

These two patients illustrate the potential for serious central nervous system side effects when topical brimonidine is used in infants. The lipophilic nature of the drug allows access to the central nervous system to act as an agonist for the alpha-2 receptors. The precise mechanism of the central nervous system depression observed in our patients is not known. Currently, the only reported cases of severe central nervous system depression attributable to topical brimonidine have been in infants. Though fatigue and drowsiness have been described in adults using topical brimonidine,³ we have neither witnessed nor seen reported the profound central nervous system depression described in this report in adults. The reasons infants appear to be hypersensitive to the central nervous system depressive effects of the drug are unclear.

We agree with the manufacturer of commercially available topical brimonidine that this drug is not recommended for use in pediatric patients until its central nervous system depressive effects are better understood. We encourage the reporting of other similar adverse events in children.

REFERENCES

1. Walters TR. Development and use of brimonidine in treating acute and chronic elevations of intraocular pressure: a review of safety, efficacy, dose response, and dosing studies. *Surv Ophthalmol* 1996;41(suppl):19–26.
2. Juzych MS, Robin AL, Novack GD. Alpha-2 agonists in glaucoma therapy. In: Zimmerman TJ, Kooner KS, Sharir M, Fechtner RD, editors. *Textbook of ocular pharmacology*. Philadelphia: Lippincott-Raven, 1997:247–254.
3. Derick RJ, Robin AL, Walters TR, et al. Brimonidine tartrate: a one-month dose response study. *Ophthalmology* 1997;104:131–136.

Compression of the Prechiasmatic Optic Nerve Produces a Junctional Scotoma

Navaz Karanjia and Daniel M. Jacobson, MD

PURPOSE: To demonstrate the clinical-radiologic correlation between a junctional scotoma and a focal lesion compressing the prechiasmatic segment of the distal optic nerve.

METHODS: Case report involving a man with a pituitary adenoma. Clinical correlation was determined by reviewing visual field evaluations and magnetic resonance images.

Accepted for publication Feb 18, 1999.

From Harvard University, Boston, Massachusetts (N.K.), and the Departments of Neurology and Ophthalmology, Marshfield Clinic, Marshfield, Wisconsin (D.M.J.).

Inquiries to Daniel M. Jacobson, MD, Neuro-ophthalmology (4F-2), Marshfield Clinic, 1000 North Oak Ave, Marshfield, WI 54449; fax: (715) 387-5727; e-mail: jacobsond@mfdclin.edu

RESULTS: The tumor compressed the prechiasmatic segment of the distal optic nerve but not the optic chiasm, producing a junctional scotoma documented by Goldmann and automated perimetry. The visual field defect resolved after neurosurgical decompression of the anterior visual pathway.

CONCLUSIONS: A junctional scotoma can be caused by focal as well as large and diffuse lesions injuring the anterior visual pathway, specifically at the junction of the optic nerve and chiasm. This finding supports the existence of Wilbrand fibers. (*Am J Ophthalmol* 1999; 128:256–258. © 1999 by Elsevier Science Inc. All rights reserved.)

INJURY OF THE ANTERIOR OPTIC CHIASM MAY PRODUCE A characteristic pattern of visual field loss, consisting of a central scotoma in one eye and a superotemporal defect in the other, commonly referred to as a “junctional scotoma.” The conventional explanation for this sign is that the central scotoma is caused by injury of the ipsilateral optic nerve, whereas the superotemporal defect is attributed to damage to Wilbrand knee, fibers from the contralateral inferonasal retina that project a short distance into the prechiasmatic segment of the optic nerve.¹ This sign is distinct from the monocular hemianopia resulting from injury to the segregated nasal or temporal retinal ganglion cell axons coursing through the optic nerve just anterior to their entry into the chiasm, the “junction scotomata of Traquair.”² Lesions that produce a junctional scotoma are typically large parasellar tumors or aneurysms that cause diffuse injury by compressing both the optic nerve, as well as the optic chiasm.³

Recently, Horton⁴ questioned the existence of Wilbrand knee fibers by demonstrating that forward-looping fibers from the contralateral optic nerve of monkeys are merely artifacts that develop as an optic nerve becomes atrophic after enucleation. Identification of Wilbrand knee fibers in humans has only been accomplished at postmortem examination of subjects who had undergone enucleation many years earlier.^{4,5} One hypothesis of how a junctional scotoma can occur in the absence of Wilbrand fibers is that it results from the superimposed visual field defects caused by simultaneous injury of the optic nerve and optic chiasm by a diffuse mass lesion.⁴ Reports supposedly providing clinical-radiologic confirmation of the existence of Wilbrand knee are incomplete because of the lack of detailed imaging, unsubstantiated clinical evidence, or presence of large mass lesions that compressed both the optic nerve and chiasm. We offer the following clinical-radiologic correlation of a junctional scotoma produced by a focal lesion at the prechiasmatic segment of the distal optic nerve.

