Intraocular lens implants and risk of endophthalmitis

EDITOR,—We would like to comment on the paper by Bainbridge et al.1

The aim of their study was to investigate the possible association between the use of three piece foldable silicone polymethyl methacrylate intraocular lenses and increased risk of endophthalmitis and indeed the investigators have met this goal and proved this association in an elegant study.

In addition, the authors have supplied the reader in their article with very important information (Table 1) that was not discussed. All of the seven cases had a medical history of one or more systematic diseases that may affect the immunological conditions of the patients and contribute to the development of postoperative endophthalmitis.2 In fact, one patient with plastic anaemia was excluded from the statistical analysis.

Comparing the patients with endophthalmitis with control subjects in a random fashion, even in small series, may reveal additional risk factors such as medical history. The addition of a controlled group of patients undergoing the same surgery who did not develop endophthalmitis could add a lot to the strength of the study.

We believe that endophthalmitis develops when several risk factors are present. We are obliged to take all these factors into consideration before, during, and after surgical procedure, especially in debilitated and immunosuppressed patients. In this kind of patient prophylactic considerations must be borne in mind, including adequate preparation of the patient and surgical field, antibiotics, experienced surgeon, safer instruments, and IOLs.

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Pupillary abnormality

EDITOR,—In a recent issue of the BJO, we had the opportunity to read the interesting case report on pupillary abnormality, by Mallà.1 The author reported a gross persistent pupillary membrane (PPM) in both eyes of a 3 year old Nepalese female. The patient was asymptomatic and near as well as distant visual acuity were normal. Although the author mentioned that the membrane bulged forward into the anterior chamber when the pupil constricted to light, it was not clear if the patient noticed any decrease in vision with bright sunlight and if the author attempted to record the visual acuity in simulated conditions (by shining the light of an indirect ophthalmoscope into the eye at an angle of 45° or after instillation of 2% pilocarpine eye drops).

We have recently reported a case of persistent pupillary membrane in both eyes of an 8 year old male child.2 The brownish membranes were detected by a school teacher. The child confirmed the presence of poor vision in bright sunlight after a precise questionnaire concerning this symptom. The visual acuity in our case under ordinary room illumination was 20/40 in both eyes. Nevertheless, when measured while the light of the indirect ophthalmoscope was shone into his eyes at an angle of 45°, visual acuity was surprisingly reduced to 20/100 in both eyes. Similarly, Kumar et al.2 also reported two cases (aged 15 and 17 years) of hyperplastic pupillary membrane presenting with marked decrease of visual acuity in bright sunlight. In the latter case, these authors recorded a reduction in visual acuity from 20/40 to 20/200 after instillation of pilocarpine eye drops or projecting the indirect ophthalmoscope light at a 30° angle.

It is widely accepted that asymptomatic cases of PPM usually don’t require excision beyond the sensitive period of amblyopia.3 Nevertheless, some cases presenting with significant visual loss in bright sunlight required surgical4 or Nd:YAG laser intervention. Besides visual acuity concerns, cosmetic and refractive disfigurement caused by PPM may also be considered as a reason for intervention in some patients.

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Reply

EDITOR,—I have just reviewed the patient with bilateral persistent pupillary membrane. Visual acuity both for distance and near remained unchanged (6/60 partly and NS in each eye) with pupils constricted by shining the light of an indirect ophthalmoscope at an angle of 45°. The patient has no visual complaints and is unaware of any decrease in vision in bright light.

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“Cyclodiode”

EDITOR,—I read with considerable interest the paper by Spencer and Vernon1 on the results of a standard protocol for transcleral diode laser cyclophotocoagulation (“cyclodiode”). The particular importance of this study with regard to more widespread use of this therapy lies in the high percentage (64%) of treated eyes with pretreatment Snellen acuity, and while a third of these eyes lost 2 or more lines of Snellen acuity, it appears in these cases, not directly attributable to the cyclodiode treatment, with particular note being made of the low rate of cystoid macular oedema.2

The authors report success rates in achieving IOP control with a standard protocol, but, as in most other published series, record findings after “repeat as necessary” retreatments (in this study up to five in number). While this is of obvious interest to clinicians, it may be of almost equal utility to know the effect of a single treatment. In an earlier paper, also using a standardised treatment protocol for cyclophotocoagulation,3 an attempt was made to elucidate any dose-effect relation from a single cyclodiode treatment session. With a single treatment totalling 90 J through 360°, a mean lowering of IOP of 48% was achieved, but the predictability of outcomes in this series was hampered by the high number of neovascular glaucoma (NVG) cases, which are recognised as having highly variable responses.4 It would seem that Spencer and Vernon are uniquely placed—with their standard protocol and low numbers of NVG cases—to provide data pertaining to any dose-effect relation from a single treatment, information which may be used to enhance the predictability of the procedure for individual patients.

