CORRESPONDENCE

Diagnosis of activated protein C resistance in retinal vein occlusion

EDITOR,—I read with interest 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 sectional 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, whereas in that in the report by Olver et al 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 contains the circle of 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 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 them 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.

JUSTO AZNAR
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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 diameter of the vessel as measured on FFA. Though we have not investigated the mean depth of the CZH within the sclera, it is possible 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 studies 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 histopathological section may give a little information about the CZH. As our previous study suggests, the CZH has tremendous morphometric and morphological variations in its components and even in the same subject through its course around the optic nerve. These interindividual variations have also been reported by Olver et al.2 Additionally, the branching pattern of the vascular circle is so complex 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, chororetinal atrophy and scleral thinning in the temporal quadrant area, especially in 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 report illustrates 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 method3 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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DONG-SEO KIM
YOON-KOO AHN
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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?

M P CLARKE
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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. 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 that 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 refer 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 further analyse it.

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MARY IRWIN
HELEN S JONES
Academic Unit of Ophthalmology, University of Birmingham

ROSEMARY J AULD
Department of Orthoptics, The Birmingham and Midland Eye Centre


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 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 the need for intervention.

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visualisation of the fundus. All injuries re-
quired 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 alterna-
tive to retrobulbar anaesthesia (RBA) but a
prospective comparative study has not been
undertaken.

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 akinsia in up to 50% of cases.
- Ocular factors such as increased axial length and technical factors such as inadequately trained personnel are associated with inadvertent injury which can result from the administration of the anaesthetic by both ophthalmologists and anaesthetists.
- Inadvertent injury produces a variety of symptoms and signs and may even be silent. Although intracocular injections of anaesthetic agents are not retinotoxic, damage can result from mechanisms such as “jet stream effects” and elevation of the intraocular pressure.

Suspicion of an inadvertent injury by the administra-
tor of the anaesthetic must be con-
veyed to the operating surgeon (if they differ), so that a full assessment can be performed before surgery.

Differentiation between pen-
etrating injuries (only an entrance site) and perfor-
ing injuries (an entrance 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 manage-
ment implications. Category one patients with an adequate fundal view without retinal detachment may be watched as local haemorr-
haege may preclude the need for prophylactic laser therapy (cryotherapy in the presence of local haemorrhage has been implicated in the development of proliferative vitreoretino-
pathy). We agree with previous reports that
trans pars plana vitrectomy in category two (adequate fundal view with retinal detach-
ment) and category three patients (an inade-
quate fundal view) is the treatment of choice. Patient outcome depends on the amount of initial damage to the retina and despite successful retinal reattachment, fac-
tors including epiretinal gliosis, optic atrophy and proliferative vitreoretinopathy can result in poor visual acuity. Adequate training of personnel and a high awareness of potential risk factors may help to reduce the incidence of this serious complication of local anaesthe-
sia. 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.

