Cellular infiltration of Cloquet's canal

EDITOR,—Biomicroscopic evaluation of the Cloquet’s canal is difficult in a normal eye. Although Goldmann, based on his extensive biomicroscopic studies,1 described as early as 1954 the detailed biomicroscopic anatomy of it, normally only parts of the Cloquet’s canal are available for direct biomicroscopic observation.

In this report I present a case in which not only the entire course of the Cloquet’s canal became visible by a marked cellular infiltration of the vitreous, but the cell infiltration itself dramatically improved the visibility of fine structures of the surrounding vitreous.

CASE REPORT

A 9-year-old white boy discovered visual deterioration of his right eye by the accidental covering of his right eye. The patient’s previous medical history was uneventful: chickenpox and only the entire course of the Cloquet’s canal was uneventful: chickenpox and epidemic parotiditis occurred in early childhood.

Uncorrected visual acuity was 20/20 right eye and 20/200 left eye, which improved to 20/40 with a +3.0 D correction. Intracocular pressures were 12 and 13 mm Hg, respectively. Biomicroscopic examination of the right eye was unremarkable. The left eye was quiet, slit lamp examination revealed partial cataract, a vitreous flare, and cell infiltration in the anterior vitreous. Biomicroscopy of the vitreous with a contact lens released diffuse flare and cell infiltration. Cloquet’s canal was made distinct by a marked cellular infiltration (Fig 1). The course of the central canal curved down in the vicinity of the retina and ascended towards the lens. Cell infiltration around the canal emphasised the radial structure of the child’s vitreous, surrounding the course of the Cloquet’s canal (Fig 2). There were no abnormalities in the fundus or on the pars plana. Fluorescein angiography could not be performed because of the cataract, the vitreous haze, and the patient’s inability to cooperate.

Results of general physical examination and chest x ray were unremarkable. Laboratory evaluation showed an erythrocye sedi mentation rate of 5 mm in the first hour, a negative rheumatoid factor, a normal complete blood, normal titres against Toxoplasma, Toxocara, Brucella, Listeria, Lyme, and HIV. A clinical diagnosis of chronic posterior uveitis was made, but specific therapy was not considered.

After follow up of 1 year, visual acuity and the partial cataract in the left eye remained unchanged, the structure of the vitreous had become disorganised, the vitreous haze and the infiltration of Cloquet’s canal disappeared, and only a limited number of cells in the vitreous was isolated from intraocular fluid containing lens aspirate, 3½ months after complete regression of the disease. We believe that this is the first such original finding to be reported.

COMMENT

During development and early childhood Cloquet’s canal extends almost in a direct anteroposterior course from the optic disc to the posterior capsule of the lens. With aging, Cloquet’s canal changes: in the adult it emerges from the optic nerve head, and in the posterior part of the vitreous cavity it curves towards the posterior capsule of the lens. With aging, Cloquet’s canal changes: in the adult it emerges from the optic nerve head, and in the posterior part of the vitreous cavity it curves down in the vicinity of the retina and ascended towards the lens. The canal becomes obliterated in its middle part in the adult eye. In some cases saccular dilatation of the canal may occur. Under pathological conditions the course or the visibility of the Cloquet’s canal can change considerably. Uveitis may cause greater delineation of the canal in the area of the retrolental space (ascending phenomena), whereas chorioretinitis of the posterior pole may cause the canal to turn downwards in front of the optic disc. Cells may even infiltrate the Cloquet’s canal in front of the retina. It has also been suggested that intravitreal traction on the optic pit by the anomalous Cloquet’s canal may have a role in the development of macular detachment in pits of the optic nerve. The present case clearly highlights these changes to the biomicroscopic anatomy of the vitreous caused by dense cellular infiltration of the vitreous. This case clearly demonstrates: the entire intravitreal course of the Cloquet’s canal an obliteration of its anterior parts the presence of saccular dilatation of the canal in the anterior third of the vitreous cavity the effect of the cellular infiltration on the intravitreal course of the infiltrated Cloquet’s canal and the subtle radial structures of the vitreous, surrounding the course of the Cloquet’s canal.


