LETTERS TO THE EDITOR

Spontaneous dissection of internal carotid artery presenting as isolated posterior ischaemic optic neuropathy

EDITOR—Posterior ischaemic optic neuropathy (PION), which is a diagnosis of exclusion, has been associated with a variety of vascular diseases such as giant cell arteritis, systemic lupus erythematosus, atherosclerosis, polyarteritis nodosa, hypotension, and rarely acute occlusion of the internal carotid artery.[1] We present a patient with monocular PION resulting from ipsilateral internal carotid artery dissection.

CASE REPORT

A 55-year-old man presented to our neuro-ophthalmology unit with blindness in the right eye, which he had noticed on awakening 3 days ago. His past medical history showed a 5 year history of hypertension and 3 year history of diabetes mellitus with poor control. He had a 1 month history of right frontal headache occasionally associated with blurred vision in the right eye. On examination, visual acuity was no light perception in the right eye, 20/20 in the left eye, and a right afferent pupillary defect was present. Extraocular motility was full in each eye. The slit-lamp examination was unremarkable. Dilated fundus examination showed a depigmentation patch in the pupillomacular bundle in the right eye, normal retinal vessels, and healthy appearing optic discs on both eyes. Fluorescein angiogram examination showed normal circulation time of retinal vessels in both eyes (Fig 1). The Humphrey visual field examination of the left eye was normal. Orbit computed tomography did not show a swollen optic nerve or any abnormality. Right carotid angiography revealed a string sign suggestive of internal carotid artery dissection 3 cm above the bifurcation (Fig 2). Anticoagulant treatment was started but was unsuccessful. Evidence of retinal ischaemia was not seen in the follow up period although by 6 weeks the optic disc had been pale. One month after the episode, he developed proptosis, ocular motility limitation, and conjunctival congeston in the right eye. There was a subjective bruit in the right retrobulbar area.

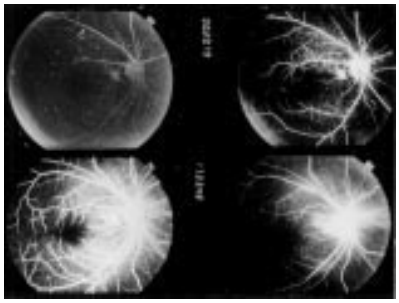

Figure 1 Fluorescein angiogram of right eye circulation at the time of vision loss. The circulation of the retinal vessels is normal.

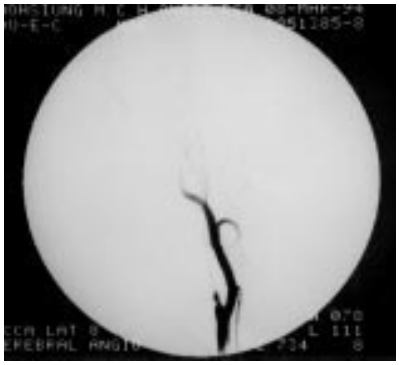

Figure 2 Cerebral angiography of right carotid circulation. There is a string sign in the extracranial internal carotid artery 3 cm above the bifurcation indicating dissection.

Repeated carotid angiography showed a dural-cavernous arteriovenous shunt in the right cavernous sinus, which was caused by external carotid arteries of both side. Neuroradiological intervention with embolisation was done. Four weeks later, the ocular symptoms disappeared but he is still blind in the right eye.

COMMENT

The reported ophthalmic manifestations of carotid dissection include postganglionic oculosympathetic paralysis,[2 3] transient monocular blindness,[2 3] homonymous visual field defect,[3] ocular ischaemic syndrome,[4] 6th nerve palsy,[5] and transient cilio-retinal artery occlusion.[6] Internal carotid artery dissection with subsequent extension of the dissection, or attendant thrombus across into the ipsilateral ophthalmic artery or embolism explains the patients with central retinal artery occlusion reported by Newman *et al*.[7] The only case with PION caused by traumatic carotid artery dissection was reported by Rivkin *et al*. They postulated that the occlusion of internal carotid perforators to the intracranial portion of the optic nerve with preservation of the circulation subserving the anterior optic nerve and retina explained the mechanism of monocular sudden blindness with normal looking fundi in their patient.[8]

As our patient had a normal ocular fluorescein angiogram, normal extraocular motility, and normal optic nerve shown by computed tomography, we believed the circulation of the ophthalmic artery was normal in the initial stage of vision loss. The lesion site should be more proximal to branches of the internal carotid artery. Unfortunately, we cannot prove this by the cerebral arteriogram.

The optic nerve head receives its blood supply from the central retinal artery and the posterior ciliary artery, both arteries are branches of the ophthalmic artery. The intraorbital and intracanalicular portions of the optic nerve also receive their blood supply from the ophthalmic artery via pial capillaries.[9] The intracranial portion of the optic nerve, however, derives most of its blood supply from anterior cerebral, anterior communicating branches, and internal carotid artery perforators.[10] Infarction of this portion of the optic nerve may cause monocular blindness yet preserve the ophthalmic, retinal, and ciliary circulation, as shown in our patient. We are not sure whether the subsequent development of ipsilateral dural-cavernous fistula is related to the occlusion of internal carotid perforators to the intracranial portion of the optic nerve or just a coincident episode.

PION causes no particular changes in the optic disc at the initial stage. It may be accompanied by retrobulbar pain, making it hard to differentiate from retrobulbar optic neuritis. Isayama *et al* had reported 14 cases of PION and proposed the detail criteria in clinical diagnosis of PION.[11] Most of their cases were over 50 years of age and always associated with systemic disease such as hypertension, diabetes mellitus, hyperlipidaemia, and hypotension, etc. Demonstration of abnormal haemodynamics in the posterior portion of the optic nerve by carotid angiography and fluorescein fundus angiography may help us establish the diagnosis.

We think our patient provides another example of spontaneous internal carotid artery dissection presenting as isolated PION. As the visual field in the left eye and fluorescein angiography in both eyes were normal, the probable mechanism was occlusion of the ipsilateral internal carotid perforators to the intracranial portion of the optic nerve with preservation of the circulation of ophthalmic artery subsurving the anterior optic nerve and retina.

RONG-KUNG TSAI
CHUN-YIH SUN
Department of Ophthalmology, School of Medicine, Kaohsiung Medical College, Kaohsiung, Taiwan

Correspondence to: Rong-Kung Tsai, MD, Department of Ophthalmology, School of Medicine, Kaohsiung Medical College, 100 Su-Chuen 1st Road, 807 Kaohsiung, Taiwan.

