

LETTERS TO THE EDITOR

Severe penetrating injury due to a burst compact disc in a child

EDITOR.—Compact discs have evolved as valuable tools in education and communication.¹ Although the material is rigid due to several layers of metals and a hard lacquered surface, ocular penetrating injuries² caused by compact discs have not been reported to our knowledge.

CASE REPORT

A 9 year old boy complained of sudden visual loss after trying to bend his father's educational compact disc at home (Fig 1). He presented with an 8.5 mm (para)central corneal laceration on his left eye extending into two T-shaped lacerations in the pupillary axis. The anterior lens capsule was opened like a "can opener", the posterior capsule destroyed, and the vitreous prolapsed. x Ray did not reveal an intraocular foreign body, and ultrasound excluded a retinal detachment. Since keratoplasty à chaud was refused by the parents tedious suturing of the cornea was followed by aspiration of lens fragments via a scleral tunnel, and anterior vitrectomy and pcIOL implantation was performed (Fig 2). The clinical course was unremarkable. Post-operative visual acuity was +2.5 sph = 20/200, and a corneal graft was further discussed with the parents.

COMMENT

Hard lacquered compact discs may result in bursting if compressed horizontally. Compact



Figure 1 Educational compact disc burst into sharp-edged splinters after extreme bending by a 9 year old child. Warnings should be attached to CD cases.



Figure 2 An 8.5 mm corneal linear and two T-shaped cuts are sutured, lens aspiration, anterior vitrectomy, and pcIOL performed. VA left eye 20/200. Keratoplasty as been advised.

discs may pose a major risk for severe penetrating injuries in children trying to bend them. We suggest that compact discs should be stored away from children, and we propose that appropriate warnings for children not to attempt to bend the material should be attached to CD cases.

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Ultrasound guided cryotherapy for retinal tears in patients with opaque ocular media

EDITOR.—A symptomatic retinal tear with vitreous traction is an indication for vitreoretinal surgery. In patients with opaque ocular media—that is, cataract or vitreous haemorrhage, it is almost impossible to observe these retinal abnormalities. Preoperative standardised echography helps to locate and assess the retinal tear and to evaluate the surrounding retina. In the presence of opaque ocular media and echographic evidence of retinal tear with a flat retina, there are three possible treatments: (1) when the ocular media is clear enough, laser photocoagulation may be performed around the hole; (2) with prolonged vitreous haemorrhage, pars plana vitrectomy can be performed with endolaser; and (3) with advanced cataract, cataract extraction can be performed with laser. A new treatment approach has been suggested by DiBernardo *et al*¹ and later by Kelly *et al*.² Kelly and associates described 11 patients with dense vitreous haemorrhage which obscured the retinal tear from direct visualisation; ultrasound guided cryotherapy was performed.

CASE REPORT

A 56 year old man was examined in our outpatient clinic because of an acute decrease in visual acuity to hand movement in his left eye. In the past, the patient had undergone several laser photocoagulation treatments for retinal tears in both eyes. Slit lamp examination revealed a dense vitreous haemorrhage obscuring the retina. Intraocular pressure was in the normal range. A-scan at tissue sensitivity (Fig 1A) and B-scan ultrasound demonstrated a flat retina with a flap retinal tear located anterior to the equator at 6 o'clock (Fig 1B). The vitreous was adherent to the anterior margin of the tear and freely moved with eye movements. Right eye visual acuity was 6/6, with no ocular abnormalities. The patient was observed over 10 days and no ocular echographic changes were detected. At this stage, we performed ultrasound guided cryotherapy to the retinal tear. Under topical anaesthesia, the retinal break was detected by B-scan ultrasound and a cryoprobe was positioned on the scleral surface at the site of the retinal break. The cryoprobe indentation, closure of the tear, and ice ball formation could be demonstrated in real time on the display screen under full control of the surgeon (Fig 2). A few applications were performed

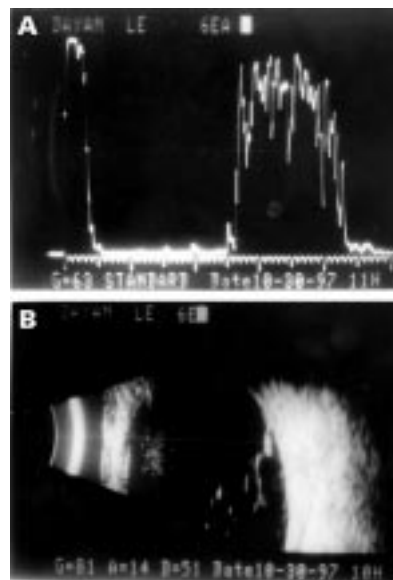


Figure 1 (A) A-scan at tissue sensitivity demonstrates a very high reflective spike from the retinal tear. (B) Longitudinal B-scan echogram demonstrates flap retinal tear with attached vitreous membrane.



Figure 2 Real time B-scan guided cryotherapy shows ice ball formation on the retinal surface around the tear.

directly on the retinal tear and to the adjacent retina under B-scan ultrasound visualisation of ice ball formation at the treated site. Four weeks later, the vitreous was clear, the retina was flat and the tear was surrounded by scar.

Ultrasound guided cryotherapy is an effective treatment for single flap retinal breaks in the presence of vitreous haemorrhage or advanced cataract obscuring direct view of the retina. Some of the patients may need laser photocoagulation after partial clearing of the vitreous haemorrhage in addition to the first treatment. Cryotherapy is inappropriate for eyes with round atrophic holes that could be missed by ultrasound. We are currently comparing the ophthalmoscopic appearance of an ice ball formation with that on echography to control the cryoapplication duration, and the amount and extent of retina treated.

This is a low cost, ambulatory procedure which is painless and relatively non-invasive compared with other methods. Further study with ultrasound guided cryotherapy is needed in cases of retinal breaks obscured by opaque ocular media.

