Case of acute zonal occult outer retinopathy with altitudinal hemianopsia

Since the clinical entity of acute zonal occult outer retinopathy (AZOOR) was initially proposed, it has been noted that the visual loss may be misattributed to lesions in the optic nerve or central nervous system. Even with a likely visual field defect for those diseases—an afferent pupillary defect and reduced subjective central flicker fusion threshold—clinicians should be always aware of the possibility of AZOOR.

Case report

A 32 year old woman noticed a large scotoma in her right eye. She was examined by an ophthalmologist who found her corrected visual acuity to be 0.4 in the right eye and 1.0 in the left eye. She also had an afferent pupillary defect in the right eye. Goldmann perimetry showed a superior altitudinal hemianopic defect in the right eye (fig 1). Subjective central flicker fusion threshold was reduced in the right eye (18 Hz) and normal in the left eye (35 Hz). Ophthalmoscopy, angiography, blood screening, and computed tomography were normal. Suspecting ischaemic optic neuropathy (ION), a 5 day course of intravenous sucinyl hydrocortisone, 100 mg/day, was used but this treatment was not effective. The patient was then referred to us for further examination. Full field rod and cone electroretinograms (ERGs) were reduced in her right eye (about 50% of those in the left eye). Multifocal ERGs (mfERGs) recorded with the VERIS Science 4.0 system (Electro-Diagnostic Imaging, San Mateo, CA, USA) revealed reduced responses in areas corresponding to the visual field defect (fig 2). These findings led us to presume the diagnosis to be AZOOR. While we have followed her for approximately a year, no retinal finding has been observed and the visual defect has not changed.

Comment

Although altitudinal hemianopsia is found in many optic nerve disorders—for example, as meningioma, optic neuritis, ION, sinususes or intracranial artery disorders, and congenital abnormalities of the optic nerve head, this type of field defect had been rarely reported in AZOOR. The visual field abnormality in AZOOR varies from case to case, but central or paracentral scotoma is most common. When the clinical entity of AZOOR was initially proposed, the visual loss was attributed to retrobulbar neuritis, a pituitary adenoma, or other intracranial lesions during the early stages of this disease. Normal fundus and fluorescein angiographic findings with dense scotoma led ophthalmologists to suspect optic nerve or intracranial diseases. In addition, the afferent pupillary defect and disc swelling in AZOOR were very misleading. Our patient had a superior altitudinal hemianopic defect, an afferent pupillary defect, the reduced subjective central flicker fusion threshold and no disc swelling, and thus had to be differentiated from ION or posterior ION (PION) without optic disc involvement. PION is an unusual subset of ION and frequently related to arteritis. ERG observations may be critical for the differential diagnosis of cases with visual field defect without observable retinal lesions that are usually attributed to optic nerve or intracranial diseases. While the full field ERG may be sufficient to make the diagnosis, in many of the cases, focal ERG or mfERG can show the correspondence of the retinal dysfunction to the visual field defect which strongly reinforce the diagnosis of AZOOR.

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Accepted for publication 9 April 2002

Grant support: Grant No 13877293 from the Ministry of Education, Culture, Sports, Science and Technology, Japan.

References


Congenital optic nerve head pit associated with reduced retinal nerve fibre thickness at the papillomacular bundle

Congenital pits of the optic nerve head result from an imperfect closure of the superior edge of the embryonic fissure. An unequal growth on both sides causes a delayed closure of the fissure at approximately 5 weeks of gestation. Optic pits appear as crater-like
Histological sections of optic pits define defects in the lamina cribrosa associated with rudimentary retinal tissue, resembling pigmented tissue and aberrant nerve fibres. These anomalous papillomacular nerve fibre bundles may be less resistant, predisposing this sector to spontaneous schisis-like retinal detachments during later life.!

We present a young patient with a unilateral optic pit and a clinically significant temporal nerve fibre loss. In vivo measurements of optical coherence tomography (OCT) determined a mean thickness of RNFL at the side of the pit and the corresponding papillomacular bundle.