Accepted for publication: 12 February 1997

1 Kupersmith MJ, Berenstein A. *Neuro-vascular neuro-ophthalmology*. New York: Springer-Verlag, 1993:232-3.
2 Mokri B, Sundt TM, Houser OW, Piepgras DG. Spontaneous dissection of the cervical internal carotid artery. *Ann Neurol* 1986;**19**:126-38.
3 Fisher CM, Ojemann RG, Roberson GH. Spontaneous dissection of cervicocerebral arteries. *Can J Neurol Sci* 1978;**5**:9-19.
4 Duker JS, Belmont JB. Ocular ischemic syndrome secondary to carotid artery dissection. *Am J Ophthalmol* 1988;**106**:750-2.
5 Maitland CG, Black JL, Smith WA. Abducens nerve palsy due to spontaneous dissection of the internal carotid artery. *Arch Neurol* 1983;**40**: 448-9.
6 Camuzet F, Mathis A, Grandperret P, Bec P. Dissecting aneurysm of the internal carotid artery with ophthalmological manifestations. *Rev Otoneuroophthalmol* 1984;**56**:27-9.
7 Newman NJ, Kline LB, Liefer D, Lessell S. Ocular stroke and carotid artery dissection. *Neurology* 1989;**39**:1462-4.
8 Rivkin MJ, Hedges III TR, Logigian EL. Carotid artery dissection presenting as posterior ischemic optic neuropathy. *Neurology* 1990;**40**: 1469.
9 Hayreh SS. Blood supply of the optic nerve head and its role in optic atrophy, glaucoma, and oedema of the optic disc. *Br J Ophthalmol* 1969; **53**:721-48.
10 Isayama Y, Hiramatsu K, Asakura S, Takahashi T. Posterior ischemic optic neuropathy. I. Blood supply of the optic nerve. *Ophthalmologica* 1983; **186**:197-203.
11 Isayama Y, Takahashi T, Inoue M, Jimura T. Posterior ischemic optic neuropathy. III. Clinical diagnosis. *Ophthalmologica* 1983;**187**:141-7.

Posterior scleritis with optic perineuritis and internal ophthalmoplegia

EDITOR,—Depending on the site of the inflammatory process within the scleral or episcleral tissue, scleritis can be identified as anterior scleritis, posterior scleritis or episcleritis.[1 2] Diagnosing of anterior scleritis and episcleritis is much easier than diagnosing posterior scleritis because lesions can be more easily observed in the first two conditions. Posterior scleritis is probably one of the most underdiagnosed conditions in ophthalmology.[1 2] Recent development in magnetic resonance imaging (MRI) technology is considered useful for diagnosing posterior scleritis.

CASE REPORT

A 40-year-old woman was first seen by her ophthalmologist on 8 December 1995, when she complained of left ocular pain. At this time, her visual acuity was 20/20 with correction in each eye. By 11 December 1995, the left visual acuity, with correction, was decreased to 20/200, and chemosis and optic perineuritis in the left eye were observed. She was referred to our clinic on 21 December 1995. On admission her visual acuity was 20/20 in the right eye and 20/400 in the left eye, with correction. No chemosis was detected on the left eye at this time. Abnormalities of extraocular movements were not observed. Funduscopy showed elevated, hyperaemic disc with a splinter haemorrhage in the left eye (Fig 1) and a normal optic disc in the right eye. Visual fields were normal on the right and constricted on the left. The left pupil was dilated and fixed (Fig 1). Instillation of 0.125% pilocarpine resulted in prompt constriction of the left pupil, indicating denervation supersensitivity (Fig 1). Fundus fluorescein angiography showed persistent dye leakage from the left disc and choroidal filling defects in the upper temporal area. Blood tests showed an erythrocyte sedimentation rate of 29 mm in the first hour, a white blood cell count of 5.9×10^9/l, an angiotensin converting enzyme level of 6.0 IU/l (normal, 8.3 to 21.4 IU/l), a lysozyme level of 5.3 μg/ml (normal, 5.0 to 10.2 μg/ml), *Treponema pallidum* hemagglutination assay and fluorescent treponemal antibody negativity, antinuclear antibodies negativity, and rheumatoid factor negativity. Virus titres for mumps, varicella zoster, herpes simplex, cytomegalovirus, rubeola, and rubella were negative. Chest and sinus *x* ray findings were normal. Because the manifestation of optic perineuritis and internal ophthalmoplegia associated with ocular pain in this patient suggested inflammation in the posterior pole of the globe around the optic nerve, ultrasonography, computed tomography, and MRI of the left orbit using a surface coil were carried out. Ultrasonography and computed tomography were unremarkable. On the other hand, T2 weighted images of MRI showed a high signal intensity area around the posterior sclera and the adjacent optic nerve sheath (Fig 2). This area was enhanced in gadolinium-DTPA T1 weighted images using a fat suppression technique (Fig 2). The patient was given methylpredonisolone (1 g per day for 3 days) intravenously from 26 December 1995 followed by predonisone orally (30 mg per day for 14 days). Her left visual acuity rapidly improved and reached 20/20 with correction by 7 January 1996. The optic disc oedema also rapidly improved. The high signal intensity area in T2 weighted images and the enhancement effects in gadolinium-DTPA T1 weighted images disappeared by 5 February 1996. Up to about 3 months after the first examination, no changes in the internal ophthalmoplegia had been detected.

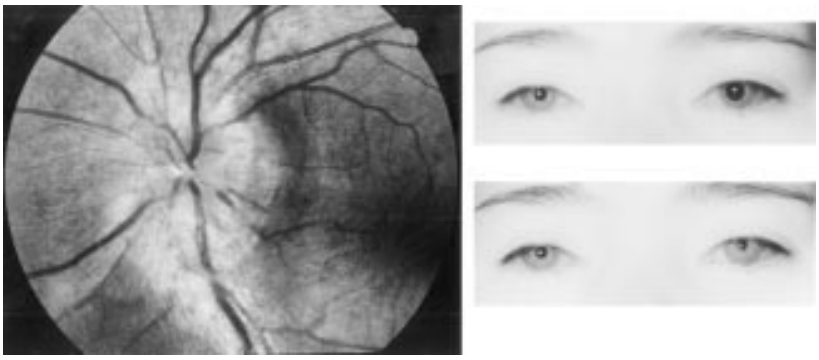

Figure 1 A fundus photograph of the left eye showing optic disc oedema. Infrared photographs taken before (upper panel) and after (lower panel) instillation of 0.125% pilocarpine.

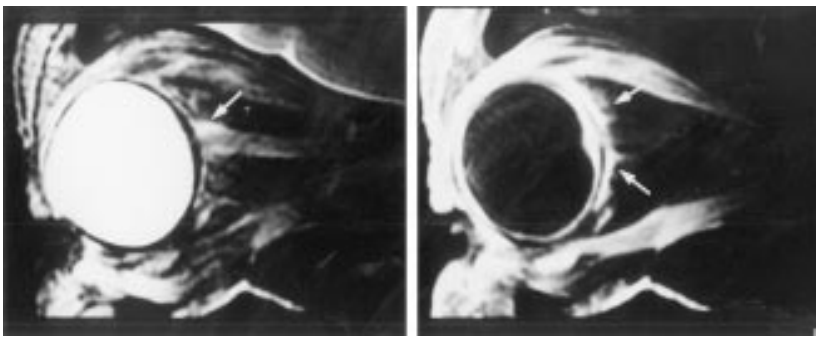

Figure 2 Sagittal T2 weighted magnetic resonance imaging scan (left) shows a high signal intensity area in the optic nerve sheath and adjacent posterior sclera (arrow). Sagittal post gadolinium-DTPA T1 weighted imaging scan using a fat suppression technique (right) shows enhancement effects around the posterior sclera and the optic nerve sheath (arrows).