Demonstration of herpes simplex virus from lens aspirate in healed acute retinal necrosis syndrome

EDITOR,—The herpes group of viruses, mainly the varicella zoster virus (HZV), and more rarely, the herpes simplex (HSV) types 1 and 2, and cytomegalovirus (CMV), have been demonstrated in aqueous humour, vitreous, or retinal biopsy specimens in the active phase of acute retinal necrosis (ARN), by isola tion, immunological methods, electron microscopy, and polymerase chain reaction. However, several reports indicate the inability to demonstrate the virus from intraocular specimens in the healed stage of the disease.

We report a case of ARN in which HSV 1 was isolated from intraocular fluid containing lens aspirate, 3½ months after complete regression of the disease. We believe that this is the first such original finding to be reported.

CASE REPORT

A 23-year-old man presented with a history of headache, sudden blurring of vision, and mild pain in the left eye for 3 weeks. Six years earlier the patient had lost his vision in the right eye following an acute attack of inflammation, the details of which were not known. His vision was no light perception in the right eye and 6/36 in the left eye. The right eye had band-shaped keratopathy, organised exudates in the anterior chamber, and complicated cataract. The left eye showed multiple large keratic precipitates, aqueous flare 2+, aqueous cells 2+. There was no view of the fundus in the right eye. The left fundus showed typical confluent areas of retinal
necrosis in the mid periphery with vitreous hemorrhage suggestive of ARN.

The patient was treated with intravenous aciclovir 1500 mg/m² of body surface area/day in three divided doses, oral prednisolone, 40 mg per day, topical betamethasone hourly, and atropine twice daily, for 1 week. His visual acuity improved to 6/18 in the left eye with regression of inflammation. He was then treated with oral aciclovir 400 mg, five times daily for 4 weeks. But subsequently the patient developed dense vitreous membranes obscuring the fundus details and a pars plana vitrectomy was carried out 10 days later. Vitreous aspirate on immunofluorescence (IF), using a panel of antibodies for HSV (Dako A/S, Denmark), VZV (polyvalent human serum), and CMV (Dako A/S, Denmark) showed the HSV viral antigen. Vitreous aspirate also revealed both anti-HSV IgM and IgG at >1:40 dilutions by enzyme linked immunosorbent assay (ELISA). An ELISA test for HVZ and CMV showed no antibody. A week later, the retina detached and was reattached by retivectomy with scleral buckling. On follow up after 1 month there was complete regression of retinitis with no evidence of inflammation in the anterior segment or vitreous cavity. However, vision deteriorated due to progressive nuclear sclerosis. An extracapsular lens extraction was performed intraocular lens implantation was performed 3½ months later. The lens aspirate was inoculated into vero cell line. It showed the growth with the characteristic cytopathic effect of HSV. The isolated virus was identified as HSV by IF (Fig 1) which was further confirmed by a neutralisation test as HSV type 1 using HSV 1 and HSV 2 antisera. CMV and VZV were not isolated. The patient had no evidence of active retinitis at 2 months’ follow up after cataract extraction.

**Comment**

Thompson *et al* have reported three cases of ARN due to possible reactivation of HSV type 2. Recurrence in ARN, though rare, has been reported in the same eye even after 6 years. Our case indicates that the virus causing ARN can remain viable in the eye long after clinical regression and can probably play a potential role in recurrence due to reactivation.

**JYOTIRMAI BISWAS**

HAJIB N MADHAVAN

SENGAMEDU S BADRINATH

Medical and Vision Research Foundation, 18 College Road, Madras - 600 006, India

Correspondence to: Dr Jyotirmay Biswas.

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**Figure 1** Large macular hole with upright edges 6 weeks after Nd:YAG laser accident. The visual acuity was 0.2.

**Figure 2** Posterior pole 9 month after vitreectomy and thrombocyte adhesion. The edges of the hole are reattached, centrally an retinal pigment epithelium hypertrophy developed. The visual acuity was 0.6.

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Vitreectomy and autologous thrombocyte adhesion of an accidental macular hole caused by Nd:YAG laser

**Editor,—** A 30-year-old physics scientist looked accidently with his left eye directly into the path of a 1.064 nm Nd:YAG laser beam. The laser beam lasted 1 second and caused a dramatic decrease in vision in this eye. Upon examination at the local hospital, the patient’s corrected vision was 0.16 for the left eye, the intraocular pressure (IOP) was within normal range, and the cornea and lens were unremarkable. On funduscopy, an obvious retinal defect, partially covered with blood, was observed in the region of the fovea. Vitreal bleeding could be seen above the temporal retinal vessels. The ophthalmic examination of the right eye was unremarkable.

