LETTERS TO THE EDITOR

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 the left eye by the incidental covering of his right eye. The patient's previous medical history 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. Intraocular 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 revealed 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 erythrocyte sedimentation 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 retrolental space could be observed. Fundus examination showed no abnormality.

COMMENT

During development and early childhood Cloquet's canal extends almost in a direct anteroposterior course from the optic disc 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, while in the anterior vitreous it emerges again towards the lens.² The canal becomes obliterated in its middle part in the adult eye. In some cases saccular dilatation of the canal may occur.¹ 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),^{2,3} 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.⁴ 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.

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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),¹ have been demonstrated in aqueous humour,² vitreous,³ or retinal biopsy⁴ specimens in the active phase of acute retinal necrosis (ARN), by isolation, 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.^{4,5}

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

Figure 1 Slit lamp biomicroscopic appearance of heavily infiltrated Cloquet's canal seen through the QuadrAspheric contact lens. Central part of the vitreous space. The normal course of the central canal is altered by the cellular infiltration. Saccular dilatations are present at the anterior end of the tractus. The whole course of the Cloquet's canal is surrounded by a layer of infiltrated cloudy vitreous where signs of the radial orientation can be seen.

Figure 2 Computer graphic of Figure 1 taken with embossed filter. Computer imaging enhances visibility of the subtle radial structures of the vitreous, surrounding the course of the Cloquet's canal.

Figure 3 Microphotograph showing HSV antigen in vero cells inoculated with lens aspirate detected by immunofluorescence (magnification $\times 112$).

necrosis in the mid periphery with vitreous haze, 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 antisera of HSV (Dako A/S, Denmark), VZV (polyclonal 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 test assay (ELISA). An ELISA test for HZV and CMV showed no antibody. A week later, the retina detached and was reattached by vitrectomy with scleral buckling. On follow up after 1 month there was complete regression of retinitis with scarring and no evidence of inflammation in the anterior segment or vitreous cavity. However, vision deteriorated due to progressive nuclear sclerosis. An extracapsular cataract extraction with 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 2.⁶ Recurrence in ARN, though rare, has been reported in the same eye even after 6 years.⁷ 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.

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

EDITOR,—A 30-year-old physics scientist looked accidentally with his left eye directly into the path of a 1.064 nm Nd:YAG laser beam. The laser variables were not known. He experienced 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 inferior vessel arcade. 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 circular 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 vitrectomy 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 vitreous chamber was subsequently filled with 30% SF₆ 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 uncomplicated.

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 lost his 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

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 months after vitrectomy and thrombocyte adhesion. The edges of the hole are reattached, centrally a retinal pigment epithelium hypertrophy developed. The visual acuity was 0.6.

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.¹⁻⁴ 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.² The mechanism of injury differs from the thermal retinal injuries caused by argon, krypton, diode, or colour lasers³ and involves the photomechanical and thermal tearing of the retinal tissue.³

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.³

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.⁴

Thach *et al* reported five cases of Nd:YAG laser beam injuries whereby the patients were treated conservatively.⁴ 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 parafoveal 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.⁵ 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 in patients with idiopathic macular holes suggests that the closure rate is higher when this adhesive is combined with the gas tamponade instead of the latter procedure alone; this is supported by the results of Gaudric *et al* in their pilot study.⁶ The preparation of the autologous thrombocyte concentrate followed the technique described by Gaudric *et al*⁶ and was performed just before vitrectomy.

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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.¹ Less common are the iris abnormalities such as depigmentation or atypical colobomas.^{2–5} We report the first

Figure 1 Characteristic facial distribution of adenoma sebaceum.

Figure 2 Hypoplasia of the left iris associated with focal atrophy. There is also a nasal subluxation of the lens.

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 disease¹ was present—mental retardation, epilepsy, and adenoma sebaceum (Fig 1). Systemic involvement also included documented renal hamartomas resulting in renal failure and kidney transplantation.

His past ocular history was unremarkable, including denial of ocular trauma or 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. Pilocarpine instillation in the left eye had no effect on the iris morphology. Gonioscopy did not show presence of anterior synechiae 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.³ 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 history nor clinical evidence of previous surgery, trauma, acute glaucoma episodes, heterochromia, or ocular inflammation. No posterior embryotoxon 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 cornea were consistent with the diagnosis of aniridia, which is a misnomer for iris hypoplasia.

Other ocular signs described in aniridia (foveal hypoplasia, nystagmus) were not found in our case. This has already been described as manifestation of the large clinical variability of the disease.⁶ 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 same patient is 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.⁷ 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 in tuberous sclerosis, but new data do not provide evidence for such a mutation.⁸ 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 chromosome studies are necessary to establish if occurrence of aniridia in tuberous sclerosis is stochastic or due to a genetic linkage.