COMMENT

Gadolinium enhanced MRI with a fat suppression technique was helpful for diagnosing the lesion in this patient, while ultrasonography and computed tomography were unremarkable. Although the aetiology of the inflammation in this patient remains unknown, the optic disc oedema and the internal ophthalmoplegia were caused by the posterior scleritis. The ciliary ganglion and short ciliary nerves, which innervate the sphincter pupillae muscle, locate around the optic nerve and penetrate the sclera. Therefore, the ciliary ganglion or short ciliary nerves were thought to be damaged by the inflammation around the posterior sclera. The inflammation of the sclera also extended to the optic nerve sheath and was thought to produce the optic disc oedema. Results of MRI in this patient were consistent with this hypothesis. Some previous reports indicated that MRI is useful for diagnosing posterior scleritis.[3 4] This report shows that inflammatory changes in the retrobulbar area can be detected by gadolinium-DTPA T1 weighted imaging using a fat suppression technique and a surface coil.

KENJI OHTSUKA
MASATO HASHIMOTO
MICHIKO MIURA
YASUSHI NAKAMURA
Department of Ophthalmology, Sapporo Medical University, School of Medicine, Sapporo, Japan

Correspondence to: Kenji Ohtsuka, MD, Department of Ophthalmology, Sapporo Medical University, School of Medicine, S-1, W-16, Chuo-ku, Sapporo 060, Japan.

Accepted for publication: 12 February 1997

1 Watson PG. The nature and the treatment of scleral inflammation. *Trans Ophthalmol Soc UK* 1982;**102**:257-81.
2 Watson PG. The diagnosis and management of scleritis. *Ophthalmology* 1980;**87**:716-20.
3 Benson WE. Posterior scleritis. *Surv Ophthalmol* 1988;**32**:297-316.
4 Chaques VJ, Lam S, Tessler HH, Mafee MF. Computed tomography and magnetic resonance imaging in the diagnosis of posterior scleritis. *Ann Ophthalmol* 1993;**25**:89-94.

Corneoscleral laceration associated with passenger-side airbag inflation

EDITOR,—There have been several reports of ocular injury associated with driver's-side airbag inflation during motor vehicle accidents. Reported injuries range from periorbital fractures,[1 2] corneal abrasions,[2 3] lens subluxation,[1] hyphaema,[1–3] vitreous haemorrhage,[1 2] retinal tears,[1] retinal haemorrhage,[3] and retinal detachment[2 4] to corneoscleral lacerations associated with broken eyeglasses[2] and a tobacco pipe.[5] There is one report of corneoscleral laceration as a direct result of driver's-side airbag inflation.[6]

We describe a patient who sustained severe ocular injury due to inflation of a passenger-side airbag. This is the second report of a corneoscleral laceration due to airbag deployment, and the first report of such an injury due to passenger-side airbag inflation, without associated eyeglass wear or external objects.

CASE REPORT

In October 1994, a 10-year-old Asian girl wearing a three point lap shoulder belt was a front seat passenger in a 1994 Nissan Altima, which was travelling at 30–40 miles (48–64

Figure 1 External photograph of the right eye showing large corneoscleral laceration with prolapsed uvea and collapsed globe.

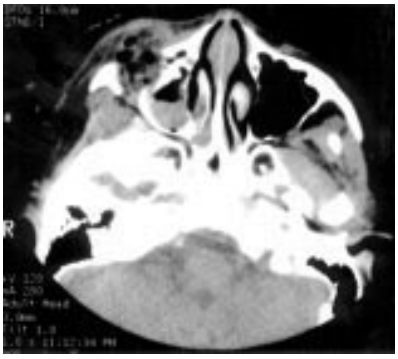

Figure 2 Computed tomography (axial view) demonstrates right inferior orbital wall fracture with haemorrhage in the right maxillary sinus.

km) per hour when it hit another car. Both front seat airbags inflated. Car damage was limited to the right front bumper and right front end. The windows and windshield were not damaged. There were no sharp objects inside the car, and the patient was not wearing eyeglasses. She sustained abrasions to her face, lacerations to her right eyelid and eye, and a bruise to her left chest. The restrained driver was uninjured.

External examination revealed moderate right periorbital and eyelid ecchymosis with two linear partial thickness lacerations of the right upper lid. There was mild ecchymosis of the left upper lid. Extraocular movements were restricted in all gazes in the right and normal in the left eye. Uncorrected Snellen visual acuity was right eye no light perception and left eye 20/40. Slit-lamp examination showed a distorted right globe with diffuse conjunctival chemosis and injection and a large linear corneoscleral laceration which extended from the superior 10 o'clock limbus, transecting the cornea, to 10 mm beyond the inferior 5 o'clock limbus (Fig 1). There was prolapsed uvea at the inferior limbus. The cornea was diffusely oedematous and hazy. The anterior chamber was collapsed. There was no view of the lens or posterior pole. The left eye had trace diffuse conjunctival injection and was otherwise normal. Computed tomography (CT) scan revealed a right orbital floor fracture with blood in the right maxillary sinus (Fig 2). There was no evidence of intraocular foreign body on CT scan.

The corneoscleral and eyelid lacerations were repaired. Visual acuity in the right eye remained no light perception and the left eye improved to 20/20. The patient's family was reluctant to consent to enucleation until 2 weeks later, at which time enucleation of the right eye and placement of a Medpor implant was performed.

COMMENT

This is the first report of a corneoscleral laceration due to passenger-side airbag inflation and the second report of a corneoscleral laceration due to airbag inflation, without associated eyeglass wear or external objects. Since the patient was not wearing eyeglasses and her arms and hands were uninjured, it is likely that the corneoscleral laceration and orbital fractures were directly related to inflation of the airbag. This patient may have been more susceptible to this type of injury, because as an Asian individual, she lacked a prominent inferior orbital ridge and maxillary prominence. Of note, the first reported case of corneoscleral laceration due to airbag inflation also involved a young Asian woman.[6]

Ophthalmologists should be aware of the potential for airbag associated ocular injuries. In addition, Asian individuals may be more prone to severe ocular injury as a result of airbag inflation. As others have suggested, reports of ocular injuries related to airbags may indicate the need to refine airbag deployment systems to prevent airbag associated morbidity.[1 3 4] In order to facilitate recording of airbag related injuries, all new incidents should be reported to the appropriate authorities. In the USA, incidents should be reported to the National Traffic Highway Safety Administration via the Auto Safety Hotline at 800-424-9393.

ABDHISH R BHAVSAR
Jules Stein Eye Institute, UCLA, Los Angeles, CA 90095, USA

TERESA C CHEN
DEBRA A GOLDSTEIN
UIC Eye Center, University of Illinois Eye and Ear Infirmary, Chicago, IL 60612, USA

Correspondence to: Abdhish R Bhavsar, MD, Jules Stein Eye Institute, 100 Stein Plaza, UCLA, Los Angeles, CA 90095-7000, USA.