Initially, the patient was treated with high dose corticosteroids and acetazolamide. Ten days after the accident, he was transferred to our department for evaluation. At this time the corrected vision in the patient’s left eye with eccentric fixation was 0.2. A well defined macular hole (approximate diameter 300 nm) with upright edges was observed; its centre corresponded exactly with the foveola (Fig 1). The vitreal haemorrhage had decreased considerably.

Over the following 4 weeks, no further treatment was given. As there was no change in the size of the foramen or an improvement in the patient’s vision 6 weeks after the accident, a vitreectomcy of the left eye was undertaken. It included removal of the posterior vitreous membrane, particularly in the area of the edges of the hole. To aid its closure, an autologous thrombocyte concentrate (ATC) was placed over the retinal hole. The vitreal chamber was subsequently filled with 30% SF, gas and the patient lay on his back for 6 hours, followed by a strict programme of prone position for 7 days. The postoperative course was uneventful.

During the follow up investigations in the first, second, third, sixth, and ninth postoperative months, a gradual increase in the patient’s vision to 0.6 was observed. The patient had no metamorphopsia and the size of the central scotoma remarkably decreased. There was no development of a nuclear cataract following gas vitreal replacement. In the first postoperative months, the edges of the former retinal hole were smoothly reattached and the foramen itself could only be seen with difficulty. From the fourth postoperative month onward, increasing pigmentation in the region of the hole could be observed (Fig 2); this corresponded with a further increase in the patient’s vision.

**Comment**

Laser beam accidents occur mainly in those professionally involved with lasers. The majority of the ocular accidents involve the Nd:YAG laser, which is used in medicine for diagnostic and therapeutic purposes as well as in many areas of research. The mechanism of injury differs from the thermal retinal injuries caused by argon, krypton, diode, or colour lasers and involves the photomechanical and thermal tearing of the retinal tissue. Nd:YAG lasers use very short laser pulses of 1 μs or shorter. They produce mechanical side effects like expansive microexplosions by plasma formation. With the Q-switched operation mode pulse durations of a few nanoseconds to a few microseconds can be created. As the energy is delivered in short time spans, very high peak powers up to hundreds of megawatts can be emitted, which lead to tissue disruption.

In milder injuries retinal or vitreal haemorrhages only are observed. The visual prognosis in the more severe injuries with tissue disruption is dependent on the distance of the retinal hole from the fovea. The usual conservatively treated patient experiences a severe and permanent decrease in vision.

Thach *et al* reported five cases of Nd:YAG laser beam injuries whereby the patients were treated conservatively. In two of these five patients, the resulting retinal holes included the fovea and 1 year later, the visual acuity remained at 0.3 and 0.05, respectively. In one of the patients with a centrally situated hole, a spontaneous closure occurred within 3 weeks following the accident. This corresponded with an increase in the patient’s visual acuity. The two remaining patients had paravascular holes and the visual acuity was not severely affected by the accident. In our patient, the retinal hole was large with involvement of the fovea and a severe decrease in visual acuity to 0.16. As there was no sign of a possible spontaneous closure 6 weeks after the accident, a similar operative procedure to that performed in older patients with idiopathic macular holes was performed. Idiopathic holes are thought to be caused by a tangential traction of the posterior vitreous or an epiretinal membrane resulting in a foveolar dehiscence and enlargement of the retinal hole. The rationale in performing a vitrectomy is to relieve the traction on the rims of the hole by vitreous cortical peeling and to achieve...
reattachment of the retina and recovery of the sensory function of the reattached photoreceptors. Chorioretinal adhesion around the region of the foramen edges has been aided by several adjuvants. Our experience with ATC and their pilot study. The preparation of the autologous thrombocyte concentrate followed the technique described by Gaudric et al. and was performed just before vitrectomy.

Correspondence to: S Potthöfer.