**Case report**
A 27-year-old woman presented with a 9-month history of blurred vision; her best corrected visual acuity was 20/20 right eye and 20/25 left eye. On Goldmann perimetry in both eyes, there were no visual field defects, acute or paracentral scotomas. On slit lamp examination the anterior segment appeared normal in both eyes. Fundus biomicroscopy of the left eye revealed a large optic nerve head with a grey oval pit at the temporal margin and a brownish rim at the temporal side. The papillomacular bundle appeared to be darker, extending from the edge of the optic nerve to the macula, compared to the superior and inferior quadrant corresponding with severe RNFL loss according to the semiquantitative assessment of Niessen et al (Fig 1). Fundus examination of the right eye was unremarkable.

Linear OCT disclosed a significantly thickened RNFL in the superior quadrant (Fig 2A) and thinned RNFL at the temporal quadrant of the optic nerve in the left eye. There were no signs of a schisis-like retinal detachment (Fig 2B). Circular OCT demonstrated a significantly reduced thickness of the RNFL in all quadrants but predominantly in the temporal (90 μm) quadrant (Fig 2C).

**Comment**
The oval depression of the optic nerve head in optic pits may relate to an enlarged optic nerve head, an incomplete closure of the embryonic fissure and a reduced RNFL. The size of the optic disc was significantly larger when compared to the mean size (1.76 mm) in normal eyes, reducing the mean RNFL density. In addition, an incomplete closure of the embryonic fissure seems to prevent a proper fusion of the temporal RNFL. OCT confirmed both a reduced mean RNFL and significant loss especially in the papillomacular bundle. Whereas normal subjects have a mean RNFL of 153 μm and 126 μm in the temporal quadrant, our patient with an optic pit had a mean RNFL of 115 μm and 90 μm in the temporal quadrant. Glaucomatous eyes with a mean RNFL below 103 μm frequently develop visual field defects, whereas in our patient with a mean RNFL of 115 μm none became apparent.

OCT precisely measured the retinal thickness with micrometer scale and provided additional evidence for pronounced reduced thickness of the RNFL in the temporal quadrant of the optic pit. Fundus photography confirmed the enlarged optic disc and temporal RNFL damage. The imperfect closure and lack of papillomacular nerve fibre bundles represent a “locus minoris resistance” in optic pits, the development of a spontaneous schisis-like detachment during ageing.

### References

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Proprietary interest: none.

Financial support: none.

Accepted for publication 20 February 2003

www.bjophthalmol.com
Ocular ischaemic syndrome in thyroid eye disease, confirmed using magnetic resonance angiography

Ocular ischaemic syndrome (OIS) is most commonly caused by severe ipsilateral carotid artery stenosis. Occasionally it is caused by ophthalmic artery stenosis. Features commonly observed are iris neovascularisation, angle neovascularisation, rubecotic glaucoma, and iritis. In the posterior segment common signs are narrowing of the retinal arterioles, mid-peripheral retinal haemorrhages, optic disc pallor or neovascularisation and, rarely, retinal neovascularisation. Fluorescein angiography characteristically demonstrates delayed filling of the retinal circulation and occasionally patchy filling of the choroidal circulation is also observed. To the best of our knowledge OIS has not previously been described in thyroid eye disease. We report a case of OIS in thyroid eye disease confirmed by magnetic resonance angiography (MRA) and treated by orbital decompression.