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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 tumours.^{1,2} This malignancy normally behaves in a non-aggressive fashion, and the most rare form of this tumour is the palpebral conjunctival squamous cell carcinoma.³ We report a

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).

case of primary conjunctival squamous cell carcinoma of the palpebra treated successfully with surgical excision and cryotherapy.

CASE REPORT

A 69-year-old woman with a 2 year history of ocular discomfort and lacrimation was referred to us with a clinical diagnosis of papilloma. The tumour was surgically excised in another institution with recurrence of the tumour in the same eyelid location.

Eversion of the left upper eyelid disclosed a papillary tumoral mass over the tarsus in the palpebral conjunctival surface, extending 2.5 mm from the posterior eyelid margin to the superior fornix, measuring 10 × 12 mm (Fig 1). Incisional biopsy revealed squamous cell

carcinoma. No regional adenopathy was present.

Treatment consisted of surgical excision of the tumour with a 2 mm safety margin, and cryotherapy was applied to the underlying tissue using the cryoprobe technique. Histopathological examination disclosed a moderately differentiated squamous cell carcinoma arising from the palpebral conjunctiva (Fig 2). Malignant cells formed nests or sheets of epithelial cells. They had scant eosinophilic cytoplasm and intercellular bridge. Mitosis was sparsely observed in the specimens. The basement membrane of the conjunctiva was infiltrated in several areas by the tumoral cells.

Three months after surgical excision of the tumour, a multiple conjunctival and tarsal biopsy was performed on the upper eyelid. The findings were negative for tumour. In the tarsus abnormal epithelial cell growth was observed but no malignant changes were found. In a 12 month follow up there was no recurrence of the tumour.

In situ hybridisation for human papilloma virus types 6, 11, 16, 18, 31, 33, and 51 was negative.

COMMENT

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.³⁻⁶

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.⁷ 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.⁴

Thygeson 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.⁵ Goldberg

et al reported on a patient with invasive squamous cell carcinoma of the palpebral conjunctiva, masquerading as chronic conjunctivitis, who was treated with radiotherapy for a total dose of 54 Gy.⁶ The tumour recurred after 7 weeks of follow up. This patient died from complications of metastatic breast carcinoma.

Our patient had a tumour histopathologically classified as moderately differentiated squamous cell carcinoma. Surgical and cryotherapy were successfully used without recurrence of the tumour or metastasis after a 1 year follow up.

Several reports of squamous cell carcinoma of the conjunctiva masquerading as chronic conjunctivitis have been reported.⁴⁻⁶ Our patient had a history of lacrimation and ocular discomfort which are symptoms of conjunctivitis. This factor probably delayed the correct diagnosis. Squamous cell carcinoma of the conjunctiva must be considered as a differential diagnosis in patients with unilateral chronic conjunctivitis.

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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 amblyopia. 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 cycloplegia 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.

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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.² 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.

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Retinopathy of prematurity

EDITOR,—Treating retinopathy of prematurity (ROP) is a procedure 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.

Ocucoat (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.¹⁻⁴ It is supplied in a 1 ml single use glass syringe.

Recently, we have used Ocucoat, 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 Ocucoat; but nevertheless we irrigate the eye thoroughly on completing the treatment. We also find that the wetting effect of Ocucoat can be prolonged by the occasional administration of hypromellose drops.

In conclusion, we have found the viscoelastic Ocucoat, most commonly used in intraocular 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

Walton Hospital, Rice Lane,
Liverpool L9 1AE

- 1 Glasser DB, Osborn DC, Nordeen JF, Yuan I-M. Endothelial protection and viscoelastic retention during phacoemulsification and intraocular implantation. *Arch Ophthalmol* 1991;109:1438-40.
- 2 Gregory Smith S, Lindstrom RL. 2% Hydroxypropyl methylcellulose as a viscous surgical adjunct. A multicentre prospective randomised trial. *J Cataract Refract Surg* 1991;17:839-42.
- 3 Monson MC, Manabu T, Mamalis N, Olson RJ, Olson RJ. Protective effects of Healon and Ocucoat against air bubble endothelial damage

during ultrasonic agitation of the anterior chamber. *J Cataract Refract Surg* 1991;17:613-6.

- 4 Fechner PU, Fechner MU. Methylcellulose and lens implantation. *Br J Ophthalmol* 1983;67:259-63.

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 Wilmslow.

