Letters to the editor

Axial length measurement discrepancies in asteroid hyalosis

Sir,—Axial length (AL) measurement is the most frequently used test in lens (IOL) power calculation. A difference of 1 mm in axial length will affect the postoperative refraction by approximately 2-5 dioptres. The ophthalmologist should therefore be aware of factors which may yield spurious measurements. These factors include posterior pole colobomas, posterior vitreous detachments, macular oedema, and others.1 Asteroid hyalosis (AH) is generally not expected to affect AL measurements. A review of the literature provided only one case report of falsely decreased AL measurement secondary to AH necessitating a second IOL implantation.2

Asteroid hyalosis is a common condition occurring most frequently in elderly individuals in the 7th and 8th decades. It appears as a myriad of tiny spherules containing calcium and phospholipids suspended throughout the vitreous. It is usually unilateral, has no sex predilection, and has not been definitively associated with systemic or ocular disease.1 We present a case in which asteroid hyalosis in a cataractous eye falsely lowered the axial length measurements, yielding an incorrect IOL power calculation. Implantation of this lens would have resulted in a significant refractive error. We studied the axial length measurements in five additional cases with asteroid hyalosis to compare measurements with predicted estimates.

A 52-year-old black diabetic female had been followed up by her retinal specialist for macular oedema OS. Examination of the left eye was remarkable for a visual acuity of 20/400 with a -1.00 sphere, a moderate nuclear sclerotic cataract, severe macular oedema, and asteroid hyalosis. The right eye had a posterior chamber IOL, a presumed pseudophakia, with a visual acuity of 20/25. Asteroid hyalosis was present OD, but ophthalmoscopic examination gave otherwise normal results.

An A scan was performed with the Cooper ABX 1000 and yielded AL measurements of 22.00 mm OD and 22.10 mm OS. The measurements were reproducible and resulted in an IOL power calculation of +2.43 D (SRK II regression formula) for both eyes. Since the average AL is 23.5 mm, these readings would be consistent with a hyperopia of 4 dioptres. However, the patient’s elicited refractive history, current visual acuity, and results on ophthalmic examination were not consistent with such a degree of hyperopia. In addition, the pseudophakic eye had received a +20.50 dioptres IOL (not 24.0 D) and had achieved emmetropia. We concluded that the AL measurement in both eyes was spuriously decreased secondarily to AH. Based on our clinical impression a +21.50 IOL was implanted OS instead of the calculated +24.0, with resultant postoperative refraction of plano.

Our case report describes artefactual lowering of the AL measurement in a patient with AH. Except for the above mentioned report1 AH has not previously been known to affect AL measurements. In our case macular oedema may have been a contributing factor in spuriously decreasing AL in the left eye. The right eye, however, had asteroid hyalosis without macular oedema but still had artefactual lowering of the AL measurement.

We believe the question of whether vascular or neurosensory dysfunction occurs first in diabetic retinopathy remains open.

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Diabetes and retinal function

Sir,—We were interested in the recent article by Bek and Lund-Andersen1 and in your accompanying editorial.2 It is true that much has been published in recent years concerning the vascular aspects of diabetic retinopathy. The earliest changes in diabetic retinopathy need not, however, be vascular. Thus, Bek and Lund-Andersen could not demonstrate areas of visual field loss corresponding to retinal hard exudates or fluorescein leakage, but this may be because their test of retinal function was not sufficiently sensitive. A similar study using a more sensitive test of retinal function such as S cone pathway sensitivity3 or luminance or chromatic threshold measurement4 might increase the likelihood of detecting differences in function between an area of apparently normal retina and an adjacent area where a breakdown in the blood-retinal barrier is obvious.