Accepted for publication 1 May 1997

Aniridia in a patient with tuberous sclerosis

EDITOR,—Tuberous sclerosis is a multisystem disorder of autosomal dominant inheritance. Associated pathological ocular findings include depigmented retinal areas and retinal hamartomas. Less common are the iris abnormalities such as depigmentation or atypical colobomas. We report the first described case of aniridia in a patient with tuberous sclerosis.

CASE REPORT

A 62-year-old white man with inherited tuberous sclerosis, diagnosed in his early childhood, was referred for ophthalmic assessment. The classic diagnostic triad of the disease was present—mental retardation, epilepsy, and anoma sebaceum (Fig 1). Systemic involvement also included documented renal hamartomas resulting in renal failure and kidney transplantation.

His past ocular history was unremarkable, including denials of any episodes of ocular redness. The patient denied instillation of any local treatment. The best corrected visual acuity was 20/80 in his right eye and 20/200 in his left eye. On biomicroscopic examination, corneas were clear except superficial opacification in the periphery. Both anterior chambers were deep. The left eye had an almost complete aniridia on 360°, with only a slim, unreactive portion of the iris attached to the angle (Fig 2). The patient stated that he had always noted the absence of the left iris. Phacotomie instillation in the left eye had no effect on the iris morphology. Gonioscopy did not show presence of anterior synchiae and the angle was normal, totally open. The pupil in the other eye was round, of normal size, but the iris presented diffuse stromal defects. Intraocular pressure, as determined with applanation tonometry was 14 mm Hg in both eyes and no signs of glaucoma were detectable. A left focal cataract was present and the lens had a minimal nasal subluxation (Fig 2). Fundus examination of the left eye revealed a yellowish-white mass above the macula, consistent with a retinal hamartoma, but the patient refused a fluorescein angiography.

COMMENT

Iris abnormalities such as hypopigmented spots or atypical iris colobomas are rare in patients with tuberous sclerosis. Previous reports suggest an embryological explanation of these anomalies by faulty migration of cells derived from the neural crest. In our patient, the origin of the almost complete absence of the iris was difficult to determine. Secondary iris atrophy was ruled out by the clinical features and as there was neither clinical evidence of previous surgery, trauma, acute glaucoma episodes, heterochromia, or ocular inflammation. No posterior embroyoxon or systemic stigmata of Rieger’s syndrome were evident.

The almost complete absence of the iris associated with a focal cataract, subluxation of the lens and peripheral opacification of the corneae were consistent with the diagnosis of aniridia, which is a mimiser for iris hypoplasia. Other ocular signs described in aniridia (focal hypoplasia, nystagmus) were not found in our case. This has already been described as manifestation of the large clinical variability of the disease. To our knowledge, inherited aniridia, as in our patient, has never been described in tuberous sclerosis.

Aniridia is an autosomal dominant inherited disease, but sporadic non-familial forms exist. Our patient had no contact with his family and had no descendants so we were unable to document the transmission of his iris hypoplasia.

It is difficult to establish whether the occurrence of aniridia and autosomal dominant tuberous sclerosis in this patient was coincidental or if there is a genetic link between them. Aniridia arises from mutations in the PAX6 gene located on the chromosome 11p13, resulting in a defect in the control of neural crest cell migration into the developing eye. In tuberous sclerosis, linkage studies revealed mutations on chromosomes 9q34 and 16p13. Genetic studies initially suggested that mutations on chromosome 11 (also involved in aniridia) might be responsible for tuberous sclerosis, but new data do not provide evidence for such a mutation. There are no available data that could suggest a common mutation for both aniridia and tuberous sclerosis. As our patient refused genetic testing, further observations and chromosomal studies are necessary to establish if occurrence of aniridia in tuberous sclerosis is stochastic or due to a genetic link.

Correspondence to: Dan Milea, MD, Clinique Ophtalmologique Universitaire, Hôpital Edouard Herriot, Lyon, France.

Accepted for publication 28 April 1997

Squamous cell carcinoma of the palpebral conjunctiva

EDITOR,—Squamous cell carcinoma is a malignancy that occurs frequently on the skin of the eyelid; however, it is uncommon on the conjunctiva. The conjunctival form of the squamous cell carcinoma constitutes 7–15% of conjunctival and corneal epithelial tumors. This malignancy normally behaves in a non-aggressive fashion, and the most rare form of this tumour is the palpebral conjunctival squamous cell carcinoma. We report a
Incisional biopsy revealed squamous cell carcinoma of the superior fornix, measuring 10 mm from the posterior eyelid margin to the palpebral conjunctival surface, extending 2.5 papillary tumoral mass over the tarsus in another institution with recurrence of the papilloma. The tumour was surgically excised and referred to us with a clinical diagnosis of ocular discomfort and lacrimation.