Accepted for publication: 10 March 1997

1 Scott IU, John GR, Stark WJ. Airbag-associated ocular injury and periorbital fractures. *Arch Ophthalmol* 1993;**111**:25.
2 Gault, JA, Vichnin MC, Jaeger EA, Jeffers JB. Ocular injuries associated with eyeglass wear and airbag inflation. *J Trauma* 1995;**38**:494–7.
3 Mishler, KE. Hyphema caused by air bag. *Arch Ophthalmol* 1991;**109**:1635.
4 Han DP. Retinal detachment caused by air bag injury. *Arch Ophthalmol* 1993;**111**:1317–8.
5 Walz FH, Mackay M, Gloor B. Airbag deployment and eye perforation by a tobacco pipe. *J Trauma* 1995;**38**:498–501.
6 Baker SB, Flowers CW, Singh P, Smith A, Casey R. Corneoscleral laceration caused by air-bag trauma. *Am J Ophthalmol* 1996;**121**:709–11.

Keratoconjunctivitis after exposure to party foam

EDITOR,—Foam parties are becoming an increasingly popular method of enhancing entertainment at dance parties and discotheques. Foam is usually shot out of a cannon onto people on the dance floor who then dance covered in foam. The effects of this foam have hardly ever been examined.[1] We would like to report three patients who had a keratoconjunctivitis and corneal epithelial defect as a result of having foam sprayed in their eyes.

CASE REPORT

Three patients arrived at the emergency room of the ophthalmology department all complaining of sudden pain and burning in both eyes after having foam sprayed into their faces during a dance party in a discotheque.

All patients had no known ophthalmological problems whatsoever and were healthy individuals otherwise. They had all tried to wash out the foam with tap water after the injury occurred.

After pH measurement, irrigation and flooding with isotonic sodium chloride solution as well as ascorbic acid was undertaken. All patients were then tested for their visual acuity. This was followed by a slit-lamp examination.

The average pH value was 7.5. The mean visual acuity was Snellen 0.5 (20/40), the exact breakdown being seen in Table 1.

Table 1 pH values and visual acuities of affected patients

	pH value		*Visual acuity*	
Patient	*Right eye*	*Left eye*	*Right eye*	*Left eye*
PC	7.6	7.8	0.6	0.4
PU	7.4	7.4	0.4	0.6
HK	7.6	7.3	0.5	0.5

All three patients had follicular and papillary conjunctival injection. The corneal involvement was either in the form of punctate epithelial defects (three of the six eyes) or as a full epithelial corneal defect (three eyes).

Full epithelial defects were then treated with a patch using gentamicin and prednisolone ointment. The punctate defects were treated with gentamicin and prednisolone drops three times daily. The next day, all patients were re-examined and in two cases further therapy with betamethasone/neomycin eyedrops four times daily was necessary for another 24 hours.

After contacting the representative of the foam manufacturer, we received a fax which described the foam as non-toxic (LD_{50} > 10.00 mg/kg) and over 90% biodegradable. The content of the foam was described as 'anionic tensoactives' (not further defined).

COMMENT

This report shows that chemical foam used in dance parties should be considered as an alkali substance which can have a very toxic effect on the eye. It is important to specify the toxicity of these substances and warn that such foams can have harmful ophthalmic effects.

As to the therapy, the treatment is identical to that of any alkali burn.[2 3] It would seem that there are no complications if treatment is initiated immediately.

RAMIN BARADARAN-DILMAGHANI
ERDEM ERGUN
KATHARINA KREPLER
Department of Ophthalmology, University of Vienna, Vienna, Austria

Correspondence to: Ramin Baradaran-Dilmaghani, MD, Department of Ophthalmology, University of Vienna, Allg Krankenhaus, Waehringer Guertel 18-20, A-1090 Vienna, Austria.

Accepted for publication: 10 March 1997

1 Cather JC, Yee RW, Kilimanjaro-Davies H. Chemical keratoconjunctivitis from a foam party. *N Engl J Med* 1996;**334**:474.
2 Alio JL, Ayala MJ, Mulet ME, Artola A, Ruiz JM, Bellot J. Topical treatment of experimental acute corneal inflammmation by dexamethasone and nonsteroidal drugs. *Ophthalmic Res* 1994;**26**:87–94.
3 Saika S, Uenoyama K, Hiroi K, Tanioka H, Takase K, Hikita M. Ascorbic acid phosphate ester and wound healing in rabbit corneal alkali burns: epithelial basement membrane and stroma. *Graefes Arch Clin Exp Ophthalmol* 1993; **231**:221–7.

Donor corneo-scleral rim cultures after organ culture

EDITOR,—Organ culture is now the most common corneal preservation technique used in European countries. We evaluated sterility of organ cultured donor corneas at the time of surgery.

REPORT

From December 1992 to March 1996, 396 consecutive corneas were organ cultured at our institution. Of these 396 corneas, 270 (68%) were grafted and 126 (32%) were discarded. Eyes were obtained by enucleation within 48 hours of death. At the eye bank, eyes were rinsed for 30 seconds with sterile water then immersed in a 1% povidone-iodine solution for 2 minutes.[1 2] Eyes were then immersed in a 1% thiosulphate solution for 30 seconds and rinsed with sterile saline. Corneas were organ cultured in Inosol medium (Opsia, Toulouse, France) at +31°C for 2 to 5 weeks. Inosol contains penicillin (100 IU/ml), streptomycin (100 µg/ml), and amphotericin (0.25 µg/ml). The culture medium was renewed on day 14 and day 28. After organ culture, the corneas were incubated in Exosol deswelling medium at room temperature for 1 to 4 days.

Four days before the end of organ culture, a sample of the preservation medium was cultured on thioglycolate broth at 37°C and trypticase soy at 22°C for 14 days. Corneas were accepted for transplantation only when the medium remained clear and red after at least 2 weeks of organ culture with negative 48 hour cultures. At the time of transplantation, the corneo-scleral rim and a sample of deswelling medium were sent to the department of microbiology for culture. Specimens were inoculated in thioglycolate broth and incubated at 37°C for 7 days.

Nine per cent (36 of 396) of the corneas were discarded because of contamination during organ culture. Contamination was either bacterial (67%) or fungal (33%). Of the 24 isolated bacteria, 22 (92%) were resistant to penicillin and 14 (58%) were resistant to gentamicin. None of the 14 tested antibiotics was effective against the 24 bacteria. Bacterial contamination occurred on day 6.2 (SD 3.8, range 2–14). Fungal contamination occurred on day 7.6 (SD 4.3, range 3–14). No positive culture occurred when the preservation medium had remained clear and red during organ culture. The incidence of donor cornea contamination decreased with time. Seven of 49 corneas from donors who died of infectious disease were contaminated (14%) while 29 of 347 corneas from other donors were contaminated (χ^2 1.18, p = 0.28). Corneo-scleral rims were sterile in 98.9% of cases (267 of 270) of the grafted corneas. Deswelling media were sterile in 100% of cases (270 of 270). The corresponding deswelling media were sterile. No endophthalmitis occurred after transplantation.

