

CORRESPONDENCE

Diagnosis of activated protein C resistance in retinal vein occlusion

EDITOR.—I read with interest the article published in the *BJO* by Hunt.¹ In this paper the author reports that the prevalence of APC resistance is more common than any other known thrombophilic state and was present in over one third of patients younger than 45 years with central retinal vein occlusion (CRVO). Therefore, many would argue that a full thrombophilia screening should be performed in a young patient (<50 years old) after CRVO. This screening should be made using an easy to perform technique and providing good discrimination between normal and APC resistant subjects. In this regard, Hunt comments that the biological technique proposed by Dhålbäck *et al.*² yields sensitivity and specificity ranging from 85% to 90%, but that it is not reliable if the patient has abnormal clotting such as a lupus anticoagulant or is receiving anticoagulants. For these reasons this technique is not suitable for the screening of patients with venous thrombotic disorders, including CRVO. With regard to this point, it is worth taking into account that our group has proposed a technique,³ which modifies the initial technique proposed by Dhålbäck *et al.*,² diluting the sample of the patient's plasma with plasma lacking factor V. With this modification, both the specificity and sensitivity of the technique is 100%⁴ and, moreover, it may be used in patients receiving anticoagulant therapy and in those who have lupus anticoagulant.

Thus, in opinion of Dhålbäck,⁵ this technique may be suitable for the screening of patients with venous thrombotic disorders since it is very sensitive and specific as well as inexpensive and easy to perform.

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Reply

EDITOR.—Since the writing of my editorial on activated protein C resistance and retinal vein occlusion, Dr Aznar has published his modification of the laboratory technique using factor V deficient plasma. It has been taken up widely by coagulation laboratories in the UK in view of its better specificity and sensitivity. When ophthalmologists are screening for activated protein C resistance they should be

aware there are now two assays available and ask which assay the laboratory is routinely using.

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Peripapillary circle of Zinn-Haller

EDITOR.—We were interested in the article by Myung-Kyoo Ko *et al.* regarding the visualisation of the circle of Zinn by fluorescein angiography. However, we do not agree that it is possible to truly visualise the circle of Zinn by this method. As such, we feel that the point of view taken by the authors to reach their conclusions is flawed based on the following observations.

We have been investigating the location of the circle of Zinn by measuring the depth of the circle from the retinal side of the scleral surface, along with the distance between the circle of Zinn and the optic nerve sheath. We have chosen the classic longitudinal serial sectioning technique of the optic nerve and surrounding tissues. Observation at both sides of the sectioned optic nerve, aided by serial sections, has enabled us to conclude that the circle of Zinn lies at a relatively constant depth around the optic nerve in any individual eye.

Moreover, the mean depth of the circle of Zinn was 345 µm from the scleral surface in our study, which is larger than that in the report by Olver *et al.* which is cited in Myung-Kyoo Ko and colleagues' work. This is equivalent to 70% of the thickness of the retina at the posterior pole, roughly 1.5 times the thickness of the choroid. It is very unlikely that the small blood vessels at this depth in the sclera can be visualised by fluorescein angiography. There is no evidence that in myopic eyes with tilted discs, peripapillary sclera is "thinner" than normal.

A third point is the size of the blood vessels that were observed in Myung-Kyoo Ko *et al.* In our study, the mean radius of the lumen of the arteriole that consists the circle of the Zinn was 45.6 µm in 29 eye bank eyes. However, the vessels indicated in the photographs are much larger than this. These vessels appear to be as large as the proximal retinal arteries in all the photographs, and peripapillary arteries are about 200 µm wide, with a lumen of 100 µm in diameter.

Clinically, we have also noted these vessels and considered their choroidal vessels, readily visualised because of the pigment epithelial degeneration. Furthermore, the report of their vessels only on the temporal side in myopes needs additional explanation.

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- Ko M-K, Kim D-S, Ahn Y-K. Peripapillary circle of Zinn-Haller revealed by fundus fluorescein angiography. *Br J Ophthalmol* 1997;**81**:663-7.