The authors also note that their cohort was largely free of cases having had previous cyclodestructive procedures: that is by definition not true, however, of all the retreatment cases, and the authors appear not only to have been reasonably forthright in their pursuit of an IOP <22 mm Hg, but also to have applied the same laser dose irrespective of the number of retreatments, with their retreatment plan leaving no untreated quadrant. In the series noted above, using a half standardised single treatment (45 J over 180°) for cases judged clinically to be at risk of hypotony (which included cases having had previous cyclodestructive procedures) a mean IOP reduction of 36% was still achieved.5 It would therefore be of great interest to know whether any cases in Spencer and Vernon’s paper were excluded from treatment, despite inadequate postoperative IOP control, because of a concern about possible hypotony; similarly, it would be useful to know whether “all comers” were treated in the study to know whether there were specific exclusions from standardised cyclodiode treatment because of this perceived risk.

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Reply

EDITOR,—We thank Dr Walland for his interest in our paper 1 and for summarising the results of his study 2 which was published following our paper’s submission. It is difficult to quantify the dose-effect from a single treatment in cyclodiode because (a) it would depend on the follow up period as the effect may diminish with time, and (b) one would have to continue all the prelaser antiglaucoma medications (not always desirable) to see the true effect.

However, we can analyse the “single dose effect allowing for a reduction of medications” from our study by examining the results of only those eyes which had one treatment session (32). This subgroup contained seven women and eight men (three referred for glaucoma, five for uncontrolled hypertension and three for other reasons). For this subgroup, decreased IOP of 4.7 mm Hg was obtained. This could be due to transitory systemic factors, or it may be due to the post-laser IOP at a mean of 10.4 months was still significantly lower (26). We cannot estimate the percentage reduction. This was associated with a decrease from a mean of 31.2 mm Hg to a mean of 16.2 mm Hg, with a 45% mean percentage reduction. This was associated with a reduction in numbers of patients taking acetazolamide from 88% to 6% and a mean medication usage from 2.2 to 1.2. None of these results differs significantly from those of the whole cohort.

In our study no eyes were denied treatment or treatment interruption because of a perceived risk of hypotony, and “all comers” were indeed treated by this modality if enhanced filtering surgery was considered contraindicated. It may be of interest to know that 71% of the cases referred were referred into our service from our consultants throughout our region (population approximately six million). We cannot state that all eligible cases were treated by us, but we believe our cohort is likely to be representative of cases referred to other glaucoma specialists with a similar population to that found in the East Midlands of England.

We note that, in Walland’s study, the mean plasma level of IOP at a mean of 10.4 months was 25.8 mm Hg with only 55% <22 mm Hg even when a “full” treatment of 90 J was delivered. Although this may be as a result of the large numbers of patients with neovascular glaucoma in this group, it may also be due to the time and power output settings used (1.5 seconds and 1.5 W). With our settings of 2 seconds and 2 J per shot we were able to control IOP with a 65.7% reduction using a mean of 1.7 treatments in our neovascular subgroup. Reducing the output per shot, as in Walland’s study, may result in a reduction in the intraocular pressures delivered. This could be due to transission attenuation in certain eyes, operator technique variation, probe output differences, and ciliary process uptake/susceptibility factors.

STEPHEN A VERNON
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Is non-arteritic anterior ischaemic optic neuropathy related to homocysteine?

EDITOR,—We read with interest the paper recently published by Kawasaki et al. 3 They suggested that hyperhomocysteinaemia may play a role in the occurrence of non-arteritic anterior ischaemic optic neuropathy (NAION) in non-diabetic patients younger than 50 years, and raised the question of the frequency of the methylene tetrahydrofolate reductase (MTHFR) C677T mutation in this population.