COMMENT

Little is known about the result of corneo-scleral rim cultures after organ culture. Most European eye banks do not culture corneo-scleral rims after surgery.[3] Erbezci *et al*[4] found a 6.7% incidence of rim contamination after organ culture. The drawback of microbiological safety assessed with organ culture is a loss of corneas through contamination during preservation. The incidence of contamination decreases with the experience of the technicians and is dependent on the expertise of protocols followed within individual eye banks. Two previous studies[3 5] reported either a 0.5% and a 4.5% incidence of contamination during organ culture.Organ culture at +31°C allows corneas to be grafted sterile in almost 100% of cases. The chance of grafting a contaminated cornea after organ culture (1.1% in our study) is lower than after corneal storage at +4°C as described in the medical literature (Table 1). This is of interest as the chance of endophthalmitis is dramatically increased when donor corneo-scleral rims are contaminated.[6 9] It is of note that no endophthalmitis occurred after transplantation of these 270 organ cultured corneas.

Table 1 Results of donor corneo-scleral rim cultures after corneal storage at + 4°C

Study	*No*	*Contaminated rims No (%)*
Leveille 1983[6]	1876	230 (12)
Mathers 1987[7]	291	82 (39)
Fong 1988[8]	302	43 (14)
Antonios 1991[9]	1399	405 (29)
Kloess 1993[10]	932	128 (14)

VINCENT M BORDERIE
MANUEL LOPEZ
LAURENT LAROCHE
Banque de Cornées Saint Antoine, ETS de l'APHP, Paris, France

Correspondence to: Vincent M Borderie, Banque de Cornées Saint Antoine, Service d'Ophtalmologie, Hôpital Saint Antoine, 184 rue du Fg St-Antoine, 75571 Paris Cedex 12, France.
Accepted for publication: 12 February 1997

1 Borderie V, Kantelip B, Delbosc B, Oppermann M, Laroche L. Morphology, histology and ultrastructure of human C31 organ-cultured corneas. *Cornea* 1995;**14**:300-10.
2 Laroche L, Borderie V, Lopez M, Saraux H. Sécurité microbiologique et contrôle de qualité endothéliale au cours de la conservation des greffons cornéens à +31°C. *J Fr Ophtalmol* 1994;**17**:314-20.
3 Hagenah M, Böhnke M, Engelmann K, Winter R. Incidence of bacterial and fungal contamination of donor corneas preserved by organ culture. *Cornea* 1995;**14**:423-6.
4 Erbezci M, Monnot PH, Michel-Briand Y, Masse M, Delbosc B. Conservation en culture d'organe à +31°C de la cornée humaine et risque infectieux. *J Fr Ophtalmol* 1995;**18**:106-13.
5 Easty DL, Armitage WJ, Bawden R, Bowerman P. Influence of donor and storage factors on quality of corneas stored by organ culture. *Invest Ophthalmol Vis Sci* 1996;**37**:S945.
6 Leveille AS, McMullan D, Cavanagh HD. Endophthalmitis following penetrating keratoplasty. *Ophthalmology* 1983;**90**:38-9.
7 Mathers WD, Lemp MA. Corneal rim cultures. *Cornea* 1987;**6**:231-3.
8 Fong LP, Gladstone D, Casey TA. Corneo-scleral rim cultures: Donor contamination. A case of fungal endophthalmitis transmitted by K-Sol stored cornea. *Eye* 1988;**2**:670-6.
9 Antonios SR, Cameron JA, Badr IA, Habash NR, Cotter JB. Contamination of donor cornea: postpenetrating keratoplasty endophthalmitis. *Cornea* 1991;**10**:217-20.
10 Kloess PM, Stulting RD, Waring GO, Wilson LA. Bacterial and fungal endophthalmitis after penetrating keratoplasty. *Am J Ophthalmol* 1993;**115**:309-16.

Anterior segment ischaemia after excision of conjunctival squamous cell carcinoma

EDITOR,—Anterior segment ischaemia was first described by Schmidt[1] in 1874 and later by Hayreh[2] in 1979. It is a serious complication of squint[3] and retinal detachment[4] surgery. We present an elderly patient who developed anterior segment ischaemia after excision of conjunctival in situ squamous cell carcinoma. To our knowledge, this is the first reported case.

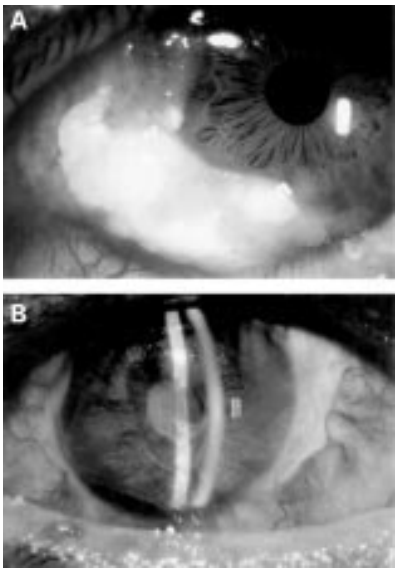

Figure 1(A) Slit-lamp photograph showing an elevated greyish lesion on the temporal and superior conjunctiva with corneal invasion in the right eye. (B) Slit-lamp photograph of the right eye: iris sector atrophy extending between 4:00–12:00, posterior synechiae and cataract.

CASE REPORT

A 68-year-old Saudi man presented with a mass at the temporal limbus of the right cornea. He had previously undergone excision of a pterygium and a trabeculectomy in the same eye.

The patient's medical history was suggestive of atrial fibrillation. Echocardiography showed no abnormalities and he was not receiving any treatment for cardiac disorders. Systemic examination was normal. Best corrected visual acuity was 20/70 in the right eye. Intraocular pressure was 12 mm Hg. Slit-lamp examination showed a vascularised, slightly elevated, reddish grey lesion extending from the 6 o'clock to the 11 o'clock position of the right limbus and invading 2 to 3 mm of the superficial cornea (Fig 1A). A filtering bleb was functional at the 12 o'clock position.

Complete excision of both lesions was performed, including a 2 to 3 mm free zone around the margin of the temporal lesion. Frozen sections of the same lesion showed in situ squamous cell carcinoma with all margins free of abnormality.

Five days postoperatively, visual acuity decreased dramatically with conjunctival injection, corneal oedema, Descemet membrane folds, anterior chamber flare, and a dilated non-reactive pupil. Intraocular pressure was 9 mm Hg. Investigations for uveitis and blood dyscrasia were unremarkable.

Anterior ischaemia was suspected. The patient was prescribed prednisolone acetate eyedrops (1%) every 3 hours. Four days later, visual acuity in the right eye improved to 20/200 with reduction of corneal oedema. Two weeks later, the intraocular pressure increased to 10 mm Hg after being 0 with minimal corneal oedema and minimal anterior chamber reaction. Posterior synechiae for 360° developed despite intensive therapy with sectorial iris atrophy (Fig 1B). The patient underwent cataract extraction with a final visual acuity of 20/50.