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Corneal ectasia after trabeculectomy with mitomycin C application

EDITOR.—Trabeculectomy combined with mitomycin C is becoming an acceptable alternative in the management of refractory glaucoma. We report on a patient who had received trabeculectomy with mitomycin C and demonstrated a large conjunctival bleb and well controlled intraocular pressure for 7 years but was lost follow up from 7-14 years postoperatively. Bilateral superior corneal ectasia was observed 15 years after the operation. The corneal ectasia may have been a coincidental episode or may have been induced by the bleb compression. However, we suppose that it was a late toxic complication of mitomycin C, which penetrated the corneal stroma during the operation and caused this episode.

CASE REPORT

A 49 year old man received the same operative procedures on both eyes as a result of open angle glaucoma in our clinic in March 1982. The procedure included a limbal based 3 × 5 mm partial thickness rectangular scleral flap centred on the 12 o'clock position and a 1 × 3 mm trapdoor combined with the intraoperative application of 0.2 mg/ml mitomycin C (MMC) soaking for 5 minutes. Before the operation, the best corrected vision was counting fingers 50 cm with -7.0 right eye and 20/100 with -6.0 -1.0 × 180 left eye. Intraocular pressure was 33 mm Hg right eye and 42 mm Hg left eye. Three months after the operation, the best corrected vision was 20/500 with -7.00 right eye and 20/200 with -3.50 left eye. During the 7 year follow up period, intraocular pressures of both eyes were well controlled around 6-12 mm Hg with prominent avascular blebs. He had been lost to follow up for 7-14 postoperative years. On a recent visit in March 1997, he complained of poor vision and saw the horizontal images much clearer than vertical images. Best corrected vision was 20/1000 with -3.25

-1.75 × 38 right eye and 20/200 with +1.75 -5.25 × 166 left eye. Slit lamp examination showed bilateral prominent cystic blebs at the superior limbal conjunctiva. The avascular blebs extended from 15 to 165 degrees, 3.7 mm wide in the right eye, and 45 to 150 degrees, 5.4 mm wide with a bleb invading across the limbus in the left. There was no evidence of a wound leak on Seidel test. Both optic discs were pale with the cup/disc ratio 0.9 right eye and 0.5 left eye. Corneal topography (Eyesys 2000) revealed bilateral ectasia at the superior corneas (Fig 1). The corneal thickness was measured by ultrasonic pachymetry at the corneal centre and 2.5 mm away from the centre in the direction of 45, 90, and 135 degrees. There were unremarkable changes in thickness around the ectatic sites on both corneas.

COMMENT

The mechanism of inducing such a late occurrence of superior corneal ectasia is not clear. It may merely have been a coincidental episode. The clinical findings are not similar to the degeneration, which frequently induces astigmatism, such as Terrien's marginal degeneration, superior pellucid marginal degeneration,¹ senile marginal degeneration, or keratoconus. Several studies have reported the "with the rule" changes in corneal astigmatism in the early postoperative period of trabeculectomy.^{2,3} The changes that were usually under 2.5 dioptres and resolved within 12 weeks were not comparable with our findings. It is possible that a large cystic bleb, which is not uncommon after the trabeculectomy combined with MMC application, compressed the superior cornea, resulting in the changes of the corneal curvature and induced the ectasia. Another possibility is that this episode is directly related to the chronic toxicity of MMC that was applied 15 years ago. Tanihara et al reported prolonged impairment of peripheral corneal epithelial barrier function after the trabeculectomy.⁴ It verified the possibility that MMC can induce a late tissue dysfunction. In our case, MMC might have penetrated the stromal layer from the cutting edge of the scleral flap during the trabeculectomy causing keratocyte suppression, collagen degeneration, and finally corneal ectasia. The two postulated mechanisms mentioned above may act together. Further studies are necessary to prove this.

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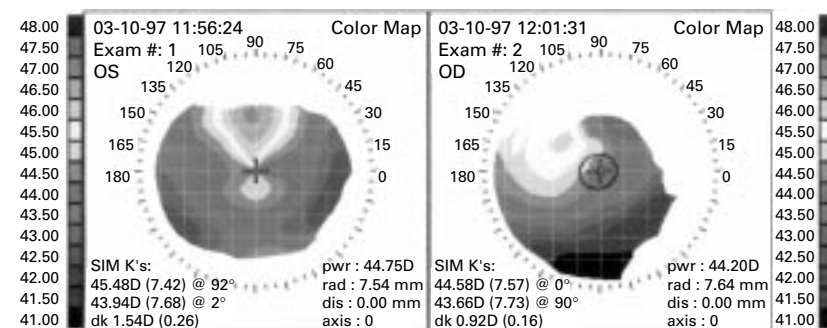


Figure 1 Computerised corneal topography showed bilateral corneal ectasia. (Left) The apex of the ectasia in the left eye located at axis 93°, 2.55 mm away from corneal centre. (Right) The apex of the ectasia in the right eye located at axis 139°, 2.18 mm away from corneal centre.

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Severe proliferative diabetic retinopathy associated with acromegaly

Acromegaly is complicated by diabetes mellitus because of the action of growth hormone (GH) which induces the production of insulin-like growth factor I (IGF-I). IGF-I may contribute to the development of diabetic retinopathy in an autocrine and/or paracrine manner.¹ In patients with acromegaly who demonstrate excessive secretion of GH, the incidence of retinopathy is reportedly very low and severe retinopathy is rare.^{2,3} This report describes a patient with severe proliferative diabetic retinopathy in both eyes associated with acromegaly. We performed pars plana vitrectomy and calculated the levels of angiogenic growth factors (IGF-I and vascular endothelial growth factor, VEGF) in the vitreous of both eyes to learn whether GH and/or IGF-I may influence the progression of diabetic retinopathy in proliferative diabetic retinopathy associated with acromegaly.^{4,5}

CASE REPORT

A 62 year old woman presented with severe proliferative diabetic retinopathy and was admitted to Osaka Kaisei Hospital for a pars plana vitrectomy. The duration of her diabetic mellitus was about 10 years and her blood sugar level was between 140-320 mg/ml. HgA_{1c} was 7.0-7.5%. The patient also had systemic hypertension. Corrected visual acuity was 30 cm/hand movement in the right eye and light perception in her left eye. Remarkable bilateral rubeosis iridis, active fibrovascular membranes, and extensive tractional retinal detachment were observed in both eyes (Fig 1). Physical examination revealed acromegaly in all four limbs (Fig 2A). The patient's serum GH level was 256 ng/ml and her serum IGF-I level was 726 ng/ml. Magnetic resonance imaging revealed the presence of pituitary adenoma (Fig 2B). Based on these findings acromegaly was diagnosed.