There have been anecdotal reports of thrombotic tendencies in patients with NAION. 4 Although NAION is most probably related to local factors compromising the posterior ciliary artery circulation at the optic nerve head (so called “disc at risk”), it is also possible that some systemic factors such as hyperhomocysteinaemia and the MTHFR C677T mutation may enhance local atherogenesis at the level of the posterior ciliary arteries, thereby precipitating the development of NAION in those at risk for the disease. 5 Kawasaki et al could not demonstrate any change in hyperhomocysteinaemia and NAION. However, the blood sample used to measure the homocysteine was obtained after the clinical event. Since homocysteinaemia fluctuates, it is possible that the general population have underestimated the frequency of hyperhomocysteinaemia. We recently investigated prospectively the presence of hyperhomocysteinaemia and the MTHFR C677T mutation in patients with acute NAION.

Blood samples from 14 newly diagnosed patients with acute NAION presenting to our centre over a 1 year period (May 1998 to May 1999) were evaluated for serum creatinine, serum and red blood cell folate, B12, and total plasma homocysteine levels, as well as the C677T polymorphism in the MTHFR gene. There were 10 men and four women (13 white and one Asian), ranging in age from 28 to 68 years (mean aged 42.8 years). All patients had a disc at risk in the fellow eye. Five patients subsequently suffered NAION in their second eye. Four patients (28.5%) were heterogeneous and one was homozygous for the C677T mutation in the MTHFR gene, which does not differ from the frequency reported in the general population. 6 Only one of these five mutation positive patients had bilateral NAION. The homocysteine level was within the normal range in all 14 patients, as were the creatinine, folate, and B12 levels. Homocysteine levels were not higher in the mutation positive than in the mutation negative patients.

Mutation positive and mutation negative patients did not differ with respect to clinical data concerning risk factors for NAION or coexisting vascular disease. Although this is a small study, these results suggest that hyperhomocysteinaemia and the C677T MTHFR polymorphism do not have a role in the occurrence of NAION. Our results are similar to those of Kawasaki et al and the frequency of the MTHFR mutation is not higher than in the general population. 7 As recently re-emphasised, 8 laboratory testing for hypercoagulable states in a patient with NAION without past medical history or family history of a thrombotic event would be unwarranted.

Immediately thereafter, his vision was blurred bilaterally and he noted a red central scotoma in each eye. These symptoms resolved spontaneously within 2 days. An eye examination performed 3 days later disclosed that his vision and visual fields were normal, but there were retinal pigment epithelial disturbances which appeared in fluorescein angiography as recurrent thrombotic events or a family history of thrombosis, or if there is no disc at risk in the fellow eye in a younger patient without vascular risk factors, an investigation for hereditary and acquired thrombophilic markers may be justifiable.

This study was supported in part by a departmental grant (department of ophthalmology) from Research to Prevent Blindness, Inc, New York, New York, by core grant P30-EY06360 (department of ophthalmology) from the National Institute of Health, Bethesda, Maryland, and a grant recipient of Research to Prevent Blindness Lew R Wasserman Merit Awards.

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Reply

EDITOR,—We thank Biousse and colleagues for their comments on our article and their corroborative study. We agree that an extensive hypercoagulable evaluation is not warranted in patients with NAION who have typical risk factors, including older age. The yield from such an evaluation in young patients with NAION, especially those without known risk factors or those who suffer recurrent events, still needs further elucidation.

AKI KAWASAKI
VALERIE PURVIN
RICHARD BURGETT


a window defect type hyperfluorescence in both eyes close to the fovea. The macular burns persisted throughout an 8 month follow up period. The literature describes two cases of unilateral macular damage from laser pointers (class 2 diode, 670 nm, maximum output 1 mW and class 3a diode and 670 nm, maximum output 5 mW), and two other cases of bilateral decreased vision due to large retinal photocoagulation scars from class 3a laser pointer.

In his review of the safety of laser pointers, Marshall comprehensively described the classification of the lasers according to hazard. However, no small part of the message of his paper was the letter to the Lancet, which he mistakably described to the reader into believing that laser pointers are harmless. After witnessing the persistent injury to our own patient and reading the reports of four others who were likewise hurt by this device, we are appalled. The laser pointer is not an innocent toy. It damages the eye and should not be made freely available to youngsters whatever its strength, while the label of the laser pointer alone will not to shine the laser pointer light into an eye.

Media “hype” underpinning reports in the popular press and the pernicious avarice of individuals lurking in wait for opportunities to claim compensation for spurious injury seem to have galvanised estimable individuals to rush to the defence of this instrument. We believe that laser pointers are harmless. We recommend that use of the potential hazards associated with improper use. We recommend that use of laser pointers in public should be controlled and closing devices should be kept away from children.