Reply

EDITOR.—We thank Dr Gauntt for comments on our article.¹ He raises some questions about the depth of the CZH within the sclera, about visualisation of this vessel by fundus fluorescein angiography (FFA), and about the diameter of the vessel appeared on FFA. Though we have not investigated the mean depth of the CZH within the sclera, it is questionable that the CZH lies at a relatively constant depth within the sclera. If the vascular circle lies in such a constant position within

the sclera, why have previous study using serial transverse or longitudinal histological sections to evaluate circulation of the anterior optic nerve failed to identify this intriguing vessel? This is partly because the CZH does not run in the same plane within the sclera, and we think that the serial histological section may give a little information about the CZH. As our previous study suggests,² the CZH has tremendous morphometric and morphological variations between subjects and even in the same subject through its course around the optic nerve. These inter-individual variations have also been reported by Olver *et al.*³ Additionally, the branching pattern of the vascular circle is so complex² that in simple histological section the main body of the circle may not be distinguished from its branches to peripapillary choroid or to the optic nerve.

Gauntt expresses doubt as to the visualisation of the CZH by FFA, because he suggested that it runs within sclera at such a depth (345 µm from the retinal side of the scleral surface) as to be invisible. However, we would like to point out again that the CZH has variable morphology in its course and does not run in the same plane anteroposteriorly and mediolaterally. Furthermore, chorioretinal atrophy and scleral thinning in the temporal crescent area, especially in pathological myopia, may offer a better chance to appreciate the temporal part of the CZH by imaging methods such as FFA or indocyanine green angiography (ICG). A recent report⁴ demonstrates the visualisation of the CZH by ICG in severely myopic eyes which supports our idea. However, the CZH could not be visualised even in severe myopic conditions when it is deeply seated within the sclera.

Regarding the diameter of the CZH, our previous study using the flat section method² showed marked variation. It ranged from 20 µm to 270 µm with a mean of 123 (SD 75) µm. Even in the same circle it showed fluctuation during its course. In some cases the diameter of the main body of the CZH was less than that of its branch. Moreover, the abrupt decrease of the vessel calibre, the narrow portion of the CZH, was noted in many of our cases. Sometimes the peripapillary choroidal arterial arcade might be confused with the CZH in FFA. However we believe that it is distinguished from the CZH by several points. It is located along, the outer margin of the temporal crescent while the CZH appeared as concentric vascular filling within the temporal crescent or in the vicinity of the optic disc margin. It is obscured in the late phase of FFA by dye leakage from the intact choriocapillaris along the temporal crescent margin. Also, some branches from the arterial circle directed to the optic disc or to the peripapillary choroid can be observed on FFA.

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- Ko M-K, Kim D-S, Ahn Y-K. Peripapillary circle of Zinn-Haller revealed by fundus fluorescein angiography. *Br J Ophthalmol* 1997;**81**:663-7.
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Effectiveness of occlusion therapy in ametropic amblyopia

EDITOR.—I congratulate the authors of this study for making the conceptual leap that control trials of intervention for amblyopia are necessary.¹ To my knowledge, this is the first study of its kind and while it has some limitations for which it will no doubt be criticised, it shows the way for future researchers in this field.

What the authors have shown is that occlusion for 1 hour a day had no long term additional benefits over spectacle correction alone. I am interested to know why they chose this occlusion regime. It would also have been helpful if the initial and final acuities attained for each patient had been documented. Are they also convinced that all their patients were truly amblyopic, and not just children with refractive errors who became more cooperative with repeated testing?

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1 Moseley MJ, Fielder AR, Irwin M, *et al.* Effectiveness of occlusion therapy in ametropic amblyopia: a pilot study. *Br J Ophthalmol* 1997;**81**:956–61.

Reply

EDITOR.—We would like to thank Mr Clarke for his support of our belief that occlusion therapy should be subject to controlled trials.

As stated in our paper, we chose to adopt a minimal occlusion regimen as this has previously been found to be beneficial while minimising the burden placed on parents and children.¹ We would stress, however, that our study was not primarily an attempt to evaluate this occlusion regimen, rather to address some methodological issues which future studies will need to take into account. We could, therefore, have legitimately adopted any regimen of our choosing.

