

AUTHOR REPLY

WE THANK DRS AREVALO AND AZAR-AREVALO FOR THEIR letter on our paper about the appearance of retinal detachments in myopic eyes after laser in situ keratomileusis. In that letter the authors referred to their experience regarding "the incidence and characteristics of rhegmatogenous retinal detachment after laser in situ keratomileusis." They referred to a low incidence of retinal detachment (0.06%) with a follow-up similar to ours (36 months vs 34.00 ± 14.78 months; range, 3 to 48 months vs 16 to 48 months) in a group of myopic patients with a spherical equivalent of -1.50 to -16.00 diopters. In our experience,¹ although the appearance of retinal detachment is an infrequent complication, the incidence has been slightly higher (0.25%). We have not found a localized preponderance of tears in the superior temporal quadrant (in fact, none of our four cases had the tear or hole in that quadrant). However, we find their observation that "the surgical microkeratome used in laser in situ keratomileusis to create the corneal flap has a temporal handle that may be responsible for extra pressure on that side of the eye" suggests an area of study in patients with myopia that has been corrected by laser in situ keratomileusis, with a long-term followup.

We agree with the decision to treat some of the selected cases by pneumatic retinopexy if there is no evident vitreous traction. Nevertheless, we do not agree to using vitrectomy as the primary choice to treat retinal detachment in these patients because of the appearance of cataract as a complication induced by this technique.² We know that the use of scleral buckling induces an undesirable myopic shift^{1,3} in patients who had previously been operated on to correct their myopia, but that small increase in the myopic spherical equivalent (-0.58 ± 0.72 vs -2.25 ± 1.1) could be corrected by laser in situ keratomileusis retreatment.⁴

Before the performance of laser in situ keratomileusis and after a careful examination of the peripheral retina, we have treated areas of lattice degeneration, atrophic holes, or flap tears by photocoagulation in all cases. This approach has been the same as that used in severely myopic patients who undergo correction with phakic anterior chamber lenses,³ as well as that of other authors, who corrected the severe myopia by clear lens extraction.⁵ These three studies^{1,3,5} have concluded that the prophylactic treatment is of doubtful efficacy. We do not know what would have been the incidence of retinal detachment in this group of patients with predisposing lesions in the peripheral retina if we had not treated them by photocoagulation, but it has been proved that the incidence of retinal detachment is higher than the incidence in cases without predisposing lesions. None-

theless, our recommendation is to treat all predisposing lesions and to warn patients of the risk.

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Compression of the Prechiasmatic Optic Nerve Produces a Junctional Scotoma

EDITOR:

KARANJIA AND JACOBSON (*AM J OPHTHALMOL* 128:256-258, August 1999) have reported a 55-year-old man with central visual loss in the left eye and a superior temporal hemianopia in the right eye from a pituitary adenoma. A magnetic resonance (MR) scan showed tumor compression of the left optic nerve and the optic chiasm. The authors felt that "the minor degree of optic chiasm distortion that existed in this patient would be unlikely to injure sufficient crossing nasal fibers and cause a temporal scotoma." For this reason, the authors suggested that the temporal scotoma in the right eye originated from compression of right optic nerve fibers entering the proximal portion of the left optic nerve (Wilbrand's knee). Although they acknowledged a study showing that Wilbrand's knee is an artefact found only after monocular enucleation,¹ they asserted that their case "supports the existence of Wilbrand fibers."

Magnanimously, Karanjia and Jacobson have provided me with complete copies of the MR scans of their patient. I have transferred the optic chiasm from the preoperative images onto the postoperative images to assess the compression and distortion caused by the tumor (Figure 1). Although the alignment is not perfect, because of technical differences between the two scans, the images show that the optic chiasm was elevated 5 mm to 7 mm by the tumor. More tellingly, the preoperative coronal image (A) shows thinning, widening, and bowing of the optic chiasm by the tumor. All of these changes resolved after surgery.