COMMENT

The eye in this case underwent an uneventful excision of a locally invading lesion with minimal bleeding, which was controlled with the wet field bipolar cautery. The cardiac rhythm, the blood pressure, and the pulse were monitored and observed to be at normal limits during the procedure. He reported 5 days later because of decrease in visual acuity, photophobia, and tearing. Mild anterior uveitis and corneal oedema resolved after intensive treatment with local corticosteroid drops.

The blood supply of the anterior segment of the eye has been described by several authors.[5–9] Ocular hypotension in our case suggested interference with the arterial supply to the anterior uvea, indicating disruption of the major arterial circle. Damage to the anterior episcleral arterial circle secondary to the type of conjunctival incision used could explain the factors predisposing to the anterior segment ischaemia. The patient developed low intraocular pressure anterior uveitis, corneal oedema, mature cataract, posterior synechiae of 360°, and iris atrophy[10] as a result of iris sector infarction. Another factor that could have predisposed to anterior segment ischaemia in our case was the trabeculectomy procedure, which possibly could have added to the damage of anterior episcleral arterial circle with poor compensation from the long posterior ciliary artery.

Anterior segment ischaemia after extensive or repeated surgery, especially in the elderly medically compromised, remains a risk. Early diagnosis and prompt treatment are of great importance in preventing permanent visual damage.

AMAL I AL-HEMIDAN
SELWA A F AL-HAZZAA
SAAD A AL-HADDAB
Department of Ophthalmology, King Faisal Specialist Hospital and Research Centre, Riyadh, Saudi Arabia

Correspondence to: Selwa A F Al-Hazzaa, MD, Department of Ophthalmology (MBC-40), King Faisal Specialist Hospital and Research Centre, PO Box 3354, Riyadh 11211, Saudi Arabia.

Accepted for publication 12 February 1997

1 Schmidt H. T-Beibrag Zur Kenntris der embolie der arterio centralis retinae. *Arch Ophthalmol* 1874;**20**:287.
2 Hayreh SS. Ischemia of the anterior segment of the eye. *Irish Faculty of Ophthalmology Yearbook* 1979/1980;21-31.
3 Lee JP, Olver JM. Anterior segment ischemia. *Eye* 1990;**4**:1-6.
4 Wilson WA, Irvine SR. Pathologic changes following disruption of blood supply to iris and ciliary body. *Trans Am Acad Ophthalmol Otolaryn* 1955;**59**:501-2.
5 Hayreh S, Scott W. Fluorescein iris angiography II. Disturbances in iris circulation following strabismus operation on the various recti. *Arch Ophthalmol* 1978;**96**:1390-400.
6 Morrison JC, VanBuskirk EM. Anterior collateral circulation in the primate eye. *Ophthalmology* 1983;**90**:707-15.
7 Wilcox LM, Keough EM, Connolly RJ, Ramberg-Las Karis K. Effect of contiguous recti (sr/mr) tenotomy on blood flow to the primate eye. *Invest Ophthalmol Vis Sci* 1981;**20**:191.
8 McKeown CA, Lambert HM, Usaf MC, Shore JW. Preservation of the anterior ciliary vessels during extraocular muscle surgery. *Ophthalmology* 1989;**90**:499-507.
9 Hayreh SS, Scott WE. Anterior segment ischemia following retinal detachment surgery. *Mod Probl Ophthalmol* 1979;**20**:148-53.
10 Olver JM, Lee JP. The effect of strabismus surgery on anterior segment circulation. *Eye* 1989;**3**:318-26.

Pseudocystic ultrasound appearance of choroidal melanoma

EDITOR,—Ultrasonography is useful in the diagnosis, treatment, planning, and monitoring of intraocular tumours. An important step in the diagnostic process is the differentiation between solid and cystic tumours. Uveal melanomas are usually solid but may rarely be cystic (W R Lee, personal communication). Cases have been reported in which pseudocystic appearances on ultrasonography have been due to haemorrhage or necrosis.[1–3] We report a patient who appeared to have a cystic tumour on B-scan ultrasonography, which was shown on histology to be due to a very lightly pigmented nodule in a deeply pigmented melanoma.

CASE REPORT

The patient, a 56-year-old Asian man, was referred to St Paul's Eye Unit from Taiwan for investigation and treatment of a choroidal tumour in the right eye. He presented with a 16 week history of blurred vision and visual field loss in the affected eye. Systematic inquiry revealed no other abnormalities. On examination, the vision was 6/12 in the right eye and 6/6 in the left eye. There was a relative afferent pupil defect in the right eye. The anterior segments and intraocular pressures were normal. The right posterior segment showed a large, pigmented choroidal tumour temporally, extending from the ora serrata to the temporal macula. This was associated with an exudative retinal detachment. The left fundus was healthy. B-scan ultrasonography showed the tumour to have sagittal and transverse basal dimensions of 17.2 mm by 16.6 mm, with a thickness of 11.3 mm. The internal tissue reflectivity suggested the presence of a large thin walled cyst (Fig 1).

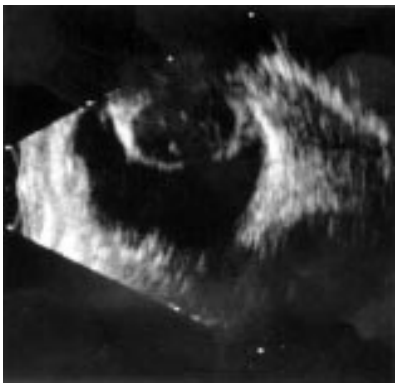

Figure 1 B-scan ultrasound demonstrating an apparently cystic choroidal mass.

Trans-scleral local resection[4] was performed and the tumour was examined by light microscopy after staining with haematoxylin and eosin in the conventional manner (Fig 2). On low power examination the tumour was shown to be entirely solid, with the pseudocystic area corresponding to a lightly pigmented nodule within a deeply pigmented tumour. High power microscopy, after melanin bleaching, revealed that the peripheral, very heavily pigmented tumour was of epithelioid cell type and the pseudocystic, lightly pigmented area consisted of spindle B type cells. There were no areas of necrosis and only a few small areas of haemorrhage.

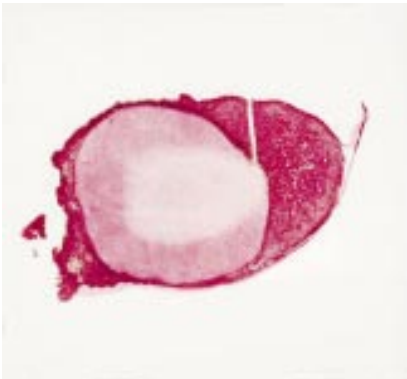

Figure 2 Light micrograph showing a lightly pigmented nodule of spindle B melanoma cells within a densely pigmented epithelioid cell tumour. (Haematoxylin and eosin, magnification × 3).

