uvea of the ciliary body.³

In normal wound healing, early fibrovascular proliferation in the incision is followed by its involution and contraction, with the result being the small scar seen at the internal aspect of a healed sclerotomy.³ Inevitably, because of the proximity of the vitreous base and anterior hyaloid, vitreous strands are adherent to the wound and fibrous tissue extends a short way into the vitreous body. This tissue may contain blood vessels, even with normal healing. From this perspective, all sclerotomy wounds heal with fibrovascular ingrowth. That is, ingrowth of tissue from the eye wall extends into the vitreous cavity. Fortunately, only in unusual circumstances does this process become exaggerated and result in what clinicians have termed FVI with its concomitant intraocular mischief.⁴

McLeod pointed out that ischaemia is an important factor in inducing FVI and that it is seen mainly following vitrectomy for ischaemic retinopathies. I agree that this is the case if one includes anterior proliferative vitreoretinopathy (APVR) in this group. Patients with APVR who have had previous vitrectomy frequently have an excessive amount of fibrovascular scarring from their sclerectomies, which significantly affects the pathological anatomy of the basal vitreous and its environs. These patients, however, often have had extensive scleral buckling and cryopexy, processes which undoubtedly induce some anterior ischaemia in themselves.³

In the series of West and Gregor, no patient was found to have a retinal detachment ultrasonographically or at the time of VCWO. In the original description of AHFP, most of the patients had retinal detachments that had required scleral buckling.⁵ Since retinal detachment and scleral buckling exacerbate anterior ischaemia, it is likely that AHFP, which is fibrovascular proliferation into the vitreous base from the retina and ciliary body, is induced by an ischaemic drive similar to that causing FVI. The two entities exist on a continuum. When there is a surgical injury such as a sclerectomy, with disruption of tissue and inoculation of blood into the surrounding vitreous, excessive proliferation may occur with less induction than that which causes AHFP.

Personally, although I have observed cases of AHFP without having previous vitrectomy, I have never seen a case of post-vitrectomy AHFP without some concurrent FVI.

Finally, I'd like to make two other points. The first is that West and Gregor used clinical criteria to determine whether or not FVI existed and caused the recurrent vitreous haemorrhage. I have observed vitreous haemorrhage in a necropsy eye from what grossly appeared to be a normally healed sclerectomy wound.⁶ Microscopically, that white scar contained numerous capillaries that were the source of the haemorrhage. Therefore, it may be that some of their non-FVI patients might actually have had vitreous haemorrhage from a subclinical FVI. Furthermore, FVI can involute with time, becoming less vascular in its appearance. So, the frequency of FVI may be even higher than reported.

Lastly, I agree that episcleral sentinel vessels, externally entering the wound site, sometimes, but not always, indicate a possible FVI. These vessels are the result of a high degree of metabolic activity during the healing of sclerectomy wounds and may persist even though wound fibroplasia becomes involutinal and clinically unimportant. Similar vessels are seen microscopically in the ciliary body.³ When present, sentinel³ vessels should raise our suspicions of FVI; but they do not rule it in, nor does their absence rule it out.

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Corneal transplantation: how successful are we?

EDITOR,—The commentary by Waldock and Cook¹ on the survival rates of corneal grafts highlights a number of issues. In particular, they focus on the lack of long term follow up data in the UK. The value of such data is clearly evident from the Australian Corneal Graft Register.² Moreover, in the present climate of clinical audit and evidence based medicine, the collection of such data has surely become a necessity.³ Many of the questions raised, whether simply comparing graft survival rates of individual units with national data or investigating more fundamental issues such as HLA matching, visual outcome, or surgeon experience require large amounts of data, properly designed studies, and appropriate statistical analysis—capabilities beyond most individual centres but readily achievable within the NHS. The way forward as shown by the organ transplant community, and to a certain extent by corneal graft surgeons, is through well organised, centralised data collection and analysis, for example.^{2,4,5}

The good news is that just such a system is now in place for all corneal graft surgeons in the UK. The Royal College of Ophthalmologists and UK Transplant (UKT) have initiated an Ocular Tissue Transplant Audit, which will provide the data for answering the sorts of questions posed by Waldock and Cook.¹ Indeed, the audit is already being used for data capture for the Corneal Transplant Follow-up Study II, which aims to resolve the uncertainty surrounding HLA-DR matching and corneal graft rejection. Instead of just 1 year follow up as in the original CTFS,² follow up for these patients will continue in the long term through the audit.