Clarke's concern that our subjects may not have been amblyopic but merely possessing refractive errors raises important questions for both taxonomy and treatment. Children presenting with significant refractive errors and a loss of visual acuity (in the absence of other pathological findings) will fall into one of three categories: those whose visual acuity loss is completely (and instantaneously) ameliorated by refractive correction; those whose acuity loss is restored over a period of weeks or months; and a third category, those in whom acuity, as in the preceding category improves over time, but never reaches 6/6. Conventionally, children in the first category would not be diagnosed as being amblyopic while those in the third category meet the standard diagnostic criteria of amblyopia. But what of those in the second category? Most authorities would also apply the label of amblyopia to these patients although we cannot differentiate the factors which may contribute to the improvement associated with "long term" spectacle wear (for example, a putative anti-amblyopic effect, perceptual learning, familiarisation with test and tester) or define the nature of the underlying pathophysiology. In our study, none of the subjects who completed the trial attained 6/6 (category 3 above) though as alluded to in our paper and confirmed by subsequent experience,² had the spectacle ("pre-treatment") phase of the trial been extended,

some subjects may have attained normal acuity (category 2 above). We acknowledged in our paper that increased cooperation with repeated testing could have contributed to the improvement seen in all of our subjects.

With the exception of one particular subject of interest, we did not, in addition to stating the changes in the mean and standard deviation of subjects' recorded visual acuity at each phase of the trial, present data from individuals. We would be happy to make this available to anyone wishing to undertake further analysis.

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High prevalence of recurrent symptoms following uncomplicated traumatic corneal abrasion

EDITOR.—We read with interest the comments by Sabri *et al*¹ on the many treatments used to manage acute corneal abrasions and the lack of any evidence based protocol. An evidence based approach requires that the natural history of the condition is well understood: a review of the literature shows a surprising lack of documentation regarding the clinical course of corneal abrasion after the initial injury has healed.² Therefore, we present some of our observations of symptoms following uncomplicated traumatic corneal abrasion.

We prospectively followed 74 adults with traumatic corneal abrasion in previously healthy eyes. This was part of a study to assess whether the nightly use of lubricating ointment could reduce the incidence of recurrent corneal erosion. In 42 cases, the injury was caused by a fingernail. All patients were treated with a standard regimen of cyclopentolate 1% eye drops immediately and chloramphenicol four times daily for 5 days; eyepads were not used.^{2,3} "Fingernail" injuries were randomised to receive either the standard treatment alone, or to continue with a topical lubricating ointment nightly for 2 months. Symptoms were assessed 3 months after the injury by a telephone questionnaire, and case notes were reviewed after 2 years.

We found a high prevalence of symptoms suggestive of recurrent corneal erosion at 3 months' follow up. Twenty one patients (28%) reported frequent and significant pain, grittiness, photophobia, or watering of the injured eye only.⁴ Symptoms were relatively minor (did not interfere with daily activities) in 9/42 (21%) of "fingernail" and 3/32 (9%) of "non-fingernail" injuries. Symptoms were severe enough to interfere with daily activities, or to cause the patient to seek advice from their general practitioner or pharmacist in four (10%) fingernail and four (12%) non-

fingernail injuries. By 2 years, two patients with fingernail injuries had presented with recurrent corneal macroform erosions,^{5,6} one at 3 months and the other after 7 months.

Our results illustrate that our understanding of this common condition is still very limited. A surprisingly high proportion of patients endure recurrent symptoms following traumatic corneal abrasion, but do not re-present to the ophthalmologist. This high prevalence of symptoms suggests that we should consider changing our initial treatment regimen. We suggest that any future evaluation of treatment for corneal abrasion should include long term follow up of patient symptoms.

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Reply

EDITOR.—We thank Morrison and colleagues for their comments regarding the long term outcome of corneal abrasions. Our survey¹ looked at the immediate treatment of corneal abrasions only. However, Morrison *et al* mention that there have been no reproducible scientific studies looking at the natural history of corneal abrasions. The overwhelming conclusion from both Morrison's and our studies is that further large scale, scientific research is needed in order to look at the most effective treatment regimens in terms of both immediate and long term outcome of corneal abrasions.