Pars plana vitrectomy was performed on both eyes. IGF-I levels in the vitreous obtained at the time of vitrectomy were determined by radioimmunoassay (assay range 0.3-2100 ng/ml) to be 14 ng/ml. VEGF levels in the vitreous were determined by an ELISA system (the lower limit of detection 2 pg/ml) to be 5.6 ng/ml. Trans-sphenoidal resection of the pituitary adenoma was indicated; however, the patient and her family refused to allow us to perform this treatment. After vitrectomy complete retinal attachment was achieved; however, rubeotic glaucoma was a complication in both eyes. Despite additional operations (trabeculectomy and

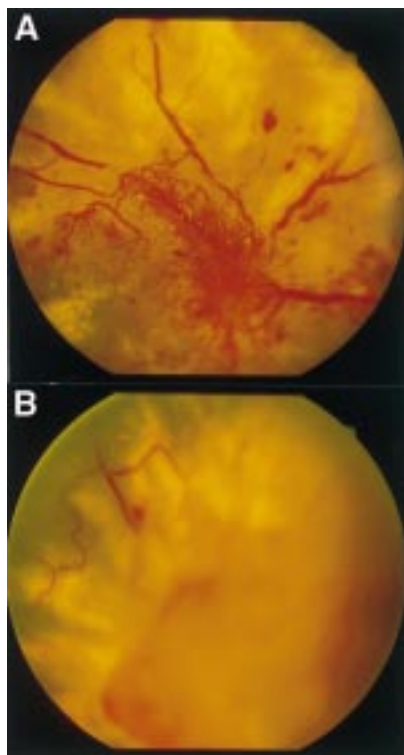


Figure 1 Remarkable neovascularisation in the patient's right eye (A) and large fibrovascular membrane in the left eye (B).

cyclocryocoagulation) intraocular pressure was uncontrollable and light perception was ultimately lost in both eyes.

COMMENT

It is well known that diabetic retinopathy is an occasional complication of diabetic mellitus secondary to acromegaly,^{4,5} although the occurrence of proliferative diabetic retinopathy in association with this disease appears to be rare. But the present case of diabetic retinopathy accompanied by acromegaly was complicated by very active fibrovascular



Figure 2 Hypertrophy of bone and cartilage in the patient's hands (A). Magnetic resonance imaging revealed the presence of pituitary adenoma (B).

proliferation and tractional retinal detachment. Diabetic mellitus secondary to acromegaly is related to the oversecretion of GH. IGF-I, an angiogenic factor, is the mediating molecule that is influenced by GH in the pathogenesis of diabetic retinopathy. However, acromegalic patients who overexpress GH with or without concomitant diabetes do not demonstrate an increased incidence of retinopathy.^{4,5} The influence of the excessive secretion of GH and downstream effector IGF-I on diabetic retinopathy and neovascularisation in acromegalic patients remains unclear, although Smith and associates⁶ recently reported that inhibition of GH can inhibit ischaemia induced retinal neovascularisation in vivo via VEGF.

Other investigators have calculated the vitreous and serum levels of IGF-I in patients with proliferative diabetic retinopathy to be in the range of 5–8 ng/ml and 80–220 ng/ml respectively.^{7–9} In this case the vitreous level of IGF-I was 14 ng/ml and the serum level of IGF-I was 726 ng/ml. Both of these values are higher than in patients with proliferative diabetic retinopathy. This is the first report to calculate the vitreous IGF-I level in an acromegalic patient with severe proliferative diabetic retinopathy. The results suggest that the excessive production of local IGF-I or the breakdown of the blood-ocular barrier (which resulted in the diffusion of serum IGF-I into the vitreous cavity), occurred in this patient and accelerated the progression of the retinopathy. We also found a relatively higher level of VEGF (5.6 ng/ml), a major angiogenic growth factor, in the vitreous of the patient.¹⁰ Some investigators reported that IGF-I accelerates the expression of VEGF in some cell lines.¹¹ Thus, GH may have played a important role in the progression of diabetic retinopathy in combination with IGF-I and VEGF.⁹ Diabetic retinopathy associated with acromegaly should be further studied to examine the correlation among GH, IGF-I, VEGF, and other angiogenic factors.

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Emergency contraception and retinal vein thrombosis

EDITOR,—The article by Vessey *et al*¹ on the ocular complications of oral contraceptives acknowledges the association between oral contraceptives and retinal vein thrombosis. We report a case of a woman with a retinal vein thrombosis affecting the superior retina directly associated with emergency contraception.

CASE REPORT

A 33 year old woman presented to the eye casualty service with a 10 day history of blurred vision in her right eye that had been present on waking the day after she took PC4 emergency contraception prescribed by her general practitioner. She had no significant ophthalmic or medical history.

On examination visual acuity was right eye 6/6, left eye 6/6. She had no afferent pupillary defect, normal anterior segments, and intraocular pressures of right eye 17 mm Hg, left eye 18 mm Hg. Dilated funduscopy identified a right superior hemiretinal vein occlusion (Fig 1). Blood pressure was 130/80, and she had no other identified risk factors for vein occlusions, including normal serum glucose, urea and electrolytes, full blood count, erythrocyte sedimentation rate, a normal clotting



Figure 1 Right fundus photograph demonstrating superior hemiretinal vein occlusion.