Although a breakdown in the blood-retinal barrier may precede neurosensory dysfunction as suggested by Bek and Lund-Andersen, recent work in our laboratory suggests the opposite may be true. We compared 36 patients with insulin-dependent diabetes with 36 age-matched controls. Funduscopy, fundal photography, and fluorescein angiography confirmed that none of the diabetics had breakdown of the blood-retinal barrier, but colour vision assessed by the Farnsworth-Munsell 100-hue test was markedly abnormal in the diabetic group compared with normal controls (mean FM 100-hue error score for diabetics 85-2 (SEM 7.6) v 29-5 (SEM 3.3) for controls, p < 0.001).

We believe the question of whether vascular or neurosensory dysfunction occurs first in diabetic retinopathy remains open.

Reply

Sir,—The question of which measure of retinal function is the best of course depends on what one wishes to study. What I think is important, however, is the impact of diabetic sensory dysfunction in practice appears to reflect clinically significant visual loss. Therefore the relation between perimetric results and extrafoveal morphology may help us to learn which pathological lesions are more likely to lead to lowered visual acuity. In this context the most important finding described in the paper might perhaps be the lack of correlation between fluorescein leakage and loss of retinal light sensitivity. The reason why some hard exudates do not produce visual field scotomata is probably that this lesion causes considerable light scattering, a phenomenon that can be directly observed with the scanning laser ophthalmoscope.

I agree that some subclinical measure of retinal neurosensory impairment may perhaps be the initial sign of retinopathy. However, I think that the reduced blue-sensitivity of diabetic patients that has been known since the 1960s is necessarily an argument in favour of this hypothesis. It has been shown that the lens (nuclear sclerosis) of diabetic lenses is higher than in normals, increasing with age and with poor metabolic control.5 This lens browning causes increased lens autofluorescence, absorption, and light scattering, and thereby less light transmission, especially in the blue-green area, an effect that can account for the decreased blue sensitivity of these patients (Larsen et al., in preparation: personal communication). Therefore I think that an evaluation of colour vision anomalies of diabetic patients at least requires a proper correction for the individual wavelength-dependent light loss in the refractive media (notably the lens) due to autofluorescence, absorption, and scatter.

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A light pipe guard to prevent iatrogenic retinal injury during vitrectomy

Sin,—Iatrogenic retinal injury has occurred in 38% of eyes undergoing vitrectomy for progressive proliferative diabetic retinopathy.1 An iatrogenic break worsens the visual prognosis.2 It is therefore essential to make every effort to avoid such injuries. One preventable cause is retinal injury due to the fibroptic light pipe. This can be avoided by using it with a guard to prevent excessive introduction. The light pipe length is excessive, 35 mm, and compares unfavourably with the average chord lengths: from pars plana port to macula of 22 mm, or to the proximal retina 16 mm (if a path parallel to the visual axis is taken).

Figure 1: The (35 mm) light pipe is covered at its proximal end with 20 mm of tarsorrhaphy tubing to guard against excessive introduction of the light pipe into the eye.

In order to minimise the risk to the retina we have restricted the introduction of the light pipe to 15 mm by covering the proximal pipe with a 20 mm length of tarsorrhaphy tubing (Fig 1). In this way it is impossible to introduce the light pipe far enough to injure the macula and yet it goes far enough to remain in view even if the pupil is not well dilated. This precaution will keep the tip 8–10 mm from the retina for most of the tip’s arc of movement within the eye.

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BOOK REVIEWS


This is a beautifully illustrated book, but really I am at a loss to comprehend just at which market it is being aimed. I have not had the opportunity to look at others in the series of Wolfe Medical Atlases. There are 244 excellent photographs, but, for instance, on gonioscopy (pp. 114–7) there are five pages of non-useful pictures, then two of angles of the anterior chamber, but no legend as to what is being viewed in the angle photographs.

There are 15 pages on fellow eye testing — the majority as provided by the instrument manufacturer — and the fields which are printed again have no explanatory caption. Photograph 58 on exophthalmometry has a legend which is somehow inaccurate, and illustration 16 on infant restraint is hardly likely to commend itself for help in primary care practice.