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- 1 Sabri K, Pandit JC, Thaller VT, *et al.* Current management of corneal abrasions: evidence based practice? *Br J Ophthalmol* 1997;**81**:1116–17.

Cluster of four cases of inadvertent injury to the globe secondary to peribulbar anaesthesia

EDITOR.—A cluster of four cases in 4 months of inadvertent injury of the globe after peribulbar anaesthesia (PBA) administered by both anaesthetists and ophthalmologists has led us to review their risk factors, management, and outcome with respect to the literature.^{1–5} Despite varying symptoms the common sign of inadvertent injury was vitreous haemorrhage either preoperatively or immediately postoperatively. Time to referral for vitreoretinal opinion ranged from immediate to 48 hours by which time all eyes had extensive vitreous haemorrhage precluding

Table 1 Summary of individual patient data

	Patient 1	Patient 2	Patient 3	Patient 4
Sex	F	M	F	F
Age (years)	73	71	69	69
Preoperative visual acuity	6/18	6/18	HM	CF
Axial length (mm)	25.52	33.43	22.00	23.25
Peribulbar anaesthetic details:				
Administered by	Anaesthetist	Anaesthetist	Ophthalmologist	Anaesthetist
Number of injections	2	2	4	2
Needle type	Sharp	Sharp	Sharp	Sharp
Interval to diagnosis (days)	1	2	Immediate	Immediate
Clinical indicator(s) of inadvertent injury:				
Pain	N	N	N	Y
Decreased visual acuity	Y	Y	Y	Y
Hypotony	N	Y	N	N
Vitreous haemorrhage	Y	Y	Y	Y
Subretinal haemorrhage	Y	N	Y	Y
Choroidal haemorrhage	Y	N	Y	Y
Other	N	Retinal detachment	N	Retinal detachment, haemorrhagic chemosis
Interval to repair (days)	4	42	10	8
Operative findings:				
Category	3	3	3	3
Vitreous haemorrhage	Y	Y	Y	Y
Choroidal haemorrhage	Y	N	Y	Y
Optic atrophy	N	Y	N	Y
Retinal detachment	N	Y (macula off)	Y	Y (macula off)
Other	Subretinal needle track, retinotomies x2	Retinal break inferotemporal to macula	Supernasal retinal tear	Scleral lacerations x2
Surgical procedure				
	Drainage of choroidal haemorrhage, TPPV, SF ₆	TPPV, argon endolaser to tear, silicone oil	Phaco and IOL, drainage of choroidal haemorrhage, TPPV, cryotherapy to tear, SF ₆	Drainage of choroidal haemorrhage, repair of scleral lacerations, TPPV, silicone oil. Phaco and IOL at weeks
Outcome:				
Follow up (weeks)	40	6	29	15
Visual acuity	6/9	HM	6/36	HM
Retina attached?	Y	Y (optic atrophy)	Y (epiretinal membrane)	N

TPPV = trans pars plana vitrectomy; SF₆ = sulphur hexafluoride gas; Phaco and IOL = phacoemulsification cataract extraction with insertion of intraocular lens.

visualisation of the fundus. All injuries required trans pars plana vitrectomy and internal tamponade with resulting visual acuity ranging from 6/9 to HM (Table 1).

PBA was first promoted as a safer alternative to retrobulbar anaesthesia (RBA) but a prospective comparative study has not been undertaken.⁶ The advantages of PBA include reduced incidence of inadvertent penetration of intraocular tissues which can result in brain stem anaesthesia while the disadvantages include inadequate akinesia in up to 50% of cases.^{1,2} Ocular factors such as increased axial length and technical factors such as inadequately trained personnel are associated with inadvertent injury which can result from administration of the anaesthetic by both ophthalmologists and anaesthetists.¹⁻³ Inadvertent injury produces a variety of symptoms and signs and may even be silent.²⁻⁵ Although intraocular injections of anaesthetic agents are not retinotoxic, damage can result from mechanisms such as "jet stream effects" and elevation of the intraocular pressure.^{3,7,8}