A 69-year-old woman with a 2 year history of conjunctival squamous cell carcinoma of the palpebra is very rare. In our review of the literature, it appears that only seven cases have been reported previously. Recently, human papilloma virus (HPV) antigens and DNA have been detected in conjunctival neoplasia, and their implication in the pathogenesis of squamous cell carcinoma and other epithelial tumours has been suggested. We applied in situ hybridisation to detect HPV types 6, 11, 16, 18, 31, 33, and 51 in paraffin embedded tissue. HPV was not present in the specimens. This finding suggests that, in this patient, HPV was not associated with squamous cell carcinoma of the conjunctiva.

Conjunctival squamous cell carcinoma can be treated successfully with excision, radiotherapy, or cryotherapy. In the series of three patients described by Theodore, conjunctival squamous cell carcinoma was treated with intense radiotherapy. Thyerosis reported on two patients with papillary squamous cell carcinoma of the palpebral conjunctiva. One of these patients received radiation therapy and the other was treated with excision of the tumour.

**Figure 1** A 10 × 12 mm papillary lesion is present at the upper palpebral conjunctiva.

**Figure 2** Histopathological study revealed a moderately differentiated squamous cell carcinoma (haematoxylin and eosin, × 130).
CORRESPONDENCE

On the treatment of amblyopia

EDITOR—I have read with interest the paper by Foley-Nolan et al.1 on the use of atropine as an alternative to occlusion for treating ambylopia. Presumably all the patients entering their trial had strabismus, otherwise there would not have been suppression but some degree of normal binocularity. It should be pointed out that the daily instillation of atropine into the fixing eye, as advocated by Worth,2 may be of use only in those cases in whom the visual acuity of the squinting eye is ‘not much less than 6/36’, in fact, the mean visual acuity in 36 of their atropine treated group was 6/60. But if amblyopia ex anopsia is of such magnitude that central fixation is lost, in the most distressing cases cyclopia of the fixing eye will prove useless, the patient will keep on using the fixing eye, treated with atropine or not. Only occlusion, permanent occlusion, 6 days a week, will do the trick. On the other hand, if there is no strabismus, but orthotropia, and some degree of normal binocularity, the results of any treatment, and the pertinent indications, would depend on the ultimate cause of the disturbance.

I felt that a chance to clarify the point was in order.

A URRETS-ZAVALLA
Casilla De Correo 101, 5000 Cordoba, Argentina


Reply

EDITOR—In our published trial,1 the patients were assigned to two treatment groups, group A where they were treated with atropine penalisation, and group P where they were treated with occlusion on therapy. In both groups approximately 90% had strabismus. The mean visual acuities in groups A and P before treatment were 6/50 and 6/60 respectively.

Previous experimental work has shown that a reduction in spatial pattern stimuli alone and not light is all that is necessary to shift dominance to the non-dominant eye.3 Therefore, it is reasonable to expect that penalisation by degrading visual stimuli should be effective even if there was no strabismic deviation.

We agree that, previously, atropine penalisation treatment of amblyopia was considered to be useful only for lesser degrees of amblyopia. However, as can be seen from our results substantial improvements were achieved in group A, where the pretreatment mean visual acuity was 6/50.

AISLING FOLEY-NOLAN
MICHAEL O'KEEFE
The Children’s Hospital, Temple Street,
Dublin 1, Ireland


Retinopathy of prematurity

EDITOR—Treating retinopathy of prematurity (ROP) is a challenge requiring significant degrees of skill and patience. The surgeon usually has to treat the peripheral retina, a task that is frequently made difficult because of the small eye in the premature baby and also because in many cases visualisation is further complicated by poor pupillary dilatation secondary to the disease process. Optical clarity, for laser treatment in particular, is therefore of paramount importance in attempting to maintain adequate visualisation in these difficult cases. However, it is unavoidably compromised as a result of corneal drying necessitated by the need not only for a speculum but also because the background ambient temperature is frequently elevated above normal ‘room temperature’. Corneal clarity is maintained by the application of balanced salt solution (BSS) or hypromellose 0.3% drops which are administered by an assistant at ‘convenient’ moments during the procedure.