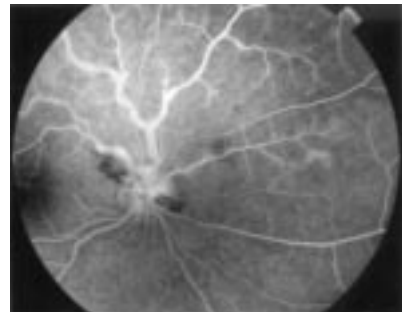


Figure 2 Fluorescein angiogram at 34 seconds demonstrating delayed blood flow with leakage of fluorescein in the affected area.

screen, c-reactive protein, antinuclear antibodies, antithrombin III, activated proteins c and s, and no lupus anticoagulant. A fluorescein angiogram was performed that confirmed the diagnosis (Fig 2). She was advised to take aspirin 75 mg once daily as tolerated. Two months later the fundus appearance had returned to normal.

COMMENT

An increased risk of retinal vein thrombosis has been recognised in the regular use of oral contraceptives and hormone replacement therapy.²⁻⁴ There are no previous reports of such a direct association between the taking of sex hormone preparations and retinal vascular events. PC4 contains 500 µg norgestrel and 50 µg ethinyloestradiol, and two tablets are taken within 72 hours of coitus with a further two 12 hours later. This is four to six times the hormone content of standard strength oral contraceptives in one 24 hour period.⁵ In the absence of any other risk factors, and with the event so closely following medication, it seems reasonable to attribute the retinal vein thrombosis to the contraception. Kirwan *et al*² suggest that a retinal vein thrombosis is a contraindication for further use of the oral contraceptive, but possibly not a contraindication for continued hormone replacement therapy. Whether this event means that patients having a retinal vein thrombosis following emergency contraception should be advised not to take regular oral contraception is difficult to determine. As there are no previous reports the risk cannot be quantified but is likely to be low. In this case the decision has been left up to the individual (after appropriate advice).

A CSM "yellow card" has been submitted for this adverse event.

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Unilateral ptosis due to isolated involvement of the levator muscle in acute orbital myositis

EDITOR.—A 46 year old woman presented with a 3 week history of discomfort, swelling, and drooping of her right upper lid. Recently she had developed rheumatoid arthritis but was otherwise fit and well. On examination her right visual acuity was 6/4 unaided, there was a complete right ptosis with very poor levator function, and mild periorbital swelling (Fig 1). Eye movements were full and the remainder of the ocular examination was normal. A computed tomograph (CT) scan showed isolated



Figure 1 Initial presentation. Note the complete right ptosis and very poor levator function.

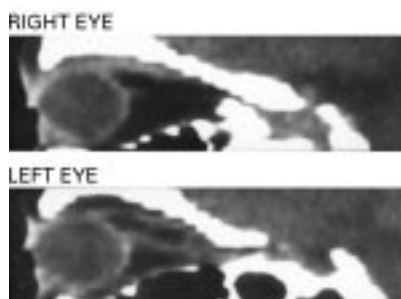


Figure 2 Computed tomograph scan showing enlargement of the right superior rectus/levator complex compared with the left side.

enlargement of the right superior rectus/levator muscle complex (Fig 2).

Three weeks later her symptoms had improved. A 3 mm ptosis remained and the levator function had increased to 7 mm. There was lid lag on downgaze (Fig 3) suggesting both abnormal contraction and relaxation of the levator muscle. The ptosis gradually resolved over the next 4 weeks and the levator function returned to normal. It has not recurred.

COMMENT

Orbital myositis is an idiopathic inflammatory condition that affects the extraocular muscles. It may occur with a number of systemic

conditions including rheumatoid arthritis.¹ Ptosis in orbital myositis is quite common.¹⁻⁴ The ptosis may result from orbital oedema secondary to inflammation or from direct involvement of the levator muscle with myositis. It is mentioned as a sign in several reports but there are no examination details such as the levator function, the degree of ptosis, or lid lag on downgaze to help establish the cause.¹⁻³ Similarly, enlargement of the superior rectus/levator complex on CT, magnetic resonance imaging, or ultrasound is noted in some cases without any details of a ptosis.^{2,3,5}

Our case had isolated involvement of the levator muscle. To our knowledge this has not been reported before. Isolated involvement of other single extraocular muscles is common. It occurred in 68% of the 75 cases reported by Siatkowski *et al*, usually the lateral or medial rectus.⁵ Isolated involvement of the oblique muscles also occurs.⁶

Siatkowski *et al* reviewed 75 patients with orbital myositis.⁵ They found a changing pattern of function in the extraocular muscles affected over the course of the disease. In the first 10 days the extraocular muscle function was normal. At 11-14 days there was a paretic phase. At 17-24 days there was a restrictive or mixed phase which partially or completely resolves. Our patient with isolated myositis of the levator muscle had a very similar course with the levator being almost completely paretic at the first visit. Three weeks later there was a mixed pattern of paresis and restriction with reduced levator function and



Figure 3 Three weeks later. Note the spontaneous improvement, a 3 mm right ptosis remains, the levator function is 7 mm and there is lid lag on downgaze.

lid lag on down gaze. A month after that it had completely resolved.

Orbital myositis should be considered in the differential diagnosis of acquired ptosis even if extraocular movements are full. We hope that the features of the ptosis will be reported in other cases to find out if the ptosis is the result of oedema or if the levator is affected by myositis.

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Immunological investigations following an adverse reaction to oxybuprocaine eye drops

EDITOR.—In 1990, a case of severe bradycardia following exposure to oxybuprocaine eye drops was reported.¹ No mechanism was identified but the reaction was assumed to be anaphylactic or secondary to rapid anaesthetic absorption. We report a similar case of a life threatening reaction.

