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. This study clearly commented that long term complications of β irradiation, such as scleral necrosis, may arise more than 10 years after the irradiation.1 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, haemorrhage, or significant differences in the scleral and corneal thickness between the treated nasal position of the operated eye (mean scleral 0.617 (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. and Cuijnen and Glery.

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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 peripapillary 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 (23%, 18/82 patients).

The authors postulated that this difference is due to the avoidance of traction on the optic nerve head by the posterior hyaloid, thus limiting damage to the peripapillary nerve fibre layer, which they believed would be most severe nasally because of former 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 inferonasally and not inferotemporarily.2 The inferotemporal location of field defects noted in most studies is based on the position 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.3 This inner retinal damage can be caused by desiccation of the retina or by direct mechanical damage by the pressurised air flow.4 However, humidiﬁcation of air did not prevent inner retinal damage in animal models,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.6 In addition, decreasing the infusion air pressure also decreased the risk of inner retinal damage.7 What I think this work by Cullinane and Glery 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, it should 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 Vitrectorial 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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Sclerectomy complications following pars plana vitrectomy

EDITOR,—The work of West and Gregor again points out the importance of sclerectomy complications following pars plana vitrectomy.1 They demonstrate that, even in the hands of a skilled 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 fibrovitreal ingrowth (FVI) as the cause of the haemorrhage.

Interestingly, in this case series of 159 eyes, no occurrences of anterior hyaloid fibrovascular proliferation (AHFV) 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 inadvisedly, suggesting that epithelial tissue grows into the eye through the sclerectomy incision.2 While epithelial tissue, scleral fibroblasts, and ciliary epithelium all contribute, the majority of the fibroproliferative healing of a sclerectomy 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 contracture, with the result being the small scar seen at the internal aspect of a healed sclerectomy.4 Inevitably, because of the proximity of the vitreous base and anterior hyaloid, vitreous strands are adherent to the wound and fibrovascular tissue extends a short way into the vitreous body. This tissue may contain blood vessels, even with normal healing. From this perspective, all sclerectomy 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

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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 scleromities, 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.

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 AHPP, most of the patients had retinal detachments that had required scleral buckling. Since retinal detachment and scleral buckling exacerbate anterior ischaemia, it is likely that AHPP, which is fibrovascular proliferation into the vitreous base from the retina and ciliary body, is induced by an ischaemic drive similar to that of APVR. The two entities exist on a continuum. When there is a surgical injury such as a sclerotomy, with disruption of tissue and inoculation of blood into the surrounding vitreous, excessive proliferation may occur with less induction than that which causes AHPP.

Personally, although I have observed cases of AHPP without having previous vitrectomy, I have never seen a case of post-vitrectomy ultrasonographically or at the time of VCWO. In the original description of AHPP, most of the patients had retinal detachments that had required scleral buckling. Since retinal detachment and scleral buckling exacerbate anterior ischaemia, it is likely that AHPP, which is fibrovascular proliferation into the vitreous base from the retina and ciliary body, is induced by an ischaemic drive similar to that of APVR. The two entities exist on a continuum. When there is a surgical injury such as a sclerotomy, with disruption of tissue and inoculation of blood into the surrounding vitreous, excessive proliferation may occur with less induction than that which causes AHPP.

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 exists and reported the recurrent vitreous haemorrhage. I have observed vitreous haemorrhage in a necropsy eye from what grossly appeared to be a normally healed sclerotomy wound. 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 coexists with time, becoming less vascular in its appearance. So, the frequency of FVI may be even higher than reported.

Lastly, I agree that episceral 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 sclerotomy wounds and may persist even though wound fibroplasia becomes involutional and clinically unimportant. Similar vessels are seen microscopically in the ciliary body. When present, sentinel 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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Conical transplantation: how successful are we?

EDITOR,—The commentary by Waldock and Cook1 on the survival rates of conical 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 Conical 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. 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 analyses. Capabilities beyond most individual centres but readily achievable within the NHS. The two entities exist on a continuum. When there is a surgical injury such as a sclerotomy, with disruption of tissue and inoculation of blood into the surrounding vitreous, excessive proliferation may occur with less induction than that which causes AHPP.

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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, 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 in this particular context 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 obscuration of the pathogenic sequence in advanced diabetic eye disease by the Boston group is reiterated. Stickler’s arthropathology is 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 tumic vascular tela.

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

DAVID McLEOD


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 essentially 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 and its associated neurology and the exercises which play a part in compromising pupil tone.

BOOK REVIEWS

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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 pupill 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 the material which serves to mark 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 “thumbnall” 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 unfolded 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 glaucoma has not been extensively commented. Developing “pupil perimetry” has not been the pharmacological influence of drugs upon pupil function and their role in therapeutics.

The First International Congress on Non-Penetrating Glaucoma Surgery will be held in Poznan, Poland on 5-6 April 2001. Further details: Professor Krystyna Pecold, Katedra I Klinika Okulistyki, ul Dlug a 1/2, 61-849 Poznam, Poland (tel/fax: 004861-8527619) or Professor Ingrid Keissig, Univ-Augenklinik, Schleischtzstrasse 12, D-72076 Tuebingen, Germany (fax: 49-7071-293746; email: ingrid.Keissig@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 9061; 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 Meisteringerhalle, Nurenberg, Germany on 17-20 May 2001. Further details: MCN Medizinische Congress-organisation Nurenberg AG, Zerabelshofstrasse 29, 90478 Nurenberg, Germany (tel: ++49-911-3931621; fax: ++49-911-3931620; email: doerflinger@mcn-nuernberg.de).

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 20770-5906, USA (tel: 301-498-4100; email: sbessling@aium.org).

Optometry Study Tour to Kenya, Tanzania, and Zambia 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, Croxton Mews, 28 Croxton 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, Hospital Ophthalmique Jules Gounin, Avenue de France 15, 1004 Lausanne, Switzerland (tel: 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: BMJ/AM Conference Unit, BMA House, Tavistock Square, London WC1H 9JP, UK (tel: +44 (0) 20 7383 6400; fax: +44 (0) 20 7383 6869; email: quality@bma.org.uk; website: www.quality.bmjgp.com).

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 20770-5906, 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@confcom.co.uk; website: www.confcom.co.uk/Melanoma).