Suspicion of an inadvertent injury by the administrator of the anaesthetic must be conveyed to the operating surgeon (if they differ), so that a full assessment can be performed before surgery.^{2,3} Differentiation between penetrating injuries (only an entry site) and perforating injuries (an entry and an exit site) and classification into one of three categories depending on the clarity of the media and the presence of retinal detachment has management implications.³ Category one patients with an adequate fundal view without retinal detachment may be watched as local haemorrhage may preclude the need for prophylactic laser therapy (cryotherapy in the presence of local haemorrhage has been implicated in the development of proliferative vitreoretinopathy).²⁻⁴ We agree with previous reports that trans pars plana vitrectomy in category two (an adequate fundal view with retinal detachment) and category three patients (an inad-

equated fundal view) is the treatment of choice.^{2,3} Patient outcome depends on the amount of initial damage to the retina and despite successful retinal reattachment, factors including epiretinal gliosis, optic atrophy and proliferative vitreoretinopathy can result in poor visual acuity.²⁻⁵ Adequate training of personnel and a high awareness of potential risk factors¹ may help to reduce the incidence of this serious complication of local anaesthesia. Early recognition of an injury and early vitreoretinal opinion to categorise the patients and formulate a management strategy may maximise a patient's visual outcome.²⁻⁵

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Telemedicine and computers in diabetic retinopathy screening

EDITOR,—Williamson and Keating in a recent commentary in the *BjO* highlight the need to provide full screening for diabetic patients for retinopathy throughout the community. They presuppose that the key to diabetic retinopathy screening is efficiently obtaining images of the retina for classification.¹ While the idea of digital fundus images being remotely assessed by trained personnel or even analysed by a computer automatically is attractive and may be practicable in the not so distant future, retinal photography is not the only way to organise screening for diabetic retinopathy. The other main approach to screen for diabetic retinopathy is a dilated fundus examination done by trained personnel.^{2,3} Optometrists are ideally suited to fulfil this role, as they are spread throughout the community and as a large section of diabetics already visit their optician.⁴

To be effective a screening programme has to achieve near total coverage of the target population. This can only be done if a database of all known diabetics is maintained in the form of a diabetes register, which will generate call and recall and monitor outcomes. This is a priority that has been recognised by the task force of the British Diabetic Association and the UK Department of Health.⁵ The setting up and maintenance of a diabetes register is largely computer dependent, involving assimilation of data from general practice and hospital computer systems. Several districts including our own have already implemented this or are well on their

way to doing so. There is a fundamental role for computers in the organisation of diabetic retinopathy screening to achieve this.

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Reply

EDITOR,—We suspect that in diabetic screening, as with other practical areas, it is not who screens or what method is used but how effectively it is done. Trained, highly motivated optometrists could well provide computer register based screening and indeed recent joint working party guidelines have been issued for optometry screening services.¹ This report recommends annual dilated funduscopy, ideally using slit lamp biomicroscopy. However, it also stresses the need for continuing annual audit and evaluation to ensure an adequate sensitivity and specificity of detection of retinopathy as previously recommended for any screening service.² This would be time consuming for the optometrist and no national arrangements for financing this service have been made; therefore, would this be cost effective for the optometrist's business and for the health purchaser?

The other issue is that not all diabetics attend an optometrist regularly, especially young, emmetropic, type 1 diabetics who are at higher risk of aggressive retinopathy. Other patients may be being screened by several different services which is unnecessary and wasteful of resources. The extent of population coverage by retinopathy screening services is currently the subject of a Department of Health supported national audit. A computerised database would undoubtedly improve the identification of diabetic patients and improve cost efficiency. A national register would be desirable allowing for allocation of resources and the geographic relocation of patients. With advancing technology this could in the future be combined with digitised image acquisition and storage systems.

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BOOK REVIEWS

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Dictionary of Optometry and Visual Science. 4th ed. By M Millodot. Pp 296. £30. Oxford: Butterworth-Heinemann, 1997. ISBN 0-7506-3145-7.