Ocucat (Storz Ophthalmics Products) is a sterile, non-pyrogenic, optically clear viscoelastic material containing 2% hydroxypropyl methylcellulose. It is usually indicated as a surgical aid in procedures of the anterior segment of the eye, most commonly cataract surgery.4 It is supplied in a 1 ml single use glass syringe.

Recently, we have used Ocucat, instead of hypromellose drops as an aid to improving and maintaining optical clarity during the treatment of ROP. We find that it permits excellent visualisation and because of the viscous nature of the material it remains in situ throughout the procedure. Its ocular safety is beyond any reasonable doubt and indeed we have seen no adverse effects on the eye since we began using Ocucat; but nevertheless we irrigate the eye thoroughly on completing the treatment. We also find that the wetting effect of Ocucat can be prolonged by the occasional administration of hypromellose drops.

In conclusion, we have found the viscoelastic Ocucat, most commonly used in intracocular surgery, to be a valuable adjunctive tool in the management of threshold ROP. Finally, we have no commercial interest in the product.

DAVID KENT
DAVID CLARK
Wilton Hospital, Rice Lane, Liverpool L9 1AE


OBITUARY

Peter Llewellyn Blaxter

Peter Blaxter died suddenly on the 3rd May 1997 from a heart attack. He had suffered from circulatory problems for some years and shortly after his retirement developed a homonymous hemianopia as a result of an occipital lobe infarct, a cruel blow which he bore with stoicism.

He was born in 1918 and educated at Tonbridge School. His medical training was at St John’s College, Cambridge and Guy’s Hospital. Following qualification in 1942 he held house appointments at Guy’s and following a three year period of National Service began his ophthalmic career at Guy’s and Moorfields. In 1950 he was appointed consultant ophthalmologist at Fulham Hospital. During the period 1948–54 he also held the post of research assistant at the Institute of Ophthalmology. In 1954 he left London and moved north to Manchester where until 1982 he was consultant ophthalmic surgeon at the Manchester Royal Eye Hospital. From 1956 to 1982 he was also ophthalmic surgeon to the Manchester Jewish Hospital. During his professional career he was a vice president of the ophthalmic section of the Royal Society of Medicine, president of the North of England Ophthalmological Society in 1971, and also held the vice presidency and was a council member of OSUK. From 1967 to 1982 he was on the Council of the Faculty of Ophthalmologists and was master of the Oxford Ophthalmological Congress in 1975 and 1976. He was an examiner for the Ophthalmic Nursing Board, the British Orthoptic Board, and on the Court of Examiners of the Royal College of Surgeons for several years.

I had the privilege of training under Peter Blaxter. He was a delight to work with and had a deceptively simple approach to clinical ophthalmology. However, behind his almost casual approach lay a very keen mind which had the ability to lay bare the important facts very rapidly. Surgically he was deft and gentle and never carried out any unnecessary manoeuvres, perhaps a result of his training with H B Stallard. His main interest was ocular motility, again perhaps influenced by another of his contemporaries, T Keith Lyle. Peter was a very generous host, and many people including his staff enjoyed his hospitality at Wilmalow.

He is survived by his wife Patricia, two sons, and a daughter who is a general practitioner in South Africa.

W G CODDINGTON
6th International Paediatric Ophthalmology Meeting
The 6th International Paediatric Ophthalmology Meeting will be held on 24–25 September 1997 in Dublin, Ireland. Topics include grand round, neuro-ophthalmology, strabismus, childhood tumours. Further details: Ms Kathleen Kelly, Suite 5, Mater Private Hospital, Eccles Street, Dublin 7, Ireland. (Tel: +353 838 4444, ext 1759; fax: +353 838 6314.)

British and Eire Association of Vitreoretinal Surgeons (BEAVRS)
A meeting of the British and Eire Association of Vitreoretinal Surgeons (BEAVRS) will be held in Birmingham on 16–17 October 1997. Further details: Mr Graham R Kirkby, consultant ophthalmic surgeon, The Birmingham and Midland Eye Centre, City Hospital, NHS Trust, Birmingham B18 7QU. (Tel: 0121-554 3801; fax: 0121-507 6791.)