As important, however, is the opportunity for all ocular tissue transplants to be recorded and the outcome audited. Indeed one can foresee the day when this will be obligatory, as is the case with solid organs. To record such data with UKT will not only provide surgeons with details of their own activity, but with an independent confidential analysis of clinical outcomes, which they will increasingly be expected to have available.

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BOOK REVIEWS

Atlas of Vitreous Biomicroscopy. By CL Schepens, M Takahashi, CL Trempe. Pp 176; £75. Oxford: Butterworth-Heinemann, 1999. ISBN 0-7506-7052-5.

If only one had time in clinic to become fully dark adapted, instead of listening and talking to patients, all these illustrated wonders of vitreous architecture would yield to routine examination. As it is, much of what we perceive under unfavourable circumstances is, in fact, inferred although vitreous surgery provides regular opportunities for confirmation of the assumed pathology. This atlas of vitreous biomicroscopy provides a wealth of photographic documentation of vitreous disorders, especially as they affect transparent gel, and is supplemented by images of scanning laser ophthalmoscopy. Much of the material appears to have been published previously over many years and in a variety of journals, but that aspect of compilation and reproduction is welcome. However, the constantly recurring theme of a detached posterior hyaloid face, whether bounding a gel that has or has not collapsed, eventually tends to pall.

The text which accompanies the colour figures is parochial in content and disappointingly dull in places. An alleged 10% incidence of PVD in the fifth decade of life is surely peculiar to the Japanese population, and the customary obfuscation of the pathogenic sequence in advanced diabetic eye disease by the Boston group is reiterated. Stickler's arthro-ophthalmopathy isn't mentioned (only Wagner's disease under "degenerations") and PVD is said to be unusual in association with giant retinal tears. The description of asteroid hyalosis implies a bag of balls instead of strings of pearls, and the text and photographs of vitreous amyloidosis fail to inspire, omitting to mention precipitation of opacity on the otherwise transparent vitreous microarchitecture and thus revealing, for example, remnants of the tunica vasculosa lentis.

This atlas is more likely to figure on the departmental coffee table than in the clinician's own collection.

DAVID McLEOD

The Pupil. Anatomy, Physiology and Clinical Applications. Irene E Loewenfeld. £180; Pp 2223. Oxford: Butterworth-Heinemann, 1999. ISBN 0750671432.

This text represents a lifetime body of work for Professor Irene Loewenfeld. Perhaps more accurately, it represents a greater part of two lifetimes' work; having been commenced in the mid-1950s as a collaborative project with Professor Otto Lowenstein at the Columbia-Presbyterian Medical Center in New York. Following Otto Lowenstein's death in 1965, Irene Loewenfeld continued writing, eventually publishing through Wayne State University Press in 1993.

Presented in two volumes, the first includes the text and runs to 1645 pages, divided into five sections, while the second volume sensibly presents a separate bibliography thereby making the text (marginally) more transportable. The first volume is a comprehensive review of the anatomy and physiology of the pupil with its associated neurology and the diseases which play a part in compromising pupil function.

As a physiologist, Loewenfeld has written a book with a thorough foundation in basic science, with comprehensive discussion covering pupillary function across the animal kingdom, not simply restricting the project to humans.

Having been inscribed over a near 40 year period the text has a strong historical perspective, presenting research work in chronological order over a period during which understanding of pupil function has evolved. In striving to be comprehensive Irene Loewenfeld has included papers which may subsequently have been reinterpreted or simply proved incorrect. She has willingly injected a subjective flavour to the book when giving her own interpretation of earlier work which serves to make the text readable. This is also true for the bibliography where she includes "reference manager" style comments about the value of many references.