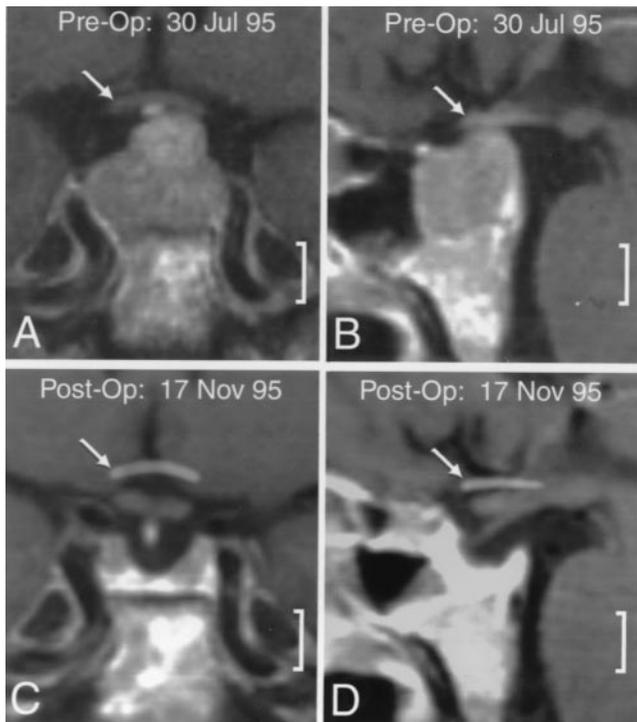


FIGURE 1. Coronal (A,C) and sagittal (B,D) postcontrast T₁-weighted MR images showing relief of optic chiasm compression by tumor removal. After aligning the images as closely as possible, I transferred the optic chiasm (arrows) from the preoperative images onto the postoperative images using Photoshop 5.0 (Adobe Systems, San Jose, California). It appears on the postoperative images as a white shadow (arrows), in a location corresponding to its preoperative position. This technique allows one to compare directly the position and shape of the optic chiasm before and after surgery. It was elevated and distorted considerably by the tumor. Scale = 1 cm.

From MR images it is always difficult to infer the degree of compression exerted by a tumor on surrounding structures. Nonetheless, these images appear to show considerable mass effect on the optic chiasm. No reason exists to assume, as Karanjia and Jacobson have done, that the tumor compressed the left optic nerve of the patient but spared his optic chiasm. Their patient had a routine pituitary adenoma, with a typical field defect produced by combined compression of the optic chiasm and one optic nerve.² There is no need to resurrect Wilbrand's knee to explain their case.

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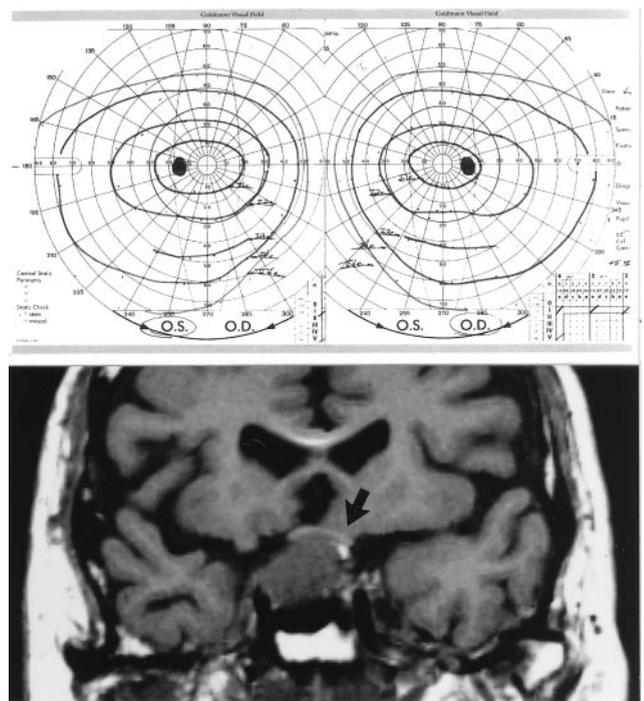


FIGURE 2. This 53-year-old man with a pituitary tumor and visual acuity of 20/20 in both eyes does not have any temporal visual field loss in either eye, despite the presence of considerably greater chiasm compression than existed in our case report. (Top) The Goldmann test targets (I1e, I2e, I3e) used to plot his visual field were the same targets that identified a central scotoma and contralateral superotemporal depression in our case report (see Figure 1 in our paper). (Bottom) Notice how the chiasm (black arrow) is thin, angulated over the dome of the tumor, and pinned against the gyrus rectus from above, which are radiographic signs indicative of much greater compression than existed in our Case Report (see Figure 2 in our paper).

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DR HORTON MAKES THE POINT, USING CLEVER SUPERIMPOSITION of preoperative and postoperative images, that the chiasm of our case was subject to considerable mass effect and, furthermore, that this mass effect was sufficient to cause injury of the crossing fibers of the chiasm to produce temporal visual field loss *in just one eye*. Despite his hypothesis, neither automated nor kinetic perimetry detected a *bitemporal* defect, the clinical sine qua non of chiasm injury. The small size of the central scotoma in the opposite eye of our patient would surely not "hide" such a defect if one truly existed. I acknowledge, however, that his figure does indeed suggest more chiasm elevation than we could appreciate by reviewing preoperative and postoperative images. As Dr Horton stated, the alignment of the corresponding images is not perfect. This imperfection should be considered when deciding how much weight to place on his rebuttal. For example, notice Figure