A 55-year-old man had visual loss in his left eye for 1 week. His visual acuity was RE: 20/20 and LE: 20/60 –1. Color vision was normal in the right eye, but the left eye

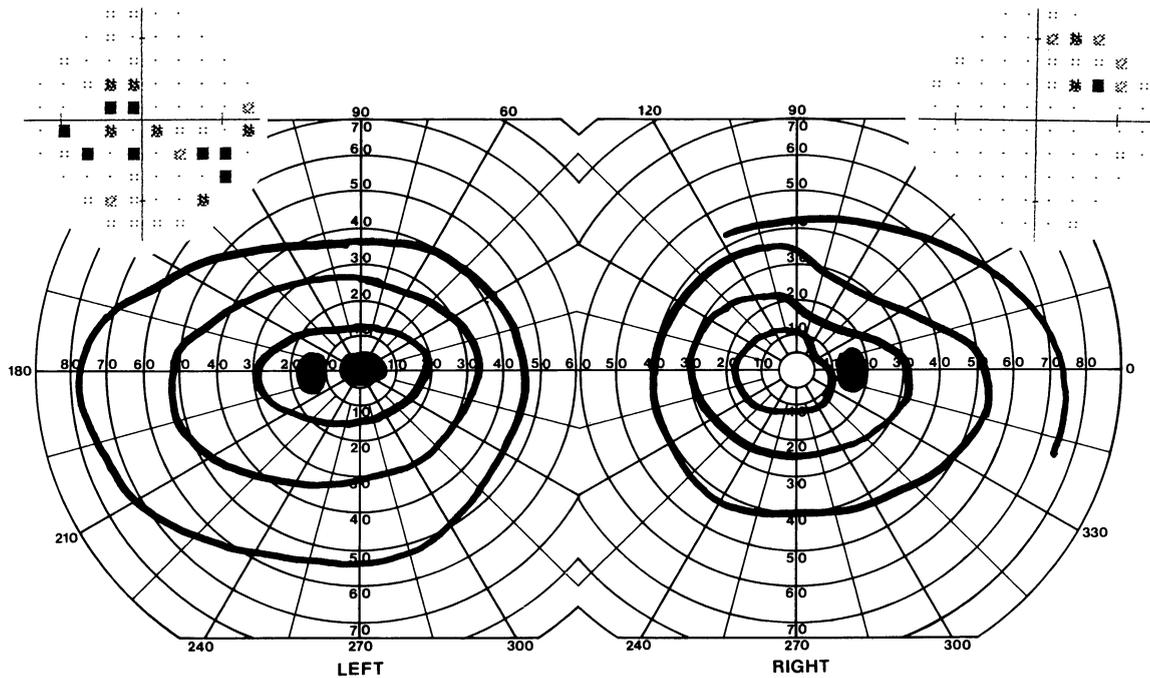


FIGURE 1. Visual fields from the Goldmann perimeter and Humphrey Automated Visual Field Analyzer Program 30-2 (Humphrey-Zeiss, San Leandro, California) (insets). Note the central scotoma in the patient's left eye along with the superotemporal depression in his right eye. Isopters shown in the right eye include I1e, I2e, I3e, and I4e. Isopters shown in the left eye include I2e, I3e, and I4e; the central scotoma is deep to the I2e test target. The pattern deviations of the automated test results are 4.19 db for the right eye and 4.89 db for the left eye.

