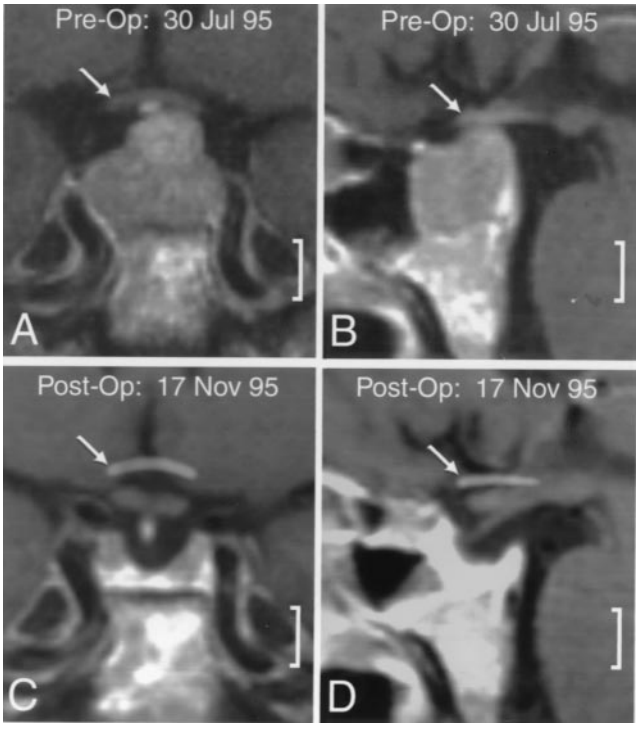

FIGURE 1. Coronal (A,C) and sagittal (B,D) postcontrast T_1-weighted MR images showing relief of optic chiasm compression by tumor removal. After aligning the images as closely as possible, I transferred the optic chiasm (arrows) from the preoperative images onto the postoperative images using Photoshop 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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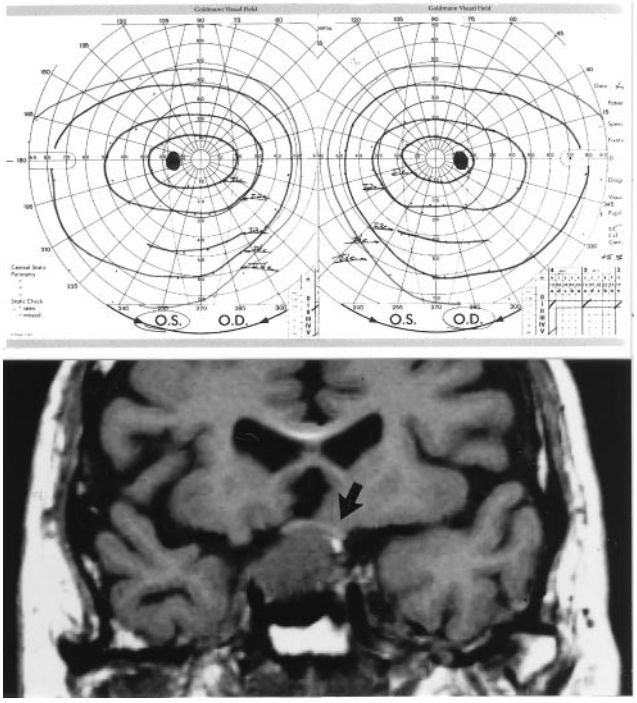

FIGURE 2. This 53-year-old man with a pituitary tumor and visual acuity of 20/20 in both eyes does not have any temporal visual field loss in either eye, despite the presence of considerably greater chiasmal compression than existed in our case report. (Top) The Goldmann test targets (I1e, I2e, I3e) 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).

AUTHOR REPLY

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 chiasmal 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 chiasmal 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

1 how A is at the level of the chiasmal bar and C is in front of the chiasm at a point where the two optic nerves still appear separate from each other.

How much anatomical distortion of the chiasm by a pituitary tumor in contact with it predicts the presence of bitemporal hemianopia? Although a number of variables (for example, rate of growth) influence this development, bitemporal hemianopia is generally not seen until marked compression of the chiasm is present, more than the amount evident in our case (Figure 2).

Finally, we were careful to specify in the last paragraph that our case "does not prove the existence of Wilbrand knee fibers. . . ." Instead, our point was a clinical one, as we implied in the last sentence of our paper. A central and contralateral superotemporal scotoma (that is, junctional scotoma) can result from, not only large tumors that compress both the chiasm and an optic nerve, but also focal lesions of the prechiasmatic optic nerve. Figure 2 of our report shows just that.

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