It would be churlish to be critical of this book. To have reached its fourth edition in 11 years, and have the previous edition reprinted three times suggests that it is doing something right. It is indeed a very useful reference book for optometrists and others with an involvement in optical science. The appeal to the student is its comprehensive nature: to have geometrical optics formulas alongside ocular anatomy makes it a daily companion. For the experienced practitioner too, it provides a concise reminder of dimly remembered definitions, and proof that there is always something new to learn. When I looked up the description given by colleagues of their method of measuring pupil size, I found that this was in fact "Broca's pupillometer"—although my recollections of using an instrument called a Wesley keratometer for this purpose were not featured!

Space is obviously at a premium in the book with over 3800 terms included, and the fourth edition has been expanded to reflect the addition to the title of "Visual Science". Terms such as magnocellular which did not appear in the previous edition are now amply covered, with copious cross referencing to lateral geniculate bodies, Y cells, parvocellular visual system, and dyslexia! This cross referencing is used very effectively to avoid repetition. To take an example, a Maddox cross is described as a "scale for measuring the angle of heterophoria and heterotropia" but it is only by following the cross reference to Maddox rod that you find out how this is done. Reading further you find that the Maddox rod can be used to measure cyclophoria, you again look elsewhere to find out what that is, and then the cross references to "test, double prism" and "test, Maddox rod" provide you with two methods of detection, one of which can also quantify the deviation. The author describes this as an effective method of self teaching—its only danger is that you become sidetracked by intriguing entries such as "test, Raubitschek" along the way. (In case you have never heard of it either, it is actually a test chart to measure astigmatism.) There is plentiful use of alternate listings to be sure you find what you are looking for, even if you don't go to it immediately: ARM, AMD, and senile macular

degeneration all lead to "maculopathy, age-related". It is a little frustrating at first, but the reader soon realises that "cover test" is under "test, cover", and ciliary muscle is "muscle, ciliary", although "frequency, spatial" is much less intuitive. A new feature in this edition is the truly excellent use of tables. As well as more conventional data such as optical constants of the eye, average size of the normal visual field, and common photometric units, there are also helpful lists of all the terms the book contains within a particular subject area—for example, ophthalmic drugs, optical dispensing, and contact lenses. Some figures and photographs have also been added, but their usefulness is more mixed. To illustrate the "target of the direct comparison eikonometer" is of enormous help in understanding the description, but "occluder in front of the left eye" is less illuminating, and the photograph of the "typoscope" is even slightly misleading.

In summary, any reader with a few minutes to browse cannot fail to find an intriguing trail of cross references to follow. For those with more definite purpose who seek enlightenment, they will surely find it in this comprehensive volume which looks set to go from strength to strength.

CHRIS DICKINSON

Fundus fluorescein angiography. By Amresh Chopdar. Pp 148. £72.50. Oxford: Butterworth-Heinemann, 1996. ISBN 0-7506-1885-X.

This relatively short book gives an introduction to fluorescein angiography. The first few chapters outline the relevant basic sciences and the reader will learn the history of fluorescein angiography, as well as discover that the melting point of sodium fluorescein is 315°C! A chapter entitled "Techniques and pitfalls" provides useful information for those who are planning to do the photography themselves.

Two chapters concentrate on the appearances of normal angiograms and how to interpret abnormal features. The bulk of the book gives a fairly standard step by step account of different retinal and choroidal diseases. There are separate chapters for age related macular degeneration and diabetic retinopathy. Throughout, the author provides high quality images to demonstrate different pathologies. The author, however, frequently digresses from describing retinal and choroidal pathology to give details on their clinical management. The last chapter very briefly, covers the advancing field of digital retinal imaging which, unfortunately, is already looking dated.

Every chapter ends with a helpful list of references and there are appendices which list the resuscitation equipment and drugs required to perform fluorescein angiography. However, the final appendix on the classification of laser burns seems rather superfluous.

Overall, this book gives a good introduction of fluorescein angiography, it will be useful to candidates who intend to sit postgraduate examinations as well as to those who wish to refresh their knowledge on this subject. The quality of the angiograms will both impress the reader as well as incite some envy.

