

This case suggests that a similar phenomenon can occur following PRK. The main reason for the error, as with RK, probably relates to erroneous keratometry readings. PRK involves the excimer laser ablation of a disc of superficial corneal tissue. To correct myopia more tissue is removed from the centre than the periphery. As a result the ablation zone becomes aspherical causing the keratometry power to be higher than that of the central cornea.

Miscalculations of IOL power may also result from the unknown refractive effect of the abnormally distributed tear film, and from the use of an incorrect estimation of corneal refractive index in the corneal radius to power conversion. One suggestion has been to use a higher refractive index for PRK corneas,³ however, this would result in a lower predicted IOL power and hence an even greater hypermetropic error following IOL implantation. Axial length measurement is a well-known source of error, particularly in myopic eyes. In the case described biometry was performed twice pre-operatively in order to reduce the risk of error; we also checked the axial length after the cataract surgery and found it to be consistent.

Several approaches can be adopted in post-PRK patients to minimise the risk of such problems occurring. Firstly, as suggested by Koch *et al.*² a more accurate keratometry power may be derived by subtracting the refractive change induced by PRK from the pre-PRK readings. Two other reports of cataract extraction after myopic refractive procedures^{4,5} address the question of IOL calculation. In the first, the authors observed a successful outcome after using post-PRK keratometry values and the SRK/T formula.

Secondly, videokeratography can be used to measure corneal power. This technique may be more helpful than keratometry because it can take measurements from the flatter central area of cornea nearer the visual axis.⁶ However, its accuracy in such cases is not known. Thirdly, it has been suggested that some of the more recently devised theoretical formulas (e.g. Hoffer Q, Holladay and SRK/T) are more accurate than the regression formulas in eyes with flatter corneas.⁷

The ideal approach in such patients may be to use the highest IOL power predicted by all the techniques described above. It would be helpful if keratometry or topography readings were routinely obtained prior to PRK and were made available for subsequent cataract extraction and IOL implantation.

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Andrew H.C. Morris ✉
Karl W. Whittaker
Robert J. Morris
Southampton Eye Unit
Southampton General Hospital
Tremona Road
Southampton SO16 6YD
UK

Melanie C. Corbett
Kings College Hospital
Denmark Hill
London SE5 9RS
UK

Sir,

Nasal epipapillary membrane causing visual field loss following macular hole surgery: Does it throw fresh light on the retinotopic arrangement of the nerve fibre layer?

Visual field defects in patients following vitrectomy for macular holes have been well reported.^{1–4} Characteristically the visual field defects are peripheral and temporal. Various suggestions have been offered to account for the mechanism and pattern of the visual field

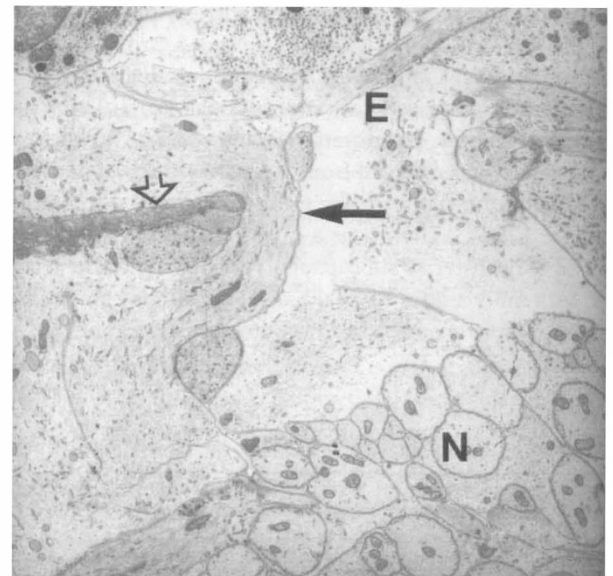


Fig. 1. A transmission electron micrograph of parapapillary retina. There is an epiretinal membrane (E) overlying the internal limiting lamina (open arrow) of the retina. A discontinuity in the internal limiting lamina is seen with a Müller cell process extending through the defect (filled arrow). N, Nerve fibre layer. (x5000)