Diabetic Retinopathy and Vitreoretinal Surgery Advanced Topics
A meeting under the auspices of the Office of Continuing Medical Education will be held on 17–18 October 1997 at the Thomas B Turner Building, Johns Hopkins Medical Institutions, Baltimore, Maryland, USA. Further details: Office of Continuing Medical education, Johns Hopkins Medical Institutions, Turner 20, 72 Rutland Avenue, Baltimore, MD 21205, USA. (Tel: (410) 955-2959; fax: (410) 955-0807; email: cmenet@som.adm.jhu.edu)

International Centennial Meeting on Pseudoanxanthoma Elasticum
PXIE International, Inc, along with the National Institute of Arthritis, Musculoskeletal and Skin Diseases (NIH), is sponsoring an International Centennial Meeting on Pseudoanxanthoma Elasticum (PXE) on 6–7 November 1997 in Bethesda, MD, USA. The meeting will focus on genetic, extracellular matrix, and clinical issues. Further details: Sharon Terry, MA, President PXE International, Inc, 23 Mountain Street, Sharon, MA 02067, USA. (Tel and fax: 617 784 3817; email: pxie@tiac.net)

10th Annual Wilmer Institute’s Current Concepts in Ophthalmology
The 10th Annual Wilmer Institute’s Current Concepts in Ophthalmology will be held on 11–13 December 1997 at the Johns Hopkins Medical Institutions, Baltimore, Maryland. Further details: Program Coordinator, Johns Hopkins Medical Institutions, Office of Continuing Medical education, Turner 20/72 Rutland Avenue, Baltimore, MD 21205, USA. (Tel: 410 955-2959; fax: 410 955-0807; email: cmenet@som.adm.jhu.edu; homepage: http://ww2.med.jhu.edu/cme)

20th Annual Wilmer Institute’s Current Concepts in Ophthalmology
The 20th Annual Wilmer Institute’s Current Concepts in Ophthalmology will be held on 5–10 February 1998 at the Hyatt Regency Cerromar Beach Hotel, Dorado, Puerto Rico. Further details: Program Coordinator, Johns Hopkins Medical Institutions, Office of Continuing Medical education, Turner 20/72 Rutland Avenue, Baltimore, MD 21205, USA. (Tel: 410 955-2959; fax: 410 955-0807; email: cmenet@som.adm.jhu.edu; homepage: http://ww2.med.jhu.edu/cme)

2nd International Symposium on ARMD
The 2nd International Symposium on ARMD will be held at Glasgow University, Scotland under the auspices of the Royal College of Ophthalmologists on 16–18 September 1997. Further details: Dr G E Marshall, Eye Department, Western Infirmary, 38 Church Street, Glasgow G11 6NT, UK. (Tel: 0141 211 2094; fax: 0141 339 7485; email: gem1b@clinmed.gla.ac.uk)

First Combined International Symposium on Ocular Immunology and Inflammation
The First Combined International Symposium on Ocular Immunology and Inflammation will be held in Amsterdam on 21–26 June 1998. Further details: Eurocongress Conference Management, Jan van Goyenkade 11, 1075 HP Amsterdam, the Netherlands. (Tel: +31-20-6793411; fax: +31-20-6737306; internet http://www.solution.nl/ico-98)

2nd International Conference on Ocular Infections
The 2nd International Conference on Ocular Infections will be held on 22–26 August 1998 in Munich, Germany. Further details: Professor J Frucht-Pery, 2nd International Conference on Ocular Infections, PO Box 50006, Tel Aviv, 61500, Israel. (Tel: 972 3 5140000; fax: 972 3 5175674 or 5140077; email: ocular@kenes.com)
INSTRUCTIONS FOR AUTHORS

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Manuscripts should be sent to the editor who selects them on the basis of their suitability for the journal and of reports from independent referees. Manuscripts are acknowledged on receipt and the majority (>80%) are sent for review. Those that are not reviewed are returned to the author as rapidly as possible so that they may be submitted elsewhere.

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Cellular infiltration of Cloquet's canal

TIBOR MILIBÁK

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