CASE REPORT

A 29 year old man attended his optician for routine tonometry. Following instillation of 0.4% oxybuprocaine eye drops (Benoxinate, Chauvin Pharmaceuticals) his vision "went white" within 1 minute with a subjective sensation of throat swelling, followed by collapse and loss of consciousness. His wife, a medical doctor, noted no respiration and a carotid pulse of 12 beats/min. There was some swelling of the soft tissues of the neck, but no lip oedema. Cardiopulmonary resuscitation was performed, and adrenaline 300 µg was administered intramuscularly by the patient's sister who has peanut allergy. There was a rapid return of cardiac output and he made a full recovery, with no abnormalities found on examination in the emergency room. He was subsequently found to have acute open angle glaucoma caused by pigment dispersion syndrome. Intraocular pressures were 56 mm Hg bilaterally at diagnosis. The condition was successfully treated with topical Timoptol and dorzolamide.

He was atopic, and wheezed when exposed to cats. There was no other history of allergic disease, and no significant medical history. His brother has asthma. He works as a physician, and regularly administers local anaesthetic agents, principally lignocaine (lidocaine), but had no symptoms while doing this. He had received prilocaine for dental procedures with no ill effect. Immunological investigations revealed a mildly elevated total IgE of

590 kU/l (normal <120 kU/l) with specific IgE against cat (grade III), house dust mite (grade IV), and grass pollen (grade II) confirming his atopic tendency. RAST tests to local anaesthetic reagents are not commercially available.

While monitored in the intensive therapy unit, the patient underwent skin prick and intradermal tests to a variety of local anaesthetic agents including oxybuprocaine eye drops at dilutions of 1/1000, 1/100, 1/10, then neat with no local or systemic reactions. Both ester and amide agents were used. The reaction to oxybuprocaine was assumed to be idiosyncratic, and not a classic type I hypersensitivity reaction. He was advised never to use oxybuprocaine again, but contact with other local anaesthetics was deemed safe.

COMMENT

This is the second reported case of life threatening reaction to oxybuprocaine. Up to February 1996, the Committee on Safety of Medicines in the UK had received no reports of anaphylactic reactions to this agent (CSM, personal communication). No reports of anaphylaxis have been made to the manufacturer (MM Martin, Chauvin Pharmaceuticals, personal communication). Of note, this preparation is made up in sterile water, without preservatives. Previously reported reactions include seizure.² In a series of 12 493 drug applications in ophthalmic clinics in the USA, eight cases (0.208%) of adverse reactions were found.³ Five of these were to oxybuprocaine but all as oxybuprocaine/fluorescein solution (Fluress), including increased intraocular pressure (one), stinging (one), dull ache with redness and swelling (two), and one case of dizziness and coldness with collapse. This subject (patient 12396) may have had a similar reaction to our patient.

Local anaesthetics can provoke adverse idiosyncratic, cardiovascular, and allergic reactions.⁴ Local anaesthetics can be divided on an immunochemical basis into two groups: group I, the benzoic acid esters include oxybuprocaine; and group II, the amides include lignocaine and prilocaine. There is generally little cross reactivity between group I and II drugs, but cross reactivity within groups is recognised.⁵

This case illustrates the importance of recognising and treating cardiovascular collapse following administration of a drug, and the careful immunological follow up investigation that is required.

No proprietary interests.

The kind assistance of Miss Christine Oates, drug information pharmacist at the John Radcliffe Hospital, is gratefully acknowledged.

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Utility of Fungiflora Y stain in rapid diagnosis of *Acanthamoeba* keratitis

EDITOR.—*Acanthamoeba* keratitis is a serious and often misdiagnosed corneal infection that can lead to severe loss of vision. Signs and symptoms of *Acanthamoeba* keratitis may also occur with bacterial, viral, or other fungal corneal disease, complicating the diagnosis of early stage *Acanthamoeba* keratitis. Since advanced *Acanthamoeba* keratitis is difficult to treat, early and accurate diagnosis is imperative for treatment. Therefore, the easy and rapid method to detect the pathogens is needed.

Recently, Fungiflora Y staining solution has been developed to detect fungi.¹ This staining solution, like calcofluor white,² has a specific affinity for chitin and cellulose which are components of fungal cell wall.¹ The endocysts of *Acanthamoeba* also contain cellulose³ and has been ascertained to be stained with Fungiflora Y.⁴ However, the clinical utility of Fungiflora Y in the diagnosis of *Acanthamoeba* keratitis has not yet been reported.

We report a case of severe *Acanthamoeba* keratitis diagnosed with the help of Fungiflora Y staining. We also discuss the possible clinical use of Fungiflora Y staining to diagnose other cases of *Acanthamoeba* keratitis.

CASE REPORTS

A 55 year old woman, who had worn soft contact lenses for 1 month, presented in February 1994 with a 2 week history of foreign body sensation and photophobia in the left eye. These symptoms gradually became worse and she was diagnosed with herpes simplex keratitis in another clinic because of a pseudodendritic corneal epithelial lesion. Despite topical applications of antibiotics, corticosteroids, and aciclovir ointment four times a day, no improvement was observed and the patient experienced severe eye pain and progressive decrease in vision in the left eye.

When the patient first visited the department of ophthalmology at Osaka University Medical School in early April 1994, her visual acuity was 0.1 in the left eye and slit lamp examination revealed stromal disciform keratitis with a central ulceration (Fig 1). Her right eye was normal.

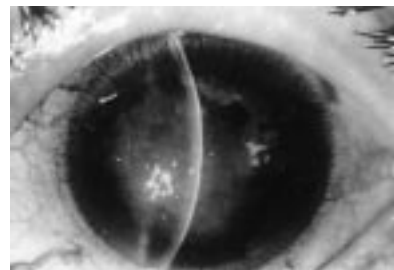


Figure 1 Slit lamp examination revealed stromal disciform keratitis with a central ulceration.

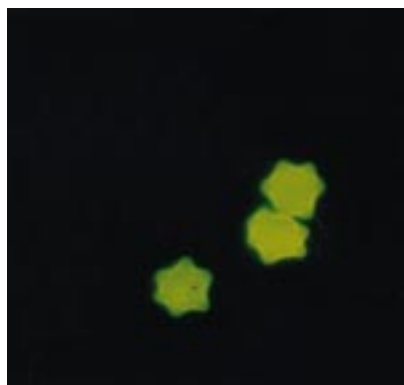


Figure 2 *Acanthamoeba* cysts stained with Fungiflora Y ($\times 400$). The staining procedure was simple and rapid and required no complex skills. *Acanthamoeba* cysts were readily identified under low magnification fluorescence microscopy.