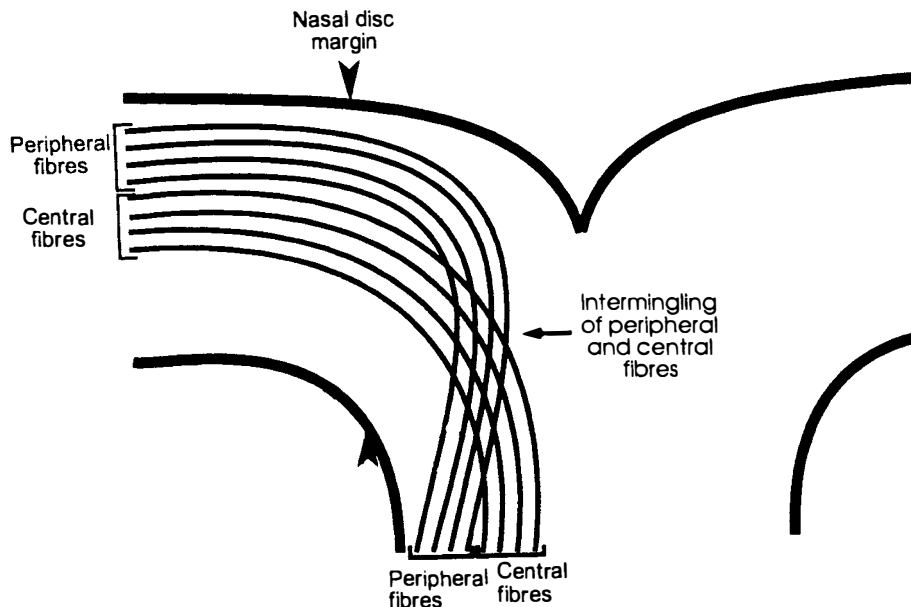


Fig. 2. A schematic diagram of the vertical retinotopic organisation of peripheral nerve fibres at the optic nerve head margin (arrowheads) to show the peripheral nerve fibres running from vitreal in the retina to scleral in the optic nerve.

loss.²⁻⁵ We believe that the significance for the mechanism and pattern of visual field loss is that it resolves a long-standing controversy on the anatomy of the optic nerve head.

For a long time it was purported by Minckler that the nerve fibres subserving the peripheral retina were external (scleral) to those subserving the central retina as they enter the optic nerve head.⁶ Ogden held the opposite viewpoint, i.e. peripheral fibres run internally and central fibres externally.⁷

We carried out a retrospective study of 37 patients who underwent macular hole surgery. We found an incidence of field defect in 7 of 30 consecutive patients (23%) undergoing macular hole surgery for stage II and III macular holes, and none in patients undergoing surgery for stage IV macular holes. Of the 7 patients with field defects, 3 were asymptomatic. All the defects were peripheral, temporal and wedge-shaped. This would localise the damage to the nasal part of the optic disc. The question remains as to why the nasal side of the optic disc is more susceptible to damage.

In a previous publication electroretinography studies showed that the photoreceptors in the retinal area corresponding to the visual defect area are intact.⁵ This suggests that the damage may be to the nerve fibre layer. Nerve fibre analysis has shown that there is indeed a reduction in nerve fibre layer thickness correlating with the visual field defects.⁸

A previous publication by Roth and Foos found an epipapillary membrane occurring in approximately 30% of autopsy eyes and in 90% of cases this membrane was nasal in location.⁹ The membranes were found to consist of one or more layers of glial cells on the surface of the internal limiting membrane connected via gaps in the internal limiting membrane to the underlying glial tissue. This arrangement leads indirectly to a strong adhesion

with the underlying nerve fibre layer. In Fig. 1 we demonstrate this with an electron micrograph of such a membrane obtained during surgery.

The presence of a nasal epipapillary membrane, its anatomical arrangement, its forced separation and its direct damage to the nerve fibre layer were proposed by us as the mechanism for temporal visual field defects following macular hole surgery. This was presented by us at the BEAVRS meeting in September 1996. Subsequently Dr G.A. Williams came to the same conclusion.¹⁰

If the proposed mechanism for visual field defects indeed holds true, it might resolve the long-standing controversy regarding the retinotopic arrangement of the nerve fibre layer (Fig. 2).

We believe that macular hole surgery has unwittingly brought about a human nerve transection study that finally resolves the controversy on the anatomical arrangement of nerve fibres at the optic nerve head. The pattern of visual field loss indicates that the fibres subserving the peripheral field lies more central or vitreal than the fibres subserving the more central field.

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- C.P. Groenewald ✉
D. Wong
I. Pearce
P. Hiscott
I. Grierson
St Paul's Eye Unit
Royal Liverpool University Hospital
Prescot Street
Liverpool L7 8XP, UK
- Tel: +44 (0)151 706 2000
Fax: +44 (0)151 706 5861
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