COMMENT

Uveal melanomas are only rarely cystic. A pseudocystic appearance on B-scan ultrasonography of a solid uveal melanoma is an uncommon phenomenon and has been previously attributed to local areas of haemorrhage or necrosis.[1–3] This is the first published case in which local variations in pigmentation of the tumour alone might account for the B-scan pseudocystic appearance. The acoustically silent area, mimicking a cystic cavity, was composed of cells that were both amelanotic and spindle-shaped. It is not possible, therefore, to determine which of these two features was responsible for the ultrasonographic appearances. Despite the difference in the cell type of the central and peripheral areas of the tumour, the most striking abnormality is the difference in pigmentation. An A-scan may allow differentiation between a pseudocyst and a true, haemorrhagic cyst by showing after-movement of spikes in the true cyst.[1] In conclusion, a solid uveal melanoma may falsely appear cystic on B-scan ultrasonography if it contains a nodule of non-reflective tissue.

IAN A PEARCE
P A SMITH
B E DAMATO
St Paul's Eye Unit, 8Z Link, Royal Liverpool University Hospital, Liverpool

Accepted for publication 12 February 1997

1 Frazier Byrne S, Green RL. *Ultrasound of the eye and orbit*. St Louis: CV Mosby, 1992:136–44.
2 Desjardins L, Berges O, Perichon Y, Martini P. Two cases of choroidal melanomas presenting as pseudocyst on echographic examination. In: Frezzotti R, Balestrazzi E, Falco L, Esente E, eds. *International symposium on intraocular and epibulbar tumours*. Bologna: Monduzzi Editore, 1994:65–9.
3 Guthoff R, Terwey B, Burk R, von Domarus D. (Attempt at preoperative differentiation of malignant melanoma of the choroid. A comparison of nuclear magnetic resonance tomography, ultrasound echography and histopathology.) *Klin Monatsbl Augenheilkd* 1987;**191**:45–9.
4 Damato BE, Foulds WS. Surgical resection of choroidal melanomas. In: Ryan SJ, Ogden TE, Schachat AP, eds. *Retina.* 2nd ed. St Louis: CV Mosby, 1993:33–5.

CORRESPONDENCE

Euphorbia sap keratouveitis

EDITOR,—I congratulate Scott and Karp for their excellent paper on *Euphorbia* sap keratopathy,[1] but would like to emphasise that the sap of certain plants of this genus can cause blindness.

Clinicians should be aware of the sight threatening uveitis caused by some euphorbias. Duke-Elder has reviewed the older literature, citing reports of severe keratouveitis caused by the sap of the European perennial herb *Euphorbia esula* (previously *Tithymalus esula*) and a Cuban species recorded as *E antiquorum*.[2] Uveitis with hypopyon has been described with sap of the European perennial herb *E cyparissias* (previously *T cyparissias*),[2] and the Indian succulent *E royleana*.[3 4] It is possible, however, that the hypopyon uveitis in some late presenting cases may simply represent a response to secondary microbial keratitis. The sap of the common European herbaceous weed *E peplus* (petty spurge) causes a marked fibrinous uveitis in association with 'typical' *Euphorbia* keratopathy, in the absence of secondary infection.[5–7] Petty spurge sap has traditionally been used as a wart cure, and it is in this context that ocular toxicity may occur.[6 7] Cases presenting early, and managed supportively, have had a good outcome.

Keratoconjunctivitis without significant uveitis has been described in relation to several species of the genus *Euphorbia*.[1 2] Though this resolves without sequelae if appropriately managed,[1] there is a risk of corneal ulceration and subsequent blindness in neglected cases.[2 3]

As with other cases of plant toxicity, the patient should be asked to provide a specimen of the offending plant for identification. Flowering or fruiting parts will greatly assist the botanist. Our understanding of *Euphorbia* ocular toxicity is still limited, and would benefit from the publication of further well prepared case reports.

The author wishes to thank Dr R J Gornall, curator of Leicester University Botanic Garden, for advice on taxonomy.

TOM EKE
Academic Unit of Ophthalmology, Leicester Royal Infirmary, Leicester LE1 5WW

1 Scott IU, Karp CL. Euphorbia sap keratopathy: four cases and a possible pathogenic mechanism. *Br J Ophthalmol* 1996;**80**:823–6.
2 Duke-Elder S. *System of ophthalmology*. Vol 14, Part 2. London: Kimpton, 1972:1185.
3 Sofat BK, Sood GC, Chandel RD, Mehrotra SK. Euphorbia royleana latex keratitis. *Am J Ophthalmol* 1972;**74**:634–7.
4 Sood GC, Sofat BK, Chandel RD. Injury to the eye by the sap of Euphorbia royleana. *Br J Ophthalmol* 1971;**55**:856–7.
5 Hartmann K. Augenschadigung durch den Saft der Euphorbia peplus (Wolfsmilch). *Klin Monatsbl Augenheilkd* 1940;**104**:324–6.
6 Eke T. Acute kerato-uveitis associated with topical self-administration of the sap of the petty spurge (Euphorbia peplus). *Eye* 1994;**8**:694–6.
7 Biedner BZ, Sachs U, Witztum A. Euphorbia peplus latex keratoconjunctivitis. *Ann Ophthalmol* 1981;**13**:739–40.

Reply

EDITOR,—We thank Dr Eke for his comments. It is important that ophthalmologists are aware of the potential complications of ocular exposure to *Euphorbia* sap.

Euphorbia sap may lead to conjunctivitis, keratitis, and uveitis.[1–3] As demonstrated in our series,[4] keratoconjunctivitis resulting from *Euphorbia* sap exposure may resolve without visually significant residue. However, as Eke's comments emphasise, sight threatening complications, including severe uveitis, corneal ulceration, scarring, and subsequent blindness,[5 6] may result from *Euphorbia* sap exposure. Therefore, patients require careful follow up and prompt treatment.

Ophthalmologists need to be aware of the potential ocular complications of *Euphorbia* sap exposure, and the importance of closely following patients with *Euphorbia* sap ocular toxicity. Patients who work with *Euphorbia* plants should be cautioned to wear eye protection.

INGRID U SCOTT
CAROL L KARP
Bascom Palmer Eye Institute, PO Box 016880, Miami, FL 33101, USA

1 Duke-Elder S. *System of ophthalmology*. Vol 14, part 2. London: Kimpton, 1972:1185.
2 Crowder JI, Sexton RR. Keratoconjunctivitis resulting from the sap of candelabra cactus and the pencil tree. *Arch Ophthalmol* 1964;**72**:476–84.
3 Eke T. Acute kerato-uveitis associated with topical self-administration of the sap of the petty spurge (Euphorbia peplus). *Eye* 1994;**8**:694–6.
4 Scott IU, Karp CL. Euphorbia sap keratopathy: four cases and a possible pathogenic mechanism. *Br J Ophthalmol* 1996;**80**:823–6.
5 Sood GC, Sofat BK, Chandel RD. Injury to the eye by the sap of Euphorbia royleana. *Br J Ophthalmol* 1971;**55**:856–7.
6 Sofat BK, Sood GC, Chandel RD, Mehrotra SK. Euphorbia royleana latex keratitis. *Am J Ophthlamol* 1972;**74**:634–7.