We obtained a deep corneal scraping for cytopathological examination and culturing. Light microscopic examination failed to disclose any Gram and Giemsa stained organisms. Furthermore, with haematoxylin and eosin staining, no pathogen could be found. However, Fungiflora Y staining to detect fungus unexpectedly demonstrated fluorescent *Acanthamoeba* cysts, which in turn led to the diagnosis of *Acanthamoeba* keratitis. The indication of the steps and time using the Fungiflora Y staining kit (Biomate Co, Ltd, Tokyo, Japan) was as follows. The scrape was transferred onto slide glass, and a drop of the counterstaining reagent (A solution) was added. After 2 minutes, the slide was rinsed with running water and stained with the staining reagent (B solution) for 5 minutes. The slide was rinsed under running water and observed under a fluorescent microscope at a wavelength 405 nm. *Acanthamoeba* cysts appeared as clear yellowish-green fluorescence and were easily identified (Fig 2).

Subsequently, cultures of the corneal scrapings were found to be positive for *Acanthamoeba* cysts and trophozoites. When mitochondrial DNA was digested by restriction enzyme it was identified as *Acanthamoeba* Ma.⁵

The appropriate treatment agents and doses were determined by in vitro sensitivity testing, which showed that *Acanthamoeba* trophozoites were sensitive to miconazole and pimaricin and cysts were sensitive to pimaricin only.

The patient received the following treatment for 1 month: 0.1% miconazole eye drops six times a day, pimaricin eye drops four times a day, and ofloxacin eye drops four times a day, in addition to a daily 400 mg intravenous dose of miconazole for 4 weeks. Eye pain disappeared and the lesions improved slowly. Central stromal infiltration gradually became less dense, but the occurrence of stromal scarring led to the need for penetrating keratoplasty in December 1995. Visual acuity returned to 0.5 in the left eye at 2 years post-operatively, and she has been free of recurrent disease.

COMMENT

In this case, no pathogens were detected on examination of corneal tissue samples, prepared with conventional stains even after repeated evaluation. To identify fungi, we examined smears of corneal scrapings stained with Fungiflora Y and unexpectedly found the *Acanthamoeba* cysts stained fluorescently. This confirmation of our diagnosis of *Acanthamoeba* keratitis allowed us to initiate the appropriate treatment with confidence.

To our knowledge, this is the first clinical case report of *Acanthamoeba* keratitis diagnosed with Fungiflora Y stain.

The procedure for staining with Fungiflora Y is simple and rapid, requiring no experience and complex skill,⁴ and in our case *Acanthamoeba* cysts were easily identified under low magnification fluorescence microscope. Furthermore, when we observed this slide

after 3 months, *Acanthamoeba* cysts were well stained fluorescently and easily identified. Consequently, fluorescent intensity of Fungiflora Y stain is stable and fluorescent attenuation is much smaller than the FITC labelled antibody or other fluorescence staining methods. The results in this case indicate that the staining with Fungiflora Y may be a useful additional technique for identifying *Acanthamoeba* cysts in keratitis specimens. Further evaluation is required to establish how this technique compares with the other available techniques.

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CORRESPONDENCE

Ophthalmic medical assistants

EDITOR.—It was with grave disappointment that I read the commentary in January's *BJO*.¹

Webber and Jeffrey painted the picture of the ophthalmic medical assistant being the panacea for all ills in the ophthalmic world. The impression is given that the role had to be created because of the restrictive practices, rules, and working patterns of ophthalmic nurses.

It is very sad that this must have been the situation in their unit.

Let me take the opportunity to point out that up and down the country ophthalmic nurses are undertaking all the roles they describe and that, in collaboration with forward thinking nurse management, such roles can be created within the auspices of the nursing profession without having to invent a new profession. As a charge nurse, senior nurse, and nurse manager I have spent a lot of energy and time in collaboration with consultants who recognise the advantages of enhancing, developing, and creating roles for ophthalmic nurses for the improvement of service and patient care. These roles too have increased efficiency and built upon existing teamwork as well as the vast knowledge base that ophthalmic nurses already have. Attendance at any UK ophthalmic nursing conference would reveal exactly that.

It may also be of interest to note that if it is appropriate for service development, nurse practitioners can be appointed to consultant firms or service teams. There are many examples of collaboration work with ophthalmic nurses and ophthalmologists where trust and mutual respect for each other's contribution exists.

It is a shame that some units are not able to achieve this within the framework of the existing professions. However, I would be more than willing to give examples of where it has been done.

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1 Webber SK, Jeffrey MN. Ophthalmic medical assistants (commentary). *Br J Ophthalmol* 1999;83:4.

EDITOR.—I was interested to read Webber and Jeffrey's elaboration of the role of the ophthalmic medical assistant in the *BJO*¹ and their translation of this role which is, as they state, quite prominent in the United States, into the UK health setting. The role which they describe seems to be suspiciously like that undertaken by many ophthalmic nurses in other centres in the United Kingdom. However, ophthalmic nurses do not merely carry out delegated "tasks", but carry out holistic care within a nursing framework, having undertaken up to 4 years' training and having proved their competence. It is true that although ophthalmic nurses understand the principles of refraction and motility testing, they rarely become more involved in these issues but tend to leave them to those professionals (optometrists and orthoptists) who have also had 3 or 4 years' training and are registered practitioners.

Nurses do, however, undertake their own outpatient clinics, run accident and emergency services, act as first assistant in theatre, assess

patients for surgery, and discharge them afterwards; they also work with individual consultants in some areas. They also have a system of professional accountability and regulation which would appear completely lacking in the OMA role. Perhaps an incentive in the development of this role is the perception of the OMA as belonging somehow to his or her consultant (...one's OMA) instead of someone in a complementary but not owned profession, and the possibility of enhanced medical power and control.

