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Editorials

Diabetic tractional papillopathy: a new (and true) nosological entity?

Partial restoration of vision was achieved following vitrectomy in 15 of 17 eyes, ostensibly through removal of diabetic fibrovascular proliferations from the nasal part of the optic disc and relief of vitreopapillary traction which, for between 6 months and 6 years, had caused a reversible functional impairment of the papillomacular bundle via stretching and kinking of ganglion cell axons and additional or consecutive effects on their prelaminar blood supply. Eyes with such features (that is, with traction primarily localised nasally on the disc and unaccountably affecting acuity without any associated disturbance of the central visual field) should be subjected to early vitrectomy in order to prevent irreversible long term damage to central vision. This is the recommendation of Kroll and colleagues in a report which is published in this issue of the *BJO* (p 261) and which merits the careful attention of all ophthalmologists involved in the management of diabetic eye disease. Indeed, some will already be asking—if this entity is so common, how have I failed to recognise it for all these years?

Diabetic papillopathy (that is, disc swelling without any tractional component) has proved difficult to characterise clinically[1]; similarly, defining the precise physiological basis of visual loss in eyes with ischaemic/proliferative diabetic retinopathy is frequently problematic. Biomicroscopic signs of subtle but visually significant vitreomacular traction, for example, may be hard to elicit given the difficulties in detecting the consequent minor distortions of the transparent outer retina or the intraretinal disruption representing tractional schisis; reports of visual benefit and reversal of macular oedema after vitreous detachment[2] or after vitrectomy and peeling of the posterior hyaloid membrane[3–5] may bear witness to a "trampoline effect" across the macula in some eyes and may obviate the need to invoke alternative mechanisms of recovery such as physiological "vitreoperfusion".[6 7] Fluorescein angiography may also be less than definitive in diabetic eyes since good correlation between the extent of enlargement of the foveal avascular zone and the deficit in vision is lacking.[8] Attributing visual loss unreservedly to diabetic tractional papillopathy, then, is no mean task and any criticism of the achievements of Kroll and colleagues must take this into account. Given that the changes in the appearance of the optic disc after surgery may not be especially informative,[9] their case rests largely on the extent to which other mechanisms of visual benefit from surgery were excluded and on electrophysiological data. Regarding the former, scrutiny of their exemplar fluorescein study fails to inspire confidence since there are clear signs of macular retinal traction and perifoveal dye leakage in the preoperative illustration notwithstanding their stated exclusion criteria. Furthermore, removal of mild to moderate vitreous haemorrhage (that is, haemorrhage of less severity than the level warranting exclusion from their study) may well have had a significant influence on the visual benefit deriving from surgery in some eyes; it would have been reassuring if parallel "control" electrophysiological studies had been undertaken in eyes undergoing vitrectomy for mild to moderate vitreous haemorrhage wherein no vitreopapillary traction was evident. More information on the state of the vitreous in these eyes would also have been valuable—was the vitreous attached or detached from the retina?

Electrophysiological data were lacking in six eyes, restricted to visually evoked potentials (VEPs) elicited by flash in eight eyes, and derived from pattern stimulation in only three of the 17 eyes studied by Kroll and colleagues. Contrary to their statement, only a small minority (that is, not "most") of the 17 eyes met their own evaluation criteria that significant increases in acuity and VEP amplitude, together with a decrease in VEP latency, resulted from surgery—though this standard may be regarded as too stringent. There was no apparent correlation between visual benefit from vitrectomy and improved VEP variables; indeed, the two eyes with the greatest rise in VEP amplitude (and the highest absolute VEP amplitude values postoperatively) enjoyed minimal visual improvement (and had continuing poor vision) after surgery. However, it may well be that changes in VEP latency are more reliable than VEP amplitude changes,[10 11] while more use of comparative studies between the operated eye and the fellow eye (the latter always showing a normal disc and flat retina in their series) might have been helpful in defining normative VEP values in these patients, in controlling for the influence of blood glucose changes,[10] and in demonstrating repeatability independent of any surgical effect. Extending the range of investigational modalities[12] might also have provided more insight into the nature of the residual visual defect seen in the operated eyes.

