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 injuries 2 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 pICOL implantation was performed (Fig 2). The clinical course was unremarkable. Postoperative 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 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 and 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 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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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.

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 pICOL performed. VA left eye 20/200. Keratoplasty as been advised.
Corneal ectasia after trabeculectomy with mitomycin C application

EDITOR—Trabeculectomy combined with mitomy cin 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 triangularly excisional scleral flap centred on the 12 o’clock position and a 1 × 3 mm trabeculectomy door 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 20/50 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 135 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,2 senile marginal degeneration, or keratoconus. Several studies have reported the “with the rule” changes in the corneal astigmatism in the early postoperative period of trabeculectomy.3–5 The changes that were usually under 2phereus were removed 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.6 It verified the possibility that MMC can induce a late tissue dysfunction. In our case, MMC might have penetrating the stromal layer from the cutting edge of the scleral flap during the trabeculectomy causing keratoocyte 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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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 proliferative 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 almost absent.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

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. HbA1c was 7.0–7.5%. The patient also had acromegaly. Her height was 166 cm and weight 58 kg. Her serum IGF-I level was 726 ng/ml. Physical examination revealed acromegaly: coarse features, mandibular prognathism, acral enlargement, and macroglossia in all four limbs (Fig 1). 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 0.3–210 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, diabetic retinopathy was a complication in both eyes. Despite additional operations (trabeculectomy and...
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.11 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 associates12 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.13 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. Some investigators reported that IGF-I accelerates the expression of VEGF in some cell lines.2 Thus, GH may have played an important role in the progression of diabetic retinopathy in combination with IGF-I and VEGF.2 Diabetic retinopathy associated with acromegaly should be further studied to examine the correlation among GH, IGF-I, VEGF, and other angiogenic factors.

COMMENT

It is well known that diabetic retinopathy is an occasional complication of diabetic mellitus secondary to acromegaly, 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 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.11 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 associates12 recently reported that inhibition of GH can inhibit ischaemia induced retinal neovascularisation in vivo via VEGF.

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Emergency contraception and retinal vein thrombosis

EDITOR.—The article by Vessey et al1 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 pupil defect, normal anterior segments, and intraocular pressures of right eye 17 mm Hg, left eye 18 mm Hg. Dilated fundoscopy 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 Remarkable neovascularisation in the patient’s right eye (A) and large fibrovascular membrane in the left eye (B).

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Figure 1 Right fundus photograph demonstrating superior hemiretinal vein occlusion.
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 enlargement of the right superior rectus/levator complex compared with the left side.

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.

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.

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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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Intraocular pressures were 56 mm Hg bilaterally at diagnosis. The condition was successfully treated with topical Timoptol and Prednisolone (Fluress), including increased intraocular pressure, which was reduced to 28 mm Hg bilaterally with no adverse effects. A full recovery, with no abnormalities found on ocular examination, was achieved 6 months later.

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 (as of 1996, 5 anaphylactic and 1 anaphylactoid reaction). The patient experienced severe eye pain and loss of vision, and one case of anaphylaxis was reported in 1996.

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 woman, who had worn soft contact lenses for 1 month, presented in February 1994 with unilateral eye pain, redness and swelling (two), and one case of anaphylactic reaction. 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.

Vidjer, A. (1990) A case of severe bradycardia following exposure to oxybuprocaine eye drops was reported.

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.

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.

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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.
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 pathogens 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 (Biomat 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, 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. Jenny 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. Attendence 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 exist- ing workforce. However, I would be more than willing to give examples of where it has been done.

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EDITOR,—I was interested to read Webber and Jeffrey’s elaboration of the role of the ophthalmic medical assistant in the BJO 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 presence of inappropriate admissions, suboptimal theatre timing, and other problems”. Later, the authors suggest that “less operating time is wasted through inappropriate theatre lists”. This seems to herald the development of an “OMA role” which would appear completely lacking in the corporate social responsibility and leadership required to play such a role. The suggestion that the introduction of the OMA role will stop medical muddles and wasted time 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 who 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 is in accord with the development of an unaccountable, unregulated personal servant with no formal training. Nurses can and do 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 possess any of the flexibility of that workforce. This is in accord with the development of an unaccountable, unregulated personal servant with no formal training. Nurses can and do possess any of the flexibility of that workforce.

JOHN LEE
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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, untalented 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 worth-while tasks. All truly dedicated 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 as an OMA for another healthcare professional. Not every person employed in patient care wishes to climb the NHS managerial ladder. As evidence of job satisfaction, we would look to the camaraderie that all OMAs enjoy 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

1 Webber SK, Jeffrey MN. Ophthalmic medical assistants (commentary). Br J Ophthalmol 1999;83:3-4.

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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

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/)

Ophthalmology 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 Ophthalmologique 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 Oogheelkunde, de Pintelaan 185, B-9000 Gent, Belgium. Deadline for applications 31 December 1999.

Correspondence, Notices
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.

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)

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