There appears to be a subtle, underlying criticism of ophthalmic nurses within this commentary; "...communication can fail even in the best of teams...lead to missed or inappropriate admissions, suboptimal theatre timing, and other problems". Later, the authors suggest that "less operating time is wasted through inappropriate theatre lists". This also raises the possibility of the OMA as a "fall guy" for medical failures of communication. Ultimately, the person responsible for inappropriate theatre lists and wasted theatre time has to be the surgeon. This responsibility is for much more than just the act of surgery itself. The suggestion that the introduction of the OMA role will stop medical muddles and therefore increase the number of patients seen and operated on is purely speculative.

More obvious problems with this role are easy to identify. Who will undertake the work when the OMA is sick or on holiday? What will they do if "their" consultant is away. There seems little chance of their acceptance by nursing staff and lack of communication between staff groups will lead to more failures in patient care. There is no career structure for OMAs no recognised training, and their standards will be those of "their" particular consultant which are technical/medical rather than holistic. The authors suggest that they may cost the same as nurses but they will not possess any of the flexibility of that workforce.

This seems to herald the development of an unaccountable, unregulated personal servant with no formal training. Nurses can and do undertake these roles from a basis of accountable, registered, educated practice. That there is a need felt to employ informally trained, unqualified personnel in this area suggests a failure in communication and in the nursing and medical management of a developing service.

JANET MARSDEN

Senior lecturer, the Manchester Metropolitan University and chair, Royal College of Nursing, Ophthalmic Nursing Forum

1 Webber SK, Jeffrey MN. Ophthalmic medical assistants (commentary). *Br J Ophthalmol* 1999;83:4.

EDITOR.—I read the commentary by Webber and Jeffrey¹ with interest, but some incredulity.

There is little doubt that in a system such as that which obtains in the United States, where every service performed under the individual physician's overall supervision may be billed, the ophthalmic medical assistant may well have a useful role. Indeed, I have recently heard the anatomically implausible term "physician extender" used by an American colleague to describe this type of relationship between ophthalmologist and assistant, in particular for staffing of distant outreach clinics.

Could such a system take root in the United Kingdom? I rather doubt it.

Firstly, where will they come from? It seems generally recognised that the shrinking supply of enthusiastic school leavers will have a serious effect on the recruitment of nurses,

whatever the health secretary, Frank Dobson, may try to do to redress this, and this job hardly opens up the professional possibilities to which a nursing degree may lead.

Secondly, will the public welcome a "practitioner" who is not a nurse, a doctor, or yet a member of one of the highly trained members of the professions supplementary to medicine? The cheerful assurance that the OMA "is not bound by restrictive rules and regulations" gives me, for one, considerable disquiet. Will the trust extend indemnity cover to an unqualified non-professional?

Thirdly, will they stay? The career structure appears to have no prospect of promotion, so I suspect that anyone with a modicum of talent and intelligence will stay in the post for as short a time as possible before seeking a greater degree of challenge elsewhere, leaving behind those who have risen to their greatest achievable height. We are all familiar with this syndrome in the NHS managerial structure.

Finally, my colleagues in our orthoptic and medical illustration departments are rather distressed to discover that they don't exist, but have promised to be very brave and to apply for the very first OMA jobs as soon as they come along.

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1 Webber SK, Jeffrey MN. Ophthalmic medical assistants (commentary). *Br J Ophthalmol* 1999;83:4.

Reply

EDITOR.—John Lee asks, from where will the OMAs come? Fielder *et al*, in their commentary in this issue (p 512), assume a very basic level of training for OMAs. This is not the case. At the moment all six OMAs in Portsmouth are highly experienced and trained ophthalmic nurses. We made the point in our commentary that they need not necessarily be from the nursing profession. Until such time (if ever) that a planned training scheme for OMAs is set up in the United Kingdom we would suggest that ophthalmic nurses are a good source for recruitment. While we share Mr Lee's concerns about the shortage of trained nurses we would remind him that these members of staff are not being removed from the workplace.

Fielder *et al* dismiss any comparison between the United Kingdom and the United States. The average patient in the USA is a much more informed and demanding individual than his/her counterpart in the UK. Do they really think that poorly qualified, untrained individuals would be permitted to work with such a demanding and litigious population? There are many people working within the NHS who are not highly qualified and yet who are accepted by the public. They carry out a limited range of important and worthwhile tasks. All trusts will indemnify people who have been trained to carry out specific tasks.

A career structure could exist for OMAs. Mr Lee implies that anybody with ambition would want to leave their job. How is this different from any other healthcare professional? Not every person employed in patient care wishes to climb the NHS managerial ladder. As evidence of job satisfaction, we would inform him that all OMAs appointed in Portsmouth since the inception of the role in 1992 are still in post.

Fielder *et al* claim that a group of OMAs, if given the opportunity, would form or join a professional organisation with rules and

regulations. They point out that the first task of such a body would "inevitably" be to break down the close working relationship that an OMA and consultant may have. Their mistake is to assume that such a body would de facto be obstructive. This, of course, is a possibility, but also one of the reasons why we have introduced OMAs. The very essence of the role of the OMA is to provide continuity of care for patients. This is simply not the case in the majority of nursing environments.

The work of the OMAs in our unit focuses on the surgical aspects of ophthalmology, but this needn't be so in all units. Surgical waiting lists in ophthalmology are unacceptably long and they have become a government priority. Over 70% of all surgical procedures carried out in ophthalmology are cataract extractions. The service needs to be streamlined to be cost effective and to enable the maximum number of people to have surgery in as short a time as possible, while maintaining high standards of patient care. Fielder *et al* admit that the roles of the "team members [should] vary according to local needs and enthusiasm and prejudices". This is exactly what the OMA post is designed to do, responding to a need in a department with a heavy surgical workload. They are rather dismissive in saying that the OMA system is inflexible and does not adapt speedily enough to meet the needs of modern ophthalmology. We wonder if any of them have ever worked with an OMA? The status quo is not an option and rather than dismissing the whole process, constructive criticism as to how it might be improved would be a more enlightened approach.