Case report

A 48 year old woman with known thyroid eye disease presented with a 4 week history of pain, redness, and reduced visual acuity in her right eye. Eight years previously she had undergone bilateral three wall orbital decompression for severe corneal exposure. On examination visual acuity was counting fingers in the right eye and 6/9 in the left. There was bilateral lid retraction and mild generalised restriction of eye movements. There was bilateral proptosis measuring 24 mm in the right eye and 23 mm in the left (Keeler exophthalmometer). A right relative afferent pupillary defect was present. Intraocular pressures were 50 mm Hg in the right eye and 20 mm Hg in the left. There was right corneal oedema, rubecotic iridis (Fig 1), and moderate anterior chamber activity. Gonioscopy showed an open, grade 2 angle (Shaffer’s classification) with rubecotic vessels present in the angle. Fundal examination was limited by the corneal oedema but no specific abnormality was identified. Examination of the left eye was normal.

Fluorescein angiography showed delayed filling of the retinal vasculature in the right eye relative to the left. Computed tomography scans of the orbits showed previous bilateral three wall orbital decompression and diffuse enlargement of extraocular muscles. Carotid duplex ultrasound examination was normal. An MRA of the orbits demonstrated that blood flow in the right ophthalmic artery was reduced. Blood flow in the left ophthalmic artery was normal (Fig 2).

The patient was admitted and treated with intravenous mannitol and acetazolamide and topical apraclonidine 0.5% and betaxolol 0.5% but intraocular pressure remained elevated at 29 mm Hg. A further right orbital decompression was performed (where the lateral orbital wall was removed as far posteriorly as the anterior wall of the middle cranial fossa and superiorly to the floor of the anterior cranial fossa). Postoperatively the right prop-tosis measured 21 mm, the relative afferent pupillary defect resolved and the intraocular pressure was controlled (<20 mm Hg) with oral acetazolamide and topical apraclonidine 0.5% and betaxolol 0.5%. The corneal oedema resolved and the visual acuity gradually improved to 6/9. At the 3 month follow up postoperatively the rubecotic iris vessels had regressed. An MRA performed 4 months postoperatively demonstrated normal blood flow in both ophthalmic arteries (Fig 3).

Comment

Imaging methods available for evaluating the ophthalmic artery include duplex ultrasonography and cerebral angiography. Duplex ultrasonography is a non-invasive technique that gives quantitative information about flow; however, it requires an experienced operator and it is not always possible to positively identify the ophthalmic artery. Cerebral angiography is an invasive technique with the inherent risk of embolisation and stroke. MRA is a relatively new technique and has not previously been used to investigate disturbances of blood flow in the ophthalmic artery. It is non-invasive and does not require the level of technical experience required for Doppler studies. MRA detects blood flow at a defined velocity. In this case 25 mm/s was chosen as it has been shown in many studies using Doppler ultrasound to be the mean ophthalmic artery blood flow. The absence of signal from the right ophthalmic artery in the preoperative MRA demonstrates that at no stage during the cardiac cycle was blood flowing at this velocity in the artery. The images shown in Figures 2 and 3 are composites of all slices taken through the orbits. Hence it is not possible that one of the ophthalmic arteries could have been missed as a result of the orientation of any one particular slice.

Various abnormalities of the orbital circulation have been reported in thyroid ophthalmopathy. Blood flow in the superior ophthalmic vein has been shown to be reduced, or even reversed in some patients. Increased central retinal artery, ophthalmic artery, and retinal blood flow have also been demonstrated. Ischaemia of the optic nerve head has been postulated to have a role in the development of optic neuropathy in some patients with thyroid ophthalmopathy. However, to the best of our knowledge ophthalmic artery obstruction as a result of thyroid eye disease has not previously been described.

In summary, this case demonstrates for the first time, the ocular ischaemic syndrome as a result of ophthalmic artery obstruction in thyroid eye disease. Furthermore, it demonstrates the usefulness of MR imaging in evaluation of the ophthalmic artery.

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Accepted for publication 21 February 2003

References

silicone oil in diabetic vitrectomy

Castellarin and colleagues recount their recent experience of infusing silicone oil in a small series of patients with advanced diabetic eye disease, either during primary vitrectomy (12 eyes) or after earlier surgery had failed (11 eyes). They compare their recent experience of infusing silicone oil in anterior segment complications that have not been considered previously.