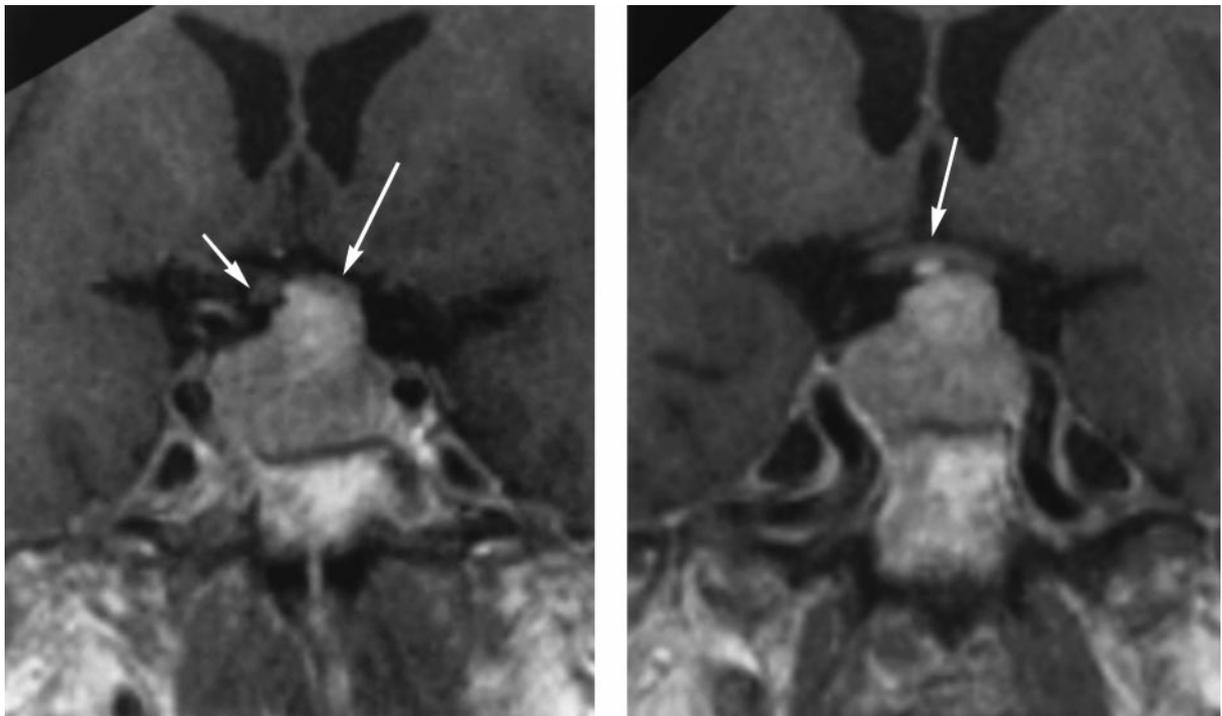


FIGURE 2. Postcontrast, T_1 -weighted (TR = 650 mseconds, TE = 14 mseconds) magnetic resonance images using a slice thickness of 3 mm. (Left) Coronal image one section in front of the optic chiasm showing tumor compressing the prechiasmatic segment of the left optic nerve (long arrow) but not the right optic nerve (short arrow). (Right) Coronal image at the level of the optic chiasm showing minimal rostral displacement (arrow) but without notable direct mass effect.

had mild dyschromatopsia. A 0.5 log unit relative afferent pupillary defect was detected in his left eye. Goldmann and automated perimetry showed a depression in the supero-temporal quadrant of his right eye and a central scotoma in his left eye (Figure 1). Magnetic resonance imaging demonstrated an enhancing intrasellar mass that extended upward to the level of the junction of the left optic nerve and chiasm (Figure 2). Serum assays for hormonal hypersecretion were negative. One month after transphenoidal resection of the pituitary adenoma, his visual acuity was LE: 20/20 +2, color vision was normal, and no relative afferent pupillary defect was detected. Goldmann and automated perimetry showed resolution of the central and junctional defects. Magnetic resonance imaging showed that the optic nerve was completely decompressed.

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. Rather, its location and configuration, along with the contralateral optic neuropathy and central scotoma, were consistent with a junctional scotoma. Although this clinical-radio-

logic correlation does not prove the existence of Wilbrand knee fibers, it illustrates that a junctional scotoma may be caused by a focal lesion at the distal (prechiasmatic) segment of the optic nerve, as one would predict if Wilbrand knee fibers were present. A junctional scotoma may be a sign of focal and diffuse injury of this region and may have exquisite localizing value.

REFERENCES

1. Wilbrand H. Schema des verlaufs der sehnervenfasern durch das chiasma. *Zeitschrift für Augenheilkunde* 1926;59:135–144.
2. Traquair HM. The interpretation of changes in the visual fields. In: *An introduction to clinical perimetry*. St Louis: CV Mosby Co, 1927:58–77.
3. Bird AC. Field loss due to lesions at the anterior angle of the chiasm. *Proc Roy Soc Med* 1972;65:519–520.
4. Horton JC. Wilbrand's knee of the primate optic chiasm is an artifact of monocular enucleation. *Trans Am Ophthalmol Soc* 1997;95:579–609.
5. Breen LA, Quagliari FC, Schochet SS. Neuro-anatomical feature photo. *J Clin Neuro-ophthalmol* 1983;3:283–284.