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The fourth edition of this standard text lives up to its enormous reputation. Jack Kanski sets out “to provide the trainee with a systematic and easily assimilated introduction to ophthalmology and a reference and update for the more experienced practitioner”. Undoubtedly these clear and circumscribed aims are well met in this beautifully and even more lavishly illustrated text. In addition to covering all those aspects of ophthalmology—classes, disorders, new, chapter on ocular trauma has been added in addition to descriptions of new surgical techniques and some pruning of outdated material.

This is and has been an extremely successful primer text for the trainee ophthalmologists and one might ask why this book rather than the many other texts available. Perhaps the answer lies in part in the approach taken with this text which is that of the “patient oriented”: one can almost envisage the author examining the patient presenting to the ophthalmic clinic by starting systematically at the front of the eye and working his way posteriorly towards the orbit and/or cortex until he finds the source of the patient’s complaints. There is less emphasis on why the patient might have his complaints than finding out what exactly the problem is and what the practical steps are to do about it. As such it works very well because it is concise but sufficiently detailed and above all immediately accessible. In fact there is a remarkable amount of detail (see, for instance, the section on corneal dystrophies) while one could debate occasional diagnoses attached to some of the fundus photographs (see, for instance, serpiginous choroiditis). There are also some very helpful line diagrams such as those included in the retina and orbit chapters.

The section on neuro-ophthalmology contains several excellent illustrative radiological scans. Overall this is an excellent starting text. If there is any criticism that can be levied at this classic text, it is that it leaves this reader thirsting for further information. If a similar effect is induced in the trainee ophthalmologist it will have achieved its aim. I can therefore recommend this book as essential reading.


This is an important and thought provoking book which has inevitably not only within the medical profession but also by interested parties such as health economists and government officials whose responsibility it is to set budgets for healthcare programmes. I think it will also be of great interest to the lay public. The practice of medicine is as susceptible to the whims of fashion and pervasive ideology as any other human activity. It is therefore interesting to investigate how these fashions are set. James Le Fanu has a background in medical and scientific journalism, having spent time on the staff of the Daily Telegraph, one of the UK’s broadsheet newspapers. His thesis is that despite the significant advances in combating disease which reached their peak in the post-war years, the promise of modern medicine as we are at the end of the century has failed to materialise. In fact, Le Fanu contends that much of the advances in the first half of the 20th century were accidental or at best serendipitous, citing as examples the discovery of antibiotics, which was never predicted, or the use of chloroquine for rheumatoid arthritis. He cites many clinical observations of patients treated for malaria. Even the success of aggressive chemotherapy for childhood cancer was the result of a determined but empirical approach of testing systematically multiple drugs in combination. The same approach has now been shown to be successful in the treatment of AIDS when three or more drugs are given in a way that one. This “success” is not based on scientific knowledge, despite the vast amount that has been discovered about the AIDS virus, but simply on a “suck it and see” approach.

In contrast, the great promise of the new genetics or of the social theory of disease has not held up according to the author. The amazing strides in molecular biology led to the rapid acceptance of the possibilities of gene therapy but these have emphatically failed to deliver, despite the intellectual satisfaction that these smart ideas generate. Similarly, in the wake of studies showing a clear epidemiological correlation between smoking and lung cancer the social theory has sought to link almost every disease for which there is not an obvious infectious cause to some lifestyle or nutritional source mostly blamed on Western society.

Le Fanu firmly believes conceptual errors at the feet of a few individuals who inveigled themselves into influential positions—for instance, in the American Medical Association, and with the support of the major drug companies have utterly changed our lifestyles to the point where the vast majority of healthy individuals are worried more about their health than ever before while being encouraged to ingest drugs such as cholesterol lowering agents for which there is little evidence that they will actually do for the individual what the statistics tell us. Let alone prevent the individual patient from dying of a heart attack. Le Fanu suggests that it would be possible to rectify this situation overnight by closing down all university departments of epidemiology. Ophthalmology has not been immune to these problems (see the revised recommendations concerning laser treatment for diabetic patients with clinically significant macular oedema). In addition there may be a 20/20 vision, Arch Ophthalmol 1999; 117:675.