The anterior diffusion of angiogenic substances released as a result of the extensive scatter laser that was often needed to prevent highly vascularised membranes from repopulating behind the silicone oil. It was hoped that the so-called “compartamentalisation” of the eye by silicone oil by which the retro-silicone oil neovascularisation was attributed) might in turn result in prevention or reversal of ruberosis iridis through its putative barrier effect against anterior diffusion of angiogenic substances derived from the ischaemic retina. Paradoxically, eyes with successful retinal reattachment (albeit with unabated ischaemia) often underwent rapid development or progression of iris neovascularisation, while those with failed surgery from post-operative rhegmatogenous recurrence of retinal detachment (and therefore eyes with an exaggerated angiogenic drive) had evidence of protection from neovascularisation, at least in the short term. Perhaps naively it was postulated that rhegmatogenous confinement of the ret detachment by intravitreal silicone oil (and the consequent 100% oil filling of the shrinking vitreous cavity) might allow an effective obstruction to anterior molecular diffusion to be established in these failed cases. Others had planned from the outset to employ silicone oil in their surgical protocol, not least for those diabetic eyes wherein earlier vitrectomy had been unsuccessful as a consequence of retinal reattachment or recurrent vitreous cavity haemorrhages. However, whether used during primary diabetic vitrectomy or secondarily, whether unpremeditated or planned, and whether infused by direct fluid-oil exchange or sequential fluid-air exchange, dealing with large or multiple posteriorly located breaks (whether pre-existing or iatrogenic) was problematic, and direct fluid-silicone oil exchange (by virtue of the optical advantages of oil over air in the phakic eye) provided a surgical escape route, obviating the need for posterior vitrectomy. Furthermore, the clarity of the media immediately postoperatively facilitated the slit lamp delivery of focal laser in order to seal retinal breaks that had been closed by the internal tamponade and, in addition, the application of scatter laser to retetched, untreated, ischaemic retina that had undergone deturgescence, in part through the “waterproofing” effect of silicone oil. All being well, the silicone oil could then be removed shortly thereafter, and some eyes that would undoubtedly have been lost were saved by the intervention of silicone oil in this way. Often, however, there were significant problems, not least the rapid development of reparative epiretinal fibrosis whereby the retina reattached under tangential traction and/or from reopening of retinal breaks. Sometimes huge areas of retinal disintegration eventually developed. Then fibroglial proliferation appeared (both clinically and pathologically) to be particularly induced by clotted blood trapped between the silicone oil and the retinal surface or, ironically, by fibrin released as a result of the extensive scatter laser that was often needed to prevent highly vascularised membranes from repopulating behind the silicone oil.

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The rate of rubeciosis iridis regression was greater than we reported initially (that is, 3/7 (43%)) because of continued regression of rubeciosis during the longer period of follow up (Table 4). Of the three eyes with NVG, one eye underwent Baerveldt valve placement with normalisation of the postoperative intraocular pressure. Five months after surgery, however, the eye developed hypotony. Subsequently, the valve was removed, and the eye underwent additional SOI. As noted above, however, the eye became phthisical. As reported initially, the NVG regressed after surgery in the second eye, and the third eye had NVG regression but no light perception postoperatively because of ischaemia. As noted in our initial report, only one eye developed de novo rubeciosis iridis. Among the 17 eyes with at least 6 months’ follow up, six (35%) had intraocular pressure <5 mm Hg. In our initial report, five (23%) of 23 eyes had intraocular pressure <5 mm Hg. Three of these six eyes had persistent retinal detachment, which we presume to be the cause of the hypotony. Among eyes with at least 6 months’ follow up, the remaining 11 (65%) had intraocular pressure ranging from 6–48 mm Hg, with four patients taking antiglaucoma medications.