By its nature, such a reference tome can be difficult to "dip into". To assist those who may want rapid access to a subject each section is presented on three levels: a "thumbnail" summary for readers in a hurry; elaboration with historical perspectives for those with more time; plus an additional level with material delving into the background for readers keen to look to the source of understanding.

One section where clinical work may be underrepresented is the chapter on glaucoma. Here the text focuses on historical record of the pharmacological influence of drugs upon pupil function and their role in therapeutics. Recent clinical work on pupillometry in glaucomatous optic neuropathy aimed towards developing "pupil perimetry" has not been presented.

However, with this one exception, this text represents the definitive work upon the pupil which all ophthalmologists will find valuable, either as an introduction to the field or as the last word on the subject.

J P DIAMOND

Ocular Immunology in Health and Disease. By Steven B Koevary. £32; Pp 224. Oxford: Butterworth-Heinemann, 1999. ISBN 0750699000.

A pocket sized book with a very large remit is hard to achieve. Presently, there are several books on the market covering this increasingly popular topic, all with different emphases and depth of given information. Over the past 20 years there have been considerable developments in general immunobiology as well as immune responses and immune regulation of the eye and its adnexa. This book is a good and brave attempt to introduce ophthalmologists to immune mechanisms during health and disease. It begins with an overview of innate and acquired immunity and is a standard introduction to basic fundamentals in immunology. However, as a result of brevity, the book fails to do justice to the mechanisms of T cell activation, expansion and recruitment, interaction of cytokines and chemokines, and the role of tolerance and regulatory cells—all massive topics in their own right. Nevertheless the book maintains a steady pace and is easy to read and digest. Although more detail of immune mechanisms follows in subsequent chapters, an overview of T cell regulation and apoptosis, for example, would have been more appropriate at the beginning. All subsequent chapters are good and succinct, covering ocular immune privilege and tissue immunology, autoimmunity, allergy, allograft rejection, and tumour immunology. I

was surprised that there was no dedicated section on infection, but there again the book does succeed in being small, readable, and well referenced.

ANDREW DICK

NOTICES

Vision 2020: cataract outcomes

The latest issue of *Community Eye Health* (35) discusses cataract surgery outcome. For further information please contact *Community Eye Health*, International Centre for Eye Health, Institute of Ophthalmology, 11-43 Bath Street, London EC1V 9EL. (Tel: (+44) (0) 20-7608 6909/6910/6923; fax: (+44) (0) 7250 3207; email: eyeresource@ucl.ac.uk) Annual subscription £25. Free to workers in developing countries.

Second Sight

Second Sight, a UK based charity whose aims are to eliminate the backlog of cataract blind in India by the year 2020 and to establish strong links between Indian and British ophthalmologists, will be sending volunteer surgeons to India early in 2001. Details can be found at the charity website at www.second-sight.org.uk or by contacting Dr Lucy Mathen (email: lucymathen@yahoo.com).

Residents' Foreign Exchange Programme

Any resident interested in spending a period of up to one month in departments of ophthalmology in the Netherlands, Finland, Ireland, Germany, Denmark, France, Austria, or Portugal should apply to: Mr Robert Acheson, European Board of Ophthalmology, Institute of Ophthalmology, University College Dublin, 60 Eccles Street, Dublin 7, Ireland.

American Institute of Ultrasound in Medicine—Millennium Ultrasound Course Series

A course entitled "Obstetrical Ultrasound" will be held in Marina del Rey, CA, on 12-14 January 2001. Further details: Stacey Bessling, Public Relations Coordinator, AIUM, 14750 Sweitzer Lane, Suite 100, Laurel, MD 20707-5906, USA (tel: 301-498-4100; email: sbessling@aium.org).