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

W G CODDINGTON

NOTICES

Retinopathy of Prematurity

The latest issue of the *Journal of Community Eye Health* (no 22) deals with retinopathy of prematurity. Editorial by Alistair R Fielder. For further information please contact Ann Naughton, ICEH, Institute of Ophthalmology, 11-43 Bath Street, London EC1V 9EL. Tel: 0171 608 6910. Annual subscription: £25. Free to eye health workers in developing countries.

Tübingen Practical Angiography Course

The Tübingen Practical Angiography Course (International Faculty) will take place on 6 September 1997 at the Auditorium, University Dental Clinic, Osianderstrasse 2-8, Tübingen, Germany. Further details: F Gelissen, MD, Congress Secretariat Dept III, University Eye Clinic, Schleichstrasse 12, 72076 Tübingen, Germany. (Tel: +49 (0) 7071 2987448; fax: +49 (0) 7071 293746; email: ingrid.kreissig@uni-tuebingen.de)

5th International Symposium on Ocular Circulation and Neovascularisation

The 5th International Symposium on Ocular Circulation and Neovascularisation will be held on 15-19 September 1997 in Kyoto, Japan. Further details: Professor Dr Masanobu Uyama, Secretary General of the Organising Committee, Department of Ophthalmology, Kansai Medical University, Moriguchi, Osaka 570, Japan. (fax: 81-6-997-3475.)

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)

XXXIst National Ophthalmology Congress

The XXXIst National Ophthalmology Congress will be held on 16-20 September 1997 in the Istanbul Convention and Exhibition Centre, Istanbul, Turkey. Further details: Murat Karacorlu, MD, Congress Scientific Secretariat, Valikonagi Cad, Sezai Selek Sok No 8/5, Nisantasi, Istanbul 80200, Turkey. (Fax: +90 (212) 233 2425; email: mkaracorlu@iris.com.tr)

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: +3531 838 4444, ext 1759; fax: +3531 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, 720 Rutland Avenue, Baltimore, MD 21205-2195, USA. (Tel: (410) 955-2959; fax: (410) 955-0807; email: cmenet@som.adm.jhu.edu)

International Centennial Meeting on Pseudoxanthoma Elasticum

PXE International, Inc, along with the National Institute of Arthritis, Musculoskeletal and Skin Diseases (NIH), is sponsoring an International Centennial Meeting on Pseudoxanthoma 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: pxe@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 20/720 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 20/720 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>)

15th Annual Wilmer Institute's Current Concepts in Ophthalmology

The 15th Annual Wilmer Institute's Current Concepts in Ophthalmology will be held on 15-20 March 1998 at Manor Vail Lodge, Vail, Colorado. Further details: Program Coordinator, Johns Hopkins Medical Institutions, Office of Continuing Medical education, Turner 20/720 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>)

XXVIIIth International Congress of Ophthalmology

The XXVIIIth International Congress of Ophthalmology will be held in Amsterdam on 21-26 June 1998. Further details: Eurocongres 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/>)

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 27 June-1 July 1998. The meeting is sponsored by the International Ocular Immunology and Inflammation Society, the International Uveitis Study Group, and the Immunology and Immunopathology of the Eye organisation. Further details: Professor Aize Kijlstra, The Netherlands Ophthalmic Research Institute, PO Box 12141, 1100 AC Amsterdam, Netherlands (email: a.kijlstra@amc.uva.nl)

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

Adherence to the following guidelines is essential if efficient and expeditious processing of your manuscript is to be achieved. Manuscripts will be returned to authors for revision before peer review if they are submitted in incorrect format. Please indicate in a covering letter which category of paper your article represents.

The *British Journal of Ophthalmology* is an international journal covering all aspects of clinical ophthalmology and the visual/ophthalmic sciences. Contributors should consider the widely varying readership and write clear, simple articles with the minimum of technical detail. Space in the journal is limited and articles should therefore be as concise as possible. One page of text is approximately 1000 words.

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.

Manuscripts may be processed by section editors who deal with specific areas of ophthalmology including surgical retina, medical retina, neuro-ophthalmology, glaucoma, paediatric ophthalmology, ocular motility, orbital disease, anterior segment disease, oncology, lens, optics and visual sciences, laboratory sciences, pathology, and immunology. A minimum of two referees, chosen for their specific expertise, review each article.

Papers are accepted on the understanding that they have not been and will not be published elsewhere, and that there are no ethical problems with the work described. If requested, authors shall produce the data upon which the manuscript is based for examination by the editor.

Categories of papers

ORIGINAL ARTICLES

(a) Clinical science

Articles on clinical topics are research reports of a general or specialised nature comprising approximately 3000 words and 4-6 display items (Figures and Tables).