Intraoperative complications were not different among eyes with at least 6 months’ follow up versus the entire cohort of 23 eyes (Table 5). Postoperative complications differed in that there was an increased prevalence of cataract, hypotony, and silicone oil in the anterior chamber over time, which is not surprising.

McLeod suggests that in addition to using data from eyes with at least 6 months’ follow up, one should use the status of the fellow eye to judge surgical success. At the time of surgery, all fellow eyes had proliferative diabetic retinopathy (in addition to other vision threatening conditions) and had undergone full panretinal photocoagulation (Table 6). Among patients with traction retinal detachment in the fellow eye, two underwent fellow eye surgery and one patient refused surgery (Table 6). Six (29%) of 21 fellow eyes were pseudophakic. Among the six fellow eyes with visual acuity <20/400, two had no light perception. Among the 21 patients we reported, the severity of disease in the fellow eye was such that two patients underwent PPV+SOI bilaterally, and results from both pairs of eyes were

### Table 1 Surgical indications

<table>
<thead>
<tr>
<th>Condition</th>
<th>Eyes with at least 6 months’ follow up</th>
<th>All operated eyes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Retinal detachment</td>
<td>$\frac{5}{9}$ (56%)</td>
<td>$\frac{8}{11}$ (73%)</td>
</tr>
<tr>
<td>Neovascular glaucoma</td>
<td>$\frac{5}{9}$ (56%)</td>
<td>$\frac{8}{11}$ (73%)</td>
</tr>
</tbody>
</table>

**Columns:**
- COHORT
- Retinal detachment rate

**Table 2 Anatomical results of pars plana vitrectomy (PPV) membrane peeling, and silicone oil infusion (SOI)**

<table>
<thead>
<tr>
<th>Cohort</th>
<th>Retinal detachment rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall</td>
<td>$\frac{12}{17}$ (71%)</td>
</tr>
<tr>
<td>PPV+SOI as primary intervention</td>
<td>$\frac{7}{8}$ (88%)</td>
</tr>
<tr>
<td>PPV+SOI for failed previous vitreoretinal intervention</td>
<td>$\frac{5}{9}$ (56%)</td>
</tr>
<tr>
<td>Overall</td>
<td>$\frac{17}{23}$ (74%)</td>
</tr>
<tr>
<td>PPV+SOI as primary intervention</td>
<td>$\frac{9}{12}$ (75%)</td>
</tr>
<tr>
<td>PPV+SOI for failed previous intervention</td>
<td>$\frac{8}{11}$ (73%)</td>
</tr>
</tbody>
</table>
that silicone oil was needed in all these cases described. Thus, the majority of our patients were visually disabled because of bilateral, severe eye disease.

McLeod points out that only seven (30%) of 23 eyes in our series had retinal breaks (either pre-existing or intraoperative), and he asks what the rationale for silicone oil use (either pre-existing or intraoperative), and he suggests that wide angle viewing systems, heavy liquids, endolaser, and long acting gases enable one to manage posterior retinal breaks and retinectomies. McLeod asks, “What is the appropriate use of silicone oil in the diabetic eye in the modern era?” He suggests that in cases where breaks can be managed with gas tamponade and silicone oil can be reversed with retinal reattachment and laser photocoagulation, the use of silicone might be “gratuitous.” McLeod suggests that appropriate uses of silicone might include patients with posturing difficulties or patients in whom there is a need for early visual rehabilitation.