**Table 1 Summary of individual patient data**

<table>
<thead>
<tr>
<th>Patient 1</th>
<th>Patient 2</th>
<th>Patient 3</th>
<th>Patient 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>F</td>
<td>F</td>
<td>F</td>
</tr>
<tr>
<td>Age (years)</td>
<td>73</td>
<td>71</td>
<td>69</td>
</tr>
<tr>
<td>Preoperative visual acuity</td>
<td>6/18</td>
<td>6/18</td>
<td>HM</td>
</tr>
<tr>
<td>Axial length (mm)</td>
<td>25.82</td>
<td>33.43</td>
<td>22.00</td>
</tr>
<tr>
<td>Peribulbar anaesthetic details:</td>
<td>Administered by Anaesthetist</td>
<td>Anaesthetist</td>
<td>Ophthalmologist</td>
</tr>
<tr>
<td>Number of injections</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Needle type</td>
<td>Sharp</td>
<td>Sharp</td>
<td>Sharp</td>
</tr>
<tr>
<td>Interval to diagnosis (days)</td>
<td>1</td>
<td>2</td>
<td>Immediate</td>
</tr>
<tr>
<td>Clinical indicator(s) of inadvertent injury:</td>
<td>Pain</td>
<td>Decreased visual acuity</td>
<td>Hypotony</td>
</tr>
<tr>
<td>Pain</td>
<td>N</td>
<td>N</td>
<td>Y</td>
</tr>
<tr>
<td>Decreased visual acuity</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>Hypotony</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>Vitreous haemorrhage</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>Subretinal haemorrhage</td>
<td>Y</td>
<td>N</td>
<td>Y</td>
</tr>
<tr>
<td>Choroidal haemorrhage</td>
<td>Y</td>
<td>N</td>
<td>Y</td>
</tr>
<tr>
<td>Other</td>
<td>N</td>
<td>Retinal detachment</td>
<td>N</td>
</tr>
<tr>
<td>Interval to repair (days)</td>
<td>4</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Operative findings:</td>
<td>Category</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Category</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Vitreous haemorrhage</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>Choroidal haemorrhage</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Optic atrophy</td>
<td>Y</td>
<td>N</td>
<td>Y</td>
</tr>
<tr>
<td>Retinal detachment</td>
<td>N</td>
<td>N</td>
<td>Y (macula off)</td>
</tr>
<tr>
<td>Subretinal needle track, retinotomies ≥2</td>
<td>Drainage of choroidal haemorrhage, TPPV, SF,</td>
<td>Retinal break inferotemporal to macula, TPPV, argon endolaser to tear, silicone oil</td>
<td>Phaco and IOL, drainage of choroidal haemorrhage, TPPV, cryotherapy to tear, SF,</td>
</tr>
<tr>
<td>Surgical procedure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Outcome:</td>
<td>Follow up (weeks)</td>
<td>40</td>
<td>6</td>
</tr>
<tr>
<td>Visual acuity</td>
<td>6/9</td>
<td>HM</td>
<td>6/36</td>
</tr>
<tr>
<td>Retina attached?</td>
<td>Y</td>
<td>Y (optic atrophy)</td>
<td>Y (epiretinal membrane)</td>
</tr>
</tbody>
</table>
| TPPV = trans pars plana vitrectomy; SF, = sulphur hexafluoride gas; Phaco and IOL = phacoemulsification cataract extraction with insertion of intraocular lens.

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. 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. Optometrists are ideally suited to fulfil this role, as they are spread throughout the community and as a large sector of diabetics already visit their optician.

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. 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 track.
Correspondence to: Dr Prasad.

EDITOR,—We suspect that in diabetic screening way to doing so. There is a fundamental role for optometry screening services.

optometrists could well provide computer screens or what method is used but how e resources and the geographic relocation of services which is unnecessary and patients may be being screened by several differ
take their time in the book with over 3800 terms included, and the fourth edition has been expanded to reflect the addi
tion to the title of “Visual Science”. Terms such as magnocellular which did not appear in the previous edition are now amply covered,
this recollection of using an instrument called a “Broca’s pupillometer”—although my measuring pupil size, I found that this was in
given by colleagues of their method of measuring pupil size, I found that this was in

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BOOK REVIEWS


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 eye science. The appeal to the student is its comprehensive nature: to have geometrical fundus 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.

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.

The second chapter concentrates 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 advances in 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 superficial.

Overall, this book gives a good introduction to fluorescein angiography, it will be useful to candidates who intend to sit postgraduate examinations as well as to ophthalmologists 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 Thyl-efors, 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, University College Dublin, 60 Eccles Street, Dublin 7, Ireland, Germany, Denmark, France, Austria, or Portugal should apply to: Mr Robert Acheson, Senior Ophthalmic Photographers in Retinal and Vitreous Surgery, World Health Organization, WHO, 123 Avenue de la Libération, 1244 Geneva 27, Switzerland.

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

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 Francois, 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: Mrs Gillian Bennerson, Senior Ophthalmic Photographer, Bristol Eye Hospital, Lower Maudlin Street, Bristol BS1 2LX. (Tel: 0117-928-4677.)

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

VIIth International Symposium on Graves’ Ophthalmology

The VIIth 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

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@congrex.se; http://www.congrex.com/soe/)


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Diagnosis of activated protein C resistance in retinal vein occlusion

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