(b) Laboratory science

Articles on ophthalmic or visual sciences are research reports of experimental work generally of the same size as clinical research reports. Laboratory science papers will be included in a designated section of the journal.

Both types of original article should include the following: title; key words (up to four); address and which author address for correspondence; structured abstract (approx 200 words, headings 'Aims/background', 'Methods', 'Results', and 'Conclusion'); introduction; materials and methods; results and discussion sections; references and acknowledgements; legends for display items (Figures and Tables).

REVIEW ARTICLES

Substantive review articles will be included under the section 'Perspective' and will address any aspect of clinical or laboratory

ophthalmology. Review articles will be approximately 3000-5000 words in length including references and may contain display items (Figures and Tables). Most review articles are commissioned but uninvited reviews are welcomed. Prior discussion with the Editor is recommended. All reviews are subject to independent refereeing.

LETTERS TO THE EDITOR

Case reports will be published as 'Letters to the editor'. These are normally 500-600 words written in the form of a letter with a maximum of two display items (Figures and Tables). The letter should include an introductory section (without heading), the case report (heading: Case report) and a comment (heading: Comment), plus a maximum of 10 references.

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Letters are normally constructed in the form of scientific correspondence and are usually 200-300 words.

Preparation of manuscripts

Manuscripts will be received on the understanding that they have not been and will not be published elsewhere while under editorial review. Manuscripts may be subject to editorial revision with the author's agreement. All communications should be sent to the Editor, *British Journal of Ophthalmology*, Department of Ophthalmology, University of Aberdeen Medical School, Foresterhill, Aberdeen AB9 2ZD, Scotland, UK. (Tel: 01224 663812; Fax: 01224 663832.)

Manuscripts must be submitted in triplicate, and typed double spaced on one side of the paper only, with one inch margins. Each author must sign the covering letter as evidence of consent to publication. Revised manuscripts should be submitted as hard copy and on disk. Detailed instructions will be sent to authors on invitation to revise.

ILLUSTRATIONS

Illustrations must be submitted in triplicate. Transparencies must be accompanied by prints. Only salient detail should be included. All must be labelled with the author's name, numbered in the same order as they are cited in the text irrespective of whether they are in colour or black and white, and have the top indicated. Radiographs must be submitted as prints. Line drawings should be clearly labelled and will be redrawn to house style. The width of illustrations for the original articles should be 68 mm, 104 mm, 140 mm or, in exceptional circumstances, 176 mm, to fit the column layout of the journal. Illustrations for 'Letters to the editor' should be 56 mm or 116 mm. Stain used and a scale bar (or magnification) should be given. Legends must be typed on a separate sheet.

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Each table should be on a separate sheet, have a heading, and contain no vertical rules.

REFERENCES

In accordance with the Vancouver agreement references are cited by the numerical system. They must be typed double spaced.

References in the text must be cited in numerical order of first appearance. References in the list must be given in the numerical order in which they first appear in the text, not in alphabetical order of authors' names. References with one to six authors must include all authors' names; for references with more than six authors the first six should be given and then *et al.* Titles of journals should be abbreviated in accordance with the *Index Medicus* or given in full.¹ References to books must include names of editor(s) if there is one, town where published, name of publisher, year, volume, page numbers.²

- 1 Kaye SB, Shimeld C, Grinfield E, Maitland NJ, Hill TJ, Easty DL. Non-traumatic acquisition of herpes simplex virus infection through the eye. *Br J Ophthalmol* 1992; 76: 412-8.
- 2 Jakobiec FA, Font RL. Orbit. In: Spencer WB, ed. *Ophthalmic pathology: an atlas and textbook*. 3rd ed. Philadelphia: Saunders, 1986: 2461-76.

References will not be checked in the editorial office. Responsibility for their accuracy and completeness lies with the author.

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The work should be reported in the units used. If these were not SI units, the equivalent in SI units should be given in parentheses.

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Particular attention should be paid to the description of any sample selection process; in particular, the representativeness of the sample should be argued and the handling of any missing data justified. Authors are asked to check tables etc to ensure that missing data are accounted for, that percentages add up to 100 and that numbers in tables are not at variance with those quoted in the text. The policy of the *British Journal of Ophthalmology* is based on the statistical guidelines published in the *British Medical Journal* in 1983 and these are a useful source of information for authors (Altman DG, Gore SM, Gardner MJ, Pocock SJ. Statistical guidelines for contributors to medical journals, *BMJ* 1983; 286: 1489-93). Blanket statements on the use of statistical techniques should be avoided; it must be made quite clear in context which procedure is being used. Authors should bear in mind that relatively simple analyses are often quite adequate to support the arguments presented.

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