Optometry Study Tour to Kenya, Tanzania, and Zanzibar

The tour offers a wonderful opportunity to optometrists and ophthalmologists to examine eye care in East Africa. It will take place from 28 January to 10 February 2001. Further details: Master Travel, Croxted Mews, 288 Croxted Road, London SE24 9BY (tel: 0208 678 5320; fax: 0208 674 2712; email: tours@mastertravel.co.uk).

First International Congress on Non-Penetrating Glaucoma Surgery

The First International Congress on Non-Penetrating Glaucoma Surgery will take place in Lausanne, Switzerland on 1-2 February 2001. Further details: Dr Tarek Shaarawy, Organising Committee, University of Lausanne, Hopital Ophtalmique Jules Gonin, Avenue de France 15, 1004 Lausanne, Switzerland (tel: 41 21 626 81 11; fax: 41 21

626 88 88; website: www.glaucoma-lausanne.org).

Call for papers—6th European Forum on Quality Improvement in Health Care, 29-31 March 2001, Bologna, Italy

Further details: BMA/BMJ Conference Unit, BMA House, Tavistock Square, London WC1H 9JP, UK (tel: +44 (0) 20 7383 6409; fax: +44 (0) 20 7383 6869; email: quality@bma.org.uk; website: www.quality.bmjpg.com).

XXV Detachment Course

The XXV Detachment course, retinal and vitreous surgery, will be held in Poznan, Poland on 5-6 April 2001. Further details: Professor Krystyna Pecold, Katedra I Klinika Okulistyki, ul Długa 1/2, 61-848 Poznan, Poland (tel/fax: 004861-8527619) or Professor Ingrid Kreissig, Univ-Augenklinik, Schleichstrasse 12, D-72076 Tuebingen, Germany (fax: 49-7071-293746; email: ingrid.kreissig@uni-tuebingen.de).

Optometry 01

Optometry 01 will take place on 21-23 April 2001 with more than 100 events—lectures and workshops—at the Atrium Gallery, NEC, Birmingham, UK. Further details: tel: 0207 261 9661; email: info@Optometry01.co.uk; website: www.optometry01.co.uk.

14th Annual Meeting of German Ophthalmic Surgeons

The 14th Annual Meeting of German Ophthalmic Surgeons will be held in the Meistersingerhalle, Nuremberg, Germany on 17-20 May 2001. Further details: MCN Medizinische Congress-organisation Nuremberg AG, Zerkabelshofstrasse 29, 90478 Nuremberg, Germany (tel: ++49-911-3931621; fax: ++49-911-3931620; email: doerflinger@mcn-nuernberg.de).

European Association for the Study of Diabetic Eye Complications (EASDEC)

The next meeting of the European Association for the Study of Diabetic Eye Complications (EASDEC) will be held in Paris, France, on 19-20 May 2001. Further details: Colloquium, 12 Rue de la Croix Faubin, 75 557 Paris Cedex 11, France (tel: +33-1-44 64 15 15; fax +33-1-44 64 15 10; email: s.mundler@colloquium.fr).

American Institute of Ultrasound in Medicine—Millennium Ultrasound Course Series

A course entitled "Obstetrical and Gynecological Ultrasound" will be held in New York City, NY, on 24-26 August 2001. Further details: Stacey Bessling, Public Relations Coordinator, AIUM, 14750 Sweitzer Lane, Suite 100, Laurel, MD 20707-5906, USA (tel: 301-498-4100; email: sbessling@aium.org).

4th International Conference on the Adjuvant Therapy of Malignant Melanoma

The 4th International Conference on the adjuvant therapy of malignant melanoma will be held at The Royal College of Physicians, London on 15-16 March 2002. Further details: Conference Secretariat, CCI Ltd, 2 Palmerston Court, Palmerston Way, London SW8 4AJ, UK (tel: + 44 (0) 20 7720 0600; fax: + 44 (0) 20 7720 7177; email: melanoma@confcomm.co.uk; website: www.confcomm.co.uk/Melanoma).