When contemplating vitreous surgery for proliferative diabetic retinopathy, most attention has so far been focused on vitreomacular traction and there is reason to hope that techniques for better defining retinal structural pathology[13] will aid clinical evaluation in future. There is

little doubt, however, that ganglion cell axons in severe diabetic eye disease are potentially subject to a variety of metabolic and ischaemic insults; an additional putative mechanical factor is one we surely should not ignore, especially one operating at a point of known vulnerability of axoplasmic transport.[14] Whether Kroll and colleagues have provided incontrovertible evidence of such mechanical effects through their report is open to debate; more precisely specified and expressed studies are needed. In the meantime, further extension of the indications for vitrectomy in proliferative diabetic retinopathy should proceed with caution. For example, many eyes with nasal ectopia of the macular neuroretina and underlying pigment epithelium (as a result of fibrovascular contraction nasal to the optic disc) retain excellent visual acuity—perhaps the full thickness retinal ectopia is protective against nerve fibre stretching and visual loss in such eyes.

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Paediatric pseudophakia—choosing the implant power

Posterior chamber lens implantation is increasingly employed in the management of paediatric cataract, sometimes in infants within a few weeks of birth. The need to implant a lens of fixed power into an eye that is still growing creates a problem—what power lens should be selected? This is especially a dilemma in infants under 18 months of age, the period of most rapid postnatal ocular growth, during which there can be a substantial myopic shift.

Using hand held instruments, reliable keratometry and axial length measurements can be obtained in children, and implant power calculations using adult formulas are accurate in the paediatric eye.[1] However, implanting a lens at the calculated power risks significant myopia at ocular maturity. The paediatric cataract surgeon therefore now requires an additional "formula", one that will predict the final refraction for any selected lens on the basis of the patient's age.

An initial suggestion was to utilise a cross sectional study of the refractive data in a normal population.[2] The objection to this approach is that the pseudophakic eye is not normal in a variety of ways and may therefore grow differently. For example, the cataract may have been one manifestation of a wider ocular abnormality affecting growth. Given its important prenatal role in ocular development, loss of the crystalline lens might also have an effect, and there is evidence that both amblyopia[3] and the physical properties of the implant[4] may alter growth in axial length. As happened for adult intraocular lens power calculations, the most accurate formula is likely to derive from a study of outcome, in this case of children followed up until growth of the eye is complete. This requires many years of follow up and adequate data are not yet available.

Long term follow up data are, however, already available for aphakic paediatric eyes and have been used to derive a theoretical model that surgeons could use to predict future refraction at any age, and to develop a computer program to make the required calculations.[5] This is an important step and in the future we can expect such a program will become an integral part of the software in standard equipment for calculating implant power, but it will be based on pseudophakic not aphakic follow up data.

For the present, the requirement is for the accumulation of data on the refractive changes in pseudophakic eyes and a study by Flitcroft and colleagues in this month's *BJO* (p 265) makes a useful contribution to that process. The authors carried out a prospective observational study of changes in refractive power, keratometry, and axial length in 35 pseudophakic eyes.

An admitted problem is that follow up for many patients was relatively short, a feature of most of the studies in this field. It does cover the period when the eye is making its most rapid growth, and during this time the pseudophakic eyes studied showed axial elongation similar to that found in normal eyes. A further report when these eyes have reached maturity will be required to confirm the authors' initial conclusions and advice on implant power selection. In the interim it should be noted that the predictions of final refraction in the younger children are based on the presumption of continuing normal growth rather than actual follow up.

Unlike some previous studies, Flitcroft *et al* do provide detailed information for each patient and this is important. As the numbers in individual series will never be large, the most reliable formula is likely to be derived by meta-analysis. In addition, other factors affecting myopic shift may be isolated—for example, amblyopia and the initial postoperative refraction.[3] Publication of full data for all studies should enable earlier assessment of the impact of such variables.

Information from animal and human studies confirms the existence of visually directed control of growth in the axial length of the eye, but its mechanism remains unknown.[6 7] With an implant of fixed power and good

vision it is possible to speculate that the control system might reduce the normal increase in axial length, and so lessen the problem of myopic overcorrection.[8] This would be a serendipitous phenomenon to enjoy, and it may have occurred in some cases.[3–11] In the longer term, there is the possibility that when we understand the control of postnatal growth of the eye we may learn to manipulate it, to reduce the refractive problems inherent in implants of fixed power.

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