Elimination of trachoma

EDITOR,—The availability of a new long lasting antibiotic, azithromycin, has sparked a new trachoma control initiative. Morocco has been chosen as the first country in the programme for the elimination of trachoma. This country was selected because it offers ideal conditions for success and may provide some insight towards sustainable control. The search for an initial success is sound, as long as it is understood that Morocco is not typical of the poorest countries in which the disease poses a greater problem. However, the Moroccan programme may provide misleading insights toward sustainable control of the disease in less optimal environments. Morocco has strong health infrastructures and the disease is limited to few pockets of moderate intensity that are isolated from other important foci of trachoma by thousands of kilometres of desert, a barrier that has proved formidable throughout history. Therefore, the risk of recrudescence of the disease caused by migration will be minimal. The vast majority of poor countries will have to defend the result achieved by the attack phase of their programme with a maintenance phase of undetermined length.

Elimination of trachoma blindness rather than elimination of trachoma may prove the feasible objective in poor African countries. To achieve this objective the following techniques should be developed:

1 mapping of the disease distribution and severity
2 flexible cut off point for mass treatment
3 definition of a maintenance phase
4 evaluation of the maintenance phase

Probably, none of these will be needed, and therefore devised, by the Moroccan programme.

If these assumptions are correct, a second effort should start as soon as possible in a poor country. Such programmes should add a note of realism to the enthusiasm that the success of the Moroccan programme may generate. It should also develop the needed techniques for effective, efficient, and sustainable control of trachoma in the countries in which the disease poses the greater problem. Otherwise, when these countries become interested in trachoma control we should ask them to wait until we find out how to do it.

G De Sole
1 Pascoe Avenue, BP 6988, Harare, Zimbabwe

CD-ROM REVIEW

ProVision interactive: clinical case studies. Volume 1. Cornea and neuro-ophthalmology. San Francisco: American Academy of Ophthalmology, 1996. $115 (members), $165 (non-members).

This is another product in the ever more rapidly growing number of CD-ROMs in ophthalmology. The program was found easy to install and ran smoothly even on machines with the minimum requirements stipulated. These are very reasonable, being a PC with a 486 SX 66 CPU running MS DOS 5.0 and Windows 3.x or a Macintosh with a 68040 processor running system 7.01. Both need a minimum of 8Mb RAM, 640×480 monitor (VGA+ for PCs) with 256 colours, 2X speed CD-ROM drive, and 5Mb of hard drive space. The vast majority of users should therefore have no problem running this CD.

The program itself is nicely laid out and reasonably intuitive. For beginners and computerphobes a clearly labelled tutorial button is available throughout the program which calls up a comprehensive and understandable breakdown of the functions of all buttons and options available.

The graphics are of high quality and there is an enlarge function for all pictures which gives good quality results. The sound element in this multimedia package consists of patient dialogue expanding on aspects of the case history. The dialogue is clear, sharp, and easy to understand although it failed to add much to the content of the clinical picture being presented.

ProVision interactive: clinical case studies. Volume 1 consists of three clinical cases with corneal pathology and three with neuro-ophthalmic pathology. The interactive format lends itself very well to approaching these cases in a manner similar to the clinical situation. On choosing a case a brief description of the presenting complaint is given and on screen buttons are made available as the user goes through the case. The buttons bring up

detailed screens, many with several sub-options, allowing the user to be practically as superficial or as aggressive as preferred in the pursuit of a diagnosis. A wide range of differential diagnoses are available and there are options for advice from other ophthalmologists, consultations from other specialties, and a vast range of investigations available. A review case button is active throughout and brings up all relevant information obtained up to that point by the user. The program gives preferred diagnoses rather than dogmatic responses to the user's conclusions. It contains several papers on each case for the user to refer to. It also has a facility for users to annotate the cases themselves.

The product is very much aimed at the American market. The American Academy of Ophthalmology gives 18 CME points for the whole six cases and the program has a button to print out forms to apply to the academy for the CME points. Investigations are given with their unadjusted national Medicare prices in the USA.

In summary, in favour of this CD-ROM are its user friendliness, quality graphics, depth of information, and facilities for adding notes; against are the heavy bias towards a US market and a rather high price tag for only three corneal and three neuro-ophthalmic cases.

PETER CARUANA

NOTICES

American Institute of Ultrasound in Medicine

The second annual bilingual America's conference on ultrasound will be held on 1–3 June 1997 in Miami Beach, Florida, USA. Further details: Stephanie Reisberg or Kimberly Mullaney. (Tel: (301) 498-4100; email: pubs_govt@aium.org)

Conferences on Angiography in Créteil

A conference on clinical cases in ICG will be held on 9 June 1997 at the University of Créteil. Further details: Professor Gisèle Soubrane, Clinique Ophtalmologique Universitaire de Créteil, 40 Avenue de Verdun, 94010 Créteil Cédex, France. (Tel: 45 17 52 22.)

British Council International Seminar

A British Council international seminar (number 97031) entitled 'Corneal and external eye disease: new surgical techniques' with Professor D L Easty as director will be held on 29 June to 5 July 1997 in Bristol, UK. The seminar will be of particular interest to all young eye surgeons from the developing and developed world. Further details: Promotions Manager, International Seminars, The British Council, 1 Beaumont Place, Oxford OX1 2PJ, UK (Tel: +44 (0) 1865 316636; fax: +44 (0) 1865 557368/516590; email: International.Seminars@britcoun.org)

European Association for the Study of Diabetic Eye Complications (EASDEC)

The 7th meeting of EASDEC will be held on 18–19 July 1997 at the Okura Hotel, Amsterdam, the Netherlands, as a pre-congress symposium of the 16th International Diabetic Federation (IDF) congress. Further details: Professor BCP Polak, Rotterdam Eye Hospital, PO Box 70030, 3000 LM Rotterdam, the Netherlands. (Fax: (31) 10 4017655.)

Continuing Medical Education

The 17th annual current concepts in ophthalmology will be held on 25–27 July 1997 at the San Diego Marriott Mission Valley, San Diego, California, USA. Further details: Marie Krygier, Medical Education Coordinator, San Diego Eye Bank, 9444 Balboa Avenue, Suite 100, San Diego, CA 92123, USA. (Fax: (619) 565-7368.)

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 Gelisken, 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.)

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)

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/)

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, Ocular Infections, PO Box 50006, Tel Aviv, 61500, Israel. (Tel: 972 3 5140000; fax: 972 3 5175674 or 5140077.)

Correction

A spelling error occurred in a reply to a letter that appeared in the August 1996 issue of the *BJO* (1996;**80**:773–4). The correct spelling of the author's name is C Karabatsas. We apologise for this error.

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.

CORRESPONDENCE

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.

TABLES

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.

SI UNITS

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.

STATISTICS

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.

Advice may be available to authors before submission of papers.

Proofs

Contributors will receive ONE proof, and should read it carefully for printers' errors. Alterations to the original text should be kept to a minimum and may be charged to the author. Responsibility for validation of the proof lies with the author.

Reprints

These may be ordered from the Offprint Office when proofs are returned.