We recognise that the conclusions of our study are limited because it is a non-randomised retrospective study without a control group. Thus, we cannot identify the “virtuous” indications for the use of silicone oil in the setting of severe proliferative diabetic retinopathy based on these data. Without the use of silicone oil, for example, rubeosis iridis might have regressed, and the retina might have remained attached in eyes exhibiting the fibrinoid syndrome. None the less, the data from our study are consistent with the notion that silicone oil is an acceptable and useful tool in the management of eyes with severe complications of proliferative diabetic retinopathy. Our experience suggests, but does not prove, that silicone oil tamponade improves the prognosis in some otherwise unsalvageable cases. Among 11 (48%) of 23 eyes in this series, silicone oil was used initially at the time of repeat vitreous surgery. Short term retinal reattachment was achieved in eight (73%) of these eyes. Among these 11 eyes, nine had follow up of at least 6 months, and retinal reattachment was maintained in five (56%) of these nine with a single operation. Overall, 10 eyes failed initial PPV with or without SOI; underwent repeat PPV and SOI, and had at least 6 months’ follow up. Retinal reattachment without phthisis was achieved in seven (70%) of these eyes. Since the initial vitrectomies employed modern surgical techniques, these results indicate that even in the modern surgical era, use of silicone oil can improve anatomical (and functional) outcome in selected cases.

References

Primary scleral buckle placement during repair of posterior segment open globe injuries

We read with interest the recent article by Arroyo and associates. They are to be commended on a very interesting study to compare the visual and anatomical outcomes of patients undergoing primary and secondary scleral buckle placement during posterior segment open globe repair with matched control patients who did not undergo primary scleral buckle placement.

Prophylactic scleral buckle of posterior segment open globe injuries has been a controversial topic in ophthalmology. The value of scleral buckling to support peripheral and especially inferior breaks is rarely disputed. However, the utility of using an encircling buckle in the absence of retinal breaks remains controversial.

The benefits of primary scleral buckle placement are that it is technically easy and there is no scarring between the wound and overlying capsule and conjunctiva. However, there are some important considerations against primary scleral buckle such as the perforating injury subsequent rhegmatogenous retinal detachment (RD) is often not directly related to the site of the posterior exit wound but develops secondary to a new retinal break in the vitreous base region within 2 clock hours of the scleral wound. In addition, it is usually difficult to place a buckle over the exit wound and involves potential high morbidity (especially in the hands of an inexperienced doctor who usually receives the patient in the emergency room (at least in Venezuela)).

To counter subsequent traction at the vitreous base, a vitreotomy may be just as effective as a prophylactic scleral buckle, avoiding the associated morbidity. If retinal incarceration occurs through the wound, secondary reconstruction must always be performed anyway, typically involving a scleral buckle and vitrectomy 10–14 days after the injury (when inflammation is under control, and the intraocular anatomical status has been assessed adequately).

We believe that the results of the study by Arroyo and associates contribute to understanding the role of prophylactic primary scleral buckle in the treatment of posterior segment open globe injuries. Their impressive results suggest that the benefit of placing a prophylactic primary scleral buckle may outweigh the risks involved. A multicentre randomised clinical trial is desirable to confirm their results.

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Accepted for publication 26 March 2003

The authors have no proprietary or financial interest in any products or techniques described in this article.

References

Author’s reply
We read with interest the comments made by Fernandez and colleagues regarding our article.1 We certainly agree that new breaks may develop in the vitreous base region within 2 clock hours of the scleral wound. Because of this, we advocate the use of encircling scleral buckles (3.5–5 mm wide) as opposed to segmental scleral buckles in patients undergoing primary open globe injury repair.

We agree that placing an encircling scleral buckle to support the posterior edge of the vitreous base does require more skill than simply closing an open globe wound. However, we have found that with adequate training, encircling scleral buckles can usually be placed within 15–30 minutes.

The timing of vitrectomy in cases of ocular trauma is controversial. We also try to wait at least 1–2 weeks before performing a vitrectomy, if necessary, in order to minimise the risks of very early (potential bleeding, inflammation, and poor visualisation) and very late (cellular proliferation) complications.

We agree that a prospective randomised clinical trial is needed to better delineate the role of primary encircling scleral buckle placement at the time of open globe injury repair.

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Correspondence to: Massachusetts Eye and Ear Infirmary, 243 Charles Street Boston, MA 02114, USA; jarroyo@massoed.org

Accepted for publication 28 May 2003

Reference
UK Transplant’s website (www.uktransplant.org.uk).