PARWEZ HOSSAIN

NOTICES

Avoidable blindness

The latest issue of the *Community Eye Health* (no 25) discusses the elimination of avoidable blindness. With an editorial by Bjorn Thygefors, the director of the WHO Programme for the Prevention of Blindness and Deafness, the issue covers treatment of cataract in regions of India and the role of patient counsellors in increasing the uptake of cataract surgery and IOLs. For further information please contact *Community Eye Health*, International Centre for Eye Health, Institute of Ophthalmology, 11-43 Bath Street, London EC1V 9EL. (Tel: (+44) 171 608 6910; fax: (+44) 171 250 3207; email: eyeresource@ucl.ac.uk) Annual subscription £25. Free to workers in developing countries.

Residents' Foreign Exchange Programme

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

2nd International Conference on Ocular Infections

The 2nd International Conference on Ocular Infections will be held on 22-26 August 1998 in Munich, Germany. Further details: Professor J Frucht-Pery, 2nd International Confer-

ence on Ocular Infections, PO Box 50006, Tel Aviv, 61500, Israel. (Tel: 972 3 5140000; fax: 972 3 5175674 or 5140077; email: ocular@kenes.com)

XVI Tuebingen Detachment Course

The XVI Tuebingen Detachment Course in retinal and vitreous surgery will be held 4-5 September 1998 in Odessa, Ukraine. Further details: Professor I M Logai, Director, The Filatov Institute, 49/51 Boulevard Francais, Odessa, 270061, Ukraine. (Tel: +38-0482-22 20 35; fax: +38-0482-68 48 51.)

International Agency for the Prevention of Blindness (IAPB)

The International Agency for the Prevention of Blindness (IAPB) will hold its next general assembly in Beijing, China on 5-10 September 1998. Further details: Gullapalli N Rao, Secretary General, IAPB Secretariat, LV Prasad Eye Institute, LV Prasad Marg, Banjara Hills, Hyderabad 500 034, India. (Tel: 091-40-215389; fax: 091-40-248267; email: IAPB@lvpeye.stph.net)

ICOP 98

The next International Conference in Ophthalmic Photography (ICOP) will be held on 19-21 September 1998. Further details: Mrs Gillian Bennerson, Senior Ophthalmic Photographer, Bristol Eye Hospital, Lower Maudlin Street, Bristol BS1 2LX. (Tel: 0117-928-4677.)

IV meeting of the European Society for Out-Patient Eye Surgery (ESOPES)

The IV meeting of the European Society for Out-Patient Eye Surgery (ESOPES) will be held in Vittel, France on 9-11 October 1998. Further details: Mrs Nicole Charron, Direc-

tor, Palais des Congrès, Av Bouloumie, BP 57, 8802 Vittel, France. (Tel: +33 329 08 18 30; fax: +33 329 08 6601.)

Vith International Symposium on Graves' Ophthalmology

The Vith International Symposium on Graves' Ophthalmology will be held on 27-28 November 1998 in Amsterdam. Further details: Amsterdam Thyroid Club, Department of Endocrinology, F5-171, Academisch Medisch Centrum, Meibergdreef 9, 1105 AZ Amsterdam, Netherlands.

Ophthalmic technologies

The 9th Ophthalmic Technology Conference will be held on 23-24 January 1999 during the International SPIE symposium on biomedical optics. Further information: The SPIE Organisation, PO Box, Bellingham, WA 98227-0010, USA. (Fax: (+1) 360-647-1445; email: www.spie.org/info/pw)

Laser eye injuries

A conference on the epidemiology, prevention, diagnosis, and therapy of laser eye injuries will be held in San Jose, California on 25-26 January 1999 during the International SPIE symposium on biomedical optics. Further information: The SPIE Organisation, PO Box, Bellingham, WA 98227-0010, USA. (Fax: (+1) 360-647-1445; email: www.spie.org/info/pw)

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@congreg.se; <http://www.congreg.com/soe/>)

British Journal of Ophthalmology - <http://www.bjophthalmol.com>

Visitors to the world wide web can now access the *British Journal of Ophthalmology* either through the BMJ Publishing Group's home page (<http://www.bmjpub.com>) or directly by using its individual URL (<http://www.bjophthalmol.com>). There they will find the following:

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Diagnosis of activated protein C resistance in retinal vein occlusion

JUSTO AZNAR

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