This book is not a sustained attack on modern medical practice nor is it written purely to debunk and expose the weaknesses of the many of these in a way that alters people’s lifestyles. In particular, the book has much to say about the dangerous part played by the major pharmaceutical companies in medicine. Many who read this book will be able to relax about their imputed health problems, to feel confident about their ability to ward off many of the supposed hidden dangers which face them out there, and to take much of what they hear from the medical pundits with a pinch of salt. The author offers hope for the future and, in particular, calls for a return of the experienced physician who exercises good clinical judgment, with a dash of common sense.
NOTICES

Community participation in eye health and trachoma and the SAFE strategy

The latest issues of Community Eye Health (nos 31 and 32) discuss community participation in eye health (issue 31) and trachoma and the SAFE strategy (issue 32). 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) 171 608 6909/6910/6923; fax: (+44) 171 280 9309; email: eyeresource@ucl.ac.uk) Annual subscription £25. Free to workers in developing countries.

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, Secretary of the Society for Ocular Circulation and Neovascularisation will be held in Jerusalem on 21–26 May 2000. Further details: Secretariat, c/o Unitours Israel Ltd, PO Box 3190, 61051 Tel Aviv, Israel (tel: (+972-3) 529099; fax: (+972-3) 523909; email: meetings@unitours.co.il).

The VIth Mediterranean Ophthalmological Society

The combined meeting of the VIth Mediterranean Ophthalmological Society and the VIth Michaelson Symposium on Ocular Circulation and Neovascularisation will be held in Jerusalem on 21–26 May 2000. Further details: Secretariat, c/o Unitours Israel Ltd, PO Box 3190, 61051 Tel Aviv, Israel (tel: (+972-3) 529099; fax: (+972-3) 523909; email: meetings@unitours.co.il).

The VIth Michaelson medal and award will be delivered on 24 May 2000 in Jerusalem. The medal and award (£15 000 monetary prize) are sponsored by the Israel Academy of Sciences and Humanities and by the Hadassah Hebrew University Hospital and Medical School of Jerusalem, Israel. Nominations are sought from the ophthalmic community at large. Suggestions for choice and CV highlights should be sent to Professor David BenEzra, Hadassah Hebrew University Hospital and Medical School of Jerusalem on 21–26 May 2000 at Banská Bystrica, Slovakia. Further details: Eye Clinic FD Roosevelt Hospital, Arm Gen L Svobodu Sq, 1, 975 17 Banská Bystrica, Slovakia (tel: 00421 88 413 4671; fax: 00421 88 413 2047).

6th Congress of the European Glaucoma Society

The 6th Congress of the European Glaucoma Society, millennium meeting 2000, will take place at the Eye Clinic FD Roosevelt Hotel, London, on 26–29 June 2000. Further details: Eurocongres Conference Management BV, Jan van Goyenkade 11, 1075 HP Amsterdam, Netherlands (tel: +31 20 679 34 11; fax: +31 20 673 73 06; email: egs@eurocongres.com).

British Ophthalmic Photographic Association

Forthcoming meetings of the British Ophthalmic Photographic Association will be held at Southampton Eye Unit. Further details: Tim Mole (tel: 01703 798747). On 17–18 November BOPA annual conference at York. Further details: Mike Geall (tel: 0113 3923506).

Joachim Kuhlmann Fellowship for Ophthalmologists 2000

The Joachim Kuhlmann AIDS Foundation, Essen, Germany, is sponsoring two fellowships per year for ophthalmologists at a well known institute, who want to train in CMV retinitis and other HIV related ophthalmological diseases. The fellowships are valued at US$5000 each. Deadline for application is 31 July. Detailed applications, including CV and publication list, should be sent to the Joachim Kuhlmann AIDS Foundation, Bismarckstrasse 55, 45128 Essen, Germany (tel: 0201 87910-87; fax: 0201 87910-99; email: jkf-stiftung@t-online.de).

DR-2000, International Forum on Diabetic Retinopathy

The International Forum on Diabetic Retinopathy will take place on 7–9 September 2000 at the Palazzo Reale, Naples, Italy. Further details: Francesco Bandello, Congress Secretariat, MGR Congressi, Via Servio Tullo, 4, 20123 Milano, Italy (tel: 39 02 4306711; fax: 39 02 48008471; email: dr2000@mgr.it).

VIII Tuebingen Angiography course

The VIII Tuebingen Angiography course with wet lab will take place on 9 September 2000 in the auditorium, University Eye Clinic, Schleichstrasse 12, 72076 Tuebingen, Germany. Further details: WT-Wissenstransfer, Universitäts-Augenklinik (tel: +49 7071 29 76439; fax: +49 7071 29 5051; email: witt@uni-tuebingen.de).