Elimination of avoidable blindness

The 56th World Health Assembly (WHA) considered the report on the elimination of avoidable blindness (doc A56/26) and urged Member States to: (1) Commit themselves to supporting the Global Initiative for the Elimination of Avoidable Blindness by setting up a national Vision 2020 plan by 2005; (2) Establish a national coordinating committee for Vision 2020, or a national blindness prevention committee to help implement the plan; (3) Implement the plan by 2007; (4) Include effective monitoring and evaluation of the plan with the aim of showing a reduction in the magnitude of avoidable blindness by 2010; (5) To support the mobilisation of resources for eliminating avoidable blindness as well as aid in the coordination and support of national capability.

Ophthalmic Anesthesia Society (OAS)—17th Scientific Meeting

The 17th Scientific Meeting of the Ophthalmic Anesthesia Society (OAS) will be held 3-5 October 2003 at the Westin Michigan Avenue Chicago, Chicago, USA. Programme co-chairs: Marc Allen Feldman MD MHS and Steven T Charles MD. The CME joint sponsor is the Cleveland Clinic Foundation; CME hours are pending. Fees for OAS members are $300; non-members $475; students $50. Further details: OAS, 793-A Foothill Blvd, PMB 119, San Luis Obispo, CA 93405 USA (tel: +1 805 534 0300; fax: +1 805 534 9030; email: info@eyeanaesthesia.org; website: www.eyeanaesthesia.org).

Glaucoma Society 24th Annual Meeting and Dinner

The Glaucoma Society 24th Annual Meeting and Dinner will take place on 20 November 2003, from 8:30 am to 5:00 pm at The Royal College of Physicians, London, UK. Further details: Ms Janet Flowers (email: glauasc@ukeire.freeserve.co.uk).

Detachment Course with international faculty on: Retinal and Vitreous Surgery with Case Presentations preceding the Annual Meeting of Iranian Society of Ophthalmology

The detachment course with international faculty on: Retinal and Vitreous Surgery with Case Presentations preceding Annual Meeting of Iranian Society of Ophthalmology will be held on 29-30 November 2003 and 1-4 December 2003 respectively, at the Razi Conference Center, Hemmat Hyw, Tehran, Iran. Further details: Scientific programme: Prof Ingrid Kreissig, University of Tuebingen, Schleichstr. 12, Bruningenbau, 72076 Tuebingen, Germany (tel: +49 7071 295209; email: ingrid.kreissig@med.uni-tuebingen.de). Local organisation: Dr Arman Masheyekhi, Dr Siamak Moradian, Dept of Ophthalmology, LabbaniJejad Medical Center, Pardangan Ave, Boosan 9, Tehran, 16666, Iran (fax: +98 21 254 9039; email: labbafi@hotmail.com).

5th International Symposium on Ocular Pharmacology and Therapeutics (ISOPT)

The 5th International Symposium on Ocular Pharmacology and Therapeutics (ISOPT) will take place 11-14 March 2004, in Monte Carlo, Monaco. Please visit our website for details of the scientific programme, registration, and accommodation. To receive a copy of the Call for Abstracts and registration brochure please submit your full mailing details to http://www.kenes.com/isopt/interest.htm. Further details: ISOPT Secretariat (website: www.kenes.com/isopt).

XVth Meeting of the International Neuro-Ophthalmology Society


4th International Congress on Autoimmunity

The 4th International Congress on Autoimmunity will take place 3-7 November 2004 in Budapest, Hungary. The deadline for the receipt of abstracts is 20 June 2004. Further details: Kenes International Global Congress Organisers and Association Management Services, 17 Rue du Cendrier, PO Box 1726, CH-1211 Geneva 1, Switzerland (tel: +41 22 908 0488; fax: +41 22 732 2850; email: autoim04@kenes.com; website: www.kenes.com/autoim2004).