It is of interest that Lee and Fielder are highly subspecialised ophthalmologists who do not carry out cataract surgery. We wonder how long it has been since either of these doctors have worked in a busy district general hospital? Ophthalmology will have increased demands made upon it by a more demanding public. At present we carry out only a third of the number of cataracts per 1000 population compared with the USA. The elderly population is increasing. It is our firm belief that a team of experienced OMAs can help with the ever increasing demands for efficiency and quality of care in ophthalmology.

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NOTICES

Blindness in children

The latest issue of the *Community Eye Health* (no 28) discusses community based rehabilitation in developing countries. For further information please contact *Community Eye Health*, International Centre for Eye Health, Institute of Ophthalmology, 11-43 Bath Street, London EC1V 9EL. (Tel: (+44) 171 608 6910; fax: (+44) 171 250 3207; email: eyeresource@ucl.ac.uk) Annual subscription £25. Free to workers in developing countries.

Residents' Foreign Exchange Programme

Any resident interested in spending a period of up to one month in departments of ophthalmology in the Netherlands, Finland, Ireland, Germany, Denmark, France, Austria, or Portugal should apply to: Mr Robert Acheson, Secretary of the Foreign Exchange Committee, European Board of Ophthalmology, Institute of Ophthalmology, University College Dublin, 60 Eccles Street, Dublin 7, Ireland.

ARVO 1999 annual meeting

The 1999 annual meeting of the Association for Research in Vision and Ophthalmology will take place on 9-14 May 1999 in Fort Lauderdale Convention Center, Fort Lauderdale, Florida. Further details: ARVO, 9650 Rockville Pike, Bethesda, MD 20814-3998, USA. (Tel: (301) 571-1844; fax: (301) 571-8311.)

12th Annual Meeting of German Ophthalmic Surgeons

The 12th annual meeting of German Ophthalmic Surgeons will be held on 10-13 June 1999 at the Meistersingerhalle, Nürnberg, Germany. Further details: MCN Medizinische Congress-Organisation Nürnberg GmbH, Weilandstrasse 6, D-90419 Nürnberg, Germany. (Tel: ++49-911-3931621; fax: ++49-911-3931620; email: doerflinger@mcn-nuernberg.de)

Royal National Institute for the Blind

A national conference of the Royal National Institute for the Blind will be held on 22-23 June 1999 at the Cedar Court Hotel, Wakefield. Further details: Kristene Wilde, Conference administrator, Royal National Institute for the Blind, RNIB Education Centre: North, Grosvenor House, Grosvenor Road, Leeds LS6 2DZ (tel: 0113-274 8855; fax: 0113-274 8800).

XII Congress European Society of Ophthalmology

The XII Congress European Society of Ophthalmology will be held in Stockholm, Sweden on 27 June-1 July 1999. Further details: Congress (Sweden) AB, PO Box 5819, S-114 86 Stockholm, Sweden. (Tel: +46 8 459 66 00; fax: +46 8 661 91 25; email: soe@congrex.se; <http://www.congrex.com/soe/>)

Vision '99: International Conference on Low Vision and Vision Rehabilitation

The International Conference on Low Vision and Vision Rehabilitation will be held on 12-16 July 1999 at the Waldorf-Astoria Hotel, New York City, New York. Further details: Lighthouse International, 111 East 59th Street, New York, NY 10022-1202, USA. (Tel: (212) 821-9482; fax: (212) 821-9705; email: vision_99@lighthouse.org)

4th Meeting of the European Neuro-Ophthalmology Society

The 4th meeting of the European Neuro-Ophthalmology Society will be held on 2 August-2 September 1999 in Jerusalem, Israel. Further details: Secretariat, 4th Meeting of the European Neuro-Ophthalmology Society, PO Box 50006, Tel Aviv, 61500, Israel. (Tel: 972-3-514000; fax: 972-3-5175674/972-3-5140077; email: Eunos99@kenes.com)

International Agency for the Prevention of Blindness

The sixth general assembly of the International Agency for the Prevention of Blindness will be held on 5-6 September 1999 at the Conference Centre, Beijing Friendship Hotel, Beijing, People's Republic of China. The theme is "The right to sight". Further details: IAPB Secretariat, LV Prasad Eye Institute, LV Prasad Marg, Banjara Hills, Hyderabad 500 034, India. (Tel: 091-40-215389; fax: 091-40-248271; email: IAPB@lvpeye.stph.net)

Ophthalmological Clinic, University of Creteil

An international symposium on the macula will be held on 1-2 October 1999 at the Ophthalmological Clinic, University of Creteil. Further details: Professor G Soubrane, Chef de Service, Clinique Ophtalmologique Universitaire de Creteil, Centre Hospitalier Intercommunal, 40 Avenue de Verdun, 94010 Creteil, France. Fax: 01 45 17 52 27.

Jules François Prize

The 2000 Jules François Prize of \$100 000 for scientific research in ophthalmology will be awarded to a young scientist who has made an important contribution to ophthalmology. All topics in the field of fundamental and/or clinical research in ophthalmology will be considered. The application should be sent jointly with a curriculum vitae, the list of all publications, and three copies of the candidate's 10 most relevant publications to Jules François Foundation Secretary, Professor Dr M Hanssens, Dienst Oogheekunde, de Pintelaan 185, B-9000 Gent, Belgium. Deadline for applications 31 December 1999.

DR-2000, International Forum on Diabetic Retinopathy

The International Forum on Diabetic Retinopathy will take place on 7-9 September 2000 at the Palazzo Reale, Naples, Italy. Further details: Francesco Bandello, Congress Secretariat, MGR Congressi, Via Servio Tullio, 4, 20123 Milano, Italy (tel: 39 02 430071; fax: 39 02 48008471; email: dr2000@mgr.it).