Correspondence to: Jimmy S M Lai

EDITOR,—We read with interest the study that presented a 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 (23%, 18/82 patients).

The authors postulated that this difference is due to the avoidance of traction on the optic nerve head and the posterior hyaloid, thus limiting damage to the peripapillary nerve fibre layer, which they believed would be most severe nasally because of firmer vitreous 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. If the infusion cannula is inferonasal, visual field defects occur inferonasally and not inferotemporally. The inferotemporal location of field defects noted in most studies is based on the 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. This inner retinal damage may be caused by desiccation of the retina or by direct mechanical damage by the pressurised air flow. However, humidification of air does not prevent inner retinal damage in animal models, and the sharp demarcation between damaged and undamaged retina on electron microscopic studies supports the theory of direct mechanical damage to the inner retina. In addition, decreasing the infusion air pressure also decreased the risk of inner retinal damage. 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, one 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 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 vitreectomy 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.

JIMMY S M LAI
Department of Ophthalmology, United Christian Hospital, Kwan Tong, Kowloon, Hong Kong

CLEMENT C Y THAM
Department of Ophthalmology and Visual Sciences, The Chinese University of Hong Kong, Eye Unit, Prince of Wales Hospital, Shatin, New Territories, Hong Kong

DENNIS C LAM
Department of Ophthalmology and Visual Sciences, The Chinese University of Hong Kong, Hong Kong Eye Hospital, 14/F, Argyle Street, Kowloon, Hong Kong

Correspondence to: Jimmy S M Lai
jumla@netvigator.com


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. They demonstrated 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. Difficulty in the relationship 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 episcleral tissue grows into the eye through the sclerectomy incision. While episcleral 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.

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 sclerectomy. 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 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 intrascleral mischief.
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 sclerotomies, 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 AHFP, 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 AHFP, which is fibrovascular proliferation into the vitreous base from the retina and ciliary body, is induced by an ischaemic drive similar to that which induces FVI. 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 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 was present. They recorded 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 sclerotomy wound. Furthermore, FVI can evolve 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 sclerotomy wounds and may persist even though wound fibroplasia becomes involuted 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 out, nor does their absence rule it out.

ALLAN KREIGER
Retina Division, Jules Stein Eye Institute, Department of Ophthalmology, UCLA School of Medicine, Los Angeles, CA 90095-7600, USA

Conical transection: 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 use of long-term follow-up data in the UK. The value of such data is clearly evident from the Australian Cononal Graft Register. 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 analysis—capabilities beyond most individual centres but readily achievable within the NHS. They are easy to follow as shown by the organ transplant community, and to a certain extent by cononal graft surgeons, is through well organised, centralised data collection and analysis, for example.

The good news is that such a system is now in place for all cononal 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 power to answer the sorts of questions posed by Waldock and Cook. Indeed, the audit is already being used for data capture for the Cononal Transplant Follow-up Study II, which aims to resolve the uncertainty surrounding HLA-DR matching, visual outcome, or surgeon experience.

As Professor Waldock observes, 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 activity, but with an independent confidential analysis of clinical outcomes, which they will increasingly be expected to have available.

W J ARMITAGE
J M CREWE
Division of Ophthalmology, University of Bristol, Bristol Eye Hospital, Bristol BS1 2LX, UK

A B TULLO
Manchester Royal Eye Hospital, Oxford Road, Manchester M13 9WH, UK

Correspondence to: W J Armitage
w.j.armitage@bristol.ac.uk


BOOK REVIEWS


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, but in a collection which can be readily identified according to 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 arthrogryphosphatopathy 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 ball of bags 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 microarchitect and thus revealing, for example, remnants of the tumic vascular coat.

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 collaboration 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 contains 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 interpretations and criticism which serves to make the text readable. This is also true for the bibliographical references 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 perimeter” has not been presented.

However, with this one exception, this text represents the definitive work upon the pupil which all ophthalmologists will find valuable, and represents the definitive work upon the pupil which all ophthalmologists will find valuable, and represents the definitive work upon the pupil which all ophthalmologists will find valuable, and represents the definitive work upon the pupil which all ophthalmologists will find valuable.

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 www.secondsight.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 20707-9006, 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, 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, Organiser, Centre d’Pages Oculaires de Lausanne, Hospital Ophthalmique 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: BMABMJ Conference Unit, BMA House, Tavistock Square, London WC1H 9JR, UK (tel: +44 (0) 20 7383 6400; fax: +44 (0) 20 7383 6869; email: quality@bma.org.uk; website: www.quality.bmj.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 Dluga 1/2, 61–849 Poznan, Poland (tel/fax: 004861-8527619) or Professor Ingrid Kreissig, Univ-Augenlinik, Schleischtarasse 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: 020 261 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, Nurenberg, Germany on 17–20 May 2001. Further details: MCN Medizinische Congress-organisation Nurenberg AG, Zerabelshofstrasse 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: m.slundner@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 20707-9006, USA (tel: 301-498-4100; email: sbessling@aium.org).

4th International Conference on the Adjutant Therapy of Malignant Melanoma
The 4th International Conference on the adjutant 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).