The presentation is excellent and the printing is superior to many other more useful publications, but I do not really see what addition it is to the voluminous range of titles already available. As claimed on the cover, this is an uncomplicated guide to testing of eye status, but I do not think optometrists would find much in the book of value, and for ophthalmologists there is really no information of use for revision, for practical or theoretical learning, or to recommend to practitioners interested in the specialty.

WILLIAM M DOIG


This book sets out to present a simple, concise, and well illustrated guide to proptosis surgery specifically directed at the surgeon who is less familiar with eyelid anatomy and who does not perform such operations on a daily or weekly basis. A logical classification based on the aetiology of proptosis is first presented with excellent photographs illustrating many different causes of it. Surgical anatomy is then covered with very clear, concise diagrams. The third chapter discusses presentation and examination, and the next four chapters detail the author's techniques for a modified Fasanella Servat procedure, sponaneous repair, anterior approach levator resection, and bone suspension. The surgery is presented with a matching series of operative photographs, diagrams, and explanatory captions which are incredibly clear and easy to follow. The actual text is kept to a minimum and supplements the operative series excellently. It is well laid out under the clear headings of anaesthesia, surgical technique, postoperative care, results, complications, and references.

It is extremely difficult to find anything to criticise in this book, and the author is to be congratulated on having produced the most beautifully clear and concise pictorial guide to ptosis that I have ever seen. Surgeons practising a lot of ptosis surgery may disagree with one or two minor details, such as the Iliff modification of the Fasanella Servat procedure, the lack of any mention of posterior approach aponeurosis and levator surgery, or the use of a buried non-absorbable suture for securing autogenous fascia lata used in a brow suspension operation. No book can cover all aspects of a subject, and this book has undoubtedly achieved the aim of being a simple, clear, illustrated guide to the main proptosis operations that any surgeon may want to use. It is without doubt the best guide to the subject that has yet been produced and cannot be warmly enough recommended to everybody who proposes to start ptosis surgery or who practises it infrequently and wants to refresh their memory prior to doing an operation.

J R O COLLIN


Edmund Speth, an ophthalmologist who practised general and ophthalmic plastic surgery in Philadelphia, USA, also wrote a surgical ophthalmic text which can now be seen as a precursor to that edited by his son George. The bond of his father and son team could have been said to have been handed over in 1971, when in a unique event in British ophthalmology both appeared on the same programme at the Oxford Congress.

Since that time Speth Jr has continued to delight his ophthalmic audiences with both his spoken and his written word. We in the audience have come to expect to be educated both in the science but also in (a much less frequently tilled pasture) the philosophy of our craft. One achievement has been to bring both these facets together in this comprehensive text. The first edition appeared in 1982, and now, eight years later, the second edition.

This book sets out to cover all aspects of the surgeon’s craft, from fundamental principles to basic elements of individual surgical procedures. In updating it the editor has kept the book at the same length as the first edition. New sections have been added on keratoectomie and laser surgery, while others have been either updated or extensively rewritten. It is copiously illustrated by means of black-and-white photographs and line drawings. Compactness of the text is maintained by means of many of the tables. Finally, chapters are concluded with (largely) up to date references.

The book represents the current practices of the American writers of the text. Even in a rapidly shrinking world some differences in practice still remain between colleagues on the American continent and elsewhere. The British reader should bear in mind that not all the possible approaches to a problem are necessarily listed, but can be found in those that will be tried and tested and actually work. The book is affordable at today's prices, practical, and a worthwhile addition to every ophthalmologist's bookshelf.

R HITCHINGS

NOTES

Fourth Eupo course

The fourth Eupo course (European Community Professors of Ophthalmology) will be held in Turin on 20–22 June 1991. Details from Organising Secretariat, CIC Srl, C.so Stati Uniti 3, 10128 Turino, Italy.

Duke spring symposium

The Duke Eye Center Spring Symposium will be held on 16–18 May 1991 at the Omni Durham Hotel and Convention Center, Durham, North Carolina, USA. Further information from George Andrews, Duke Eye Center, Box 3802, Durham, NC 27710, USA.