dazed at the scene and required assistance with ambulation immediately after the injury. The patient complained of complete right upper lid ptosis. He did not complain of weakness or uncoordination.

Examination revealed corrected visual acuities of 20/20 OU. He had ecchymosis of the medial right lower lid without skin laceration. There was complete right upper lid ptosis. He had limitation of supraduction, adduction, and abduction of the right eye. His left eye moved normally. The right pupil was fixed and dilated and there was no relative afferent pupillary defect. A subconjunctival hemorrhage was found on the inferior right globe without obvious laceration. There was a slight left hemiparesis with mild dysmetria of the left side.

While a computed tomographic scan revealed no fractures or abnormalities of the retrobulbar structures, a magnetic resonance image showed a hemorrhagic lesion of the right upper pons that extended to the cerebellar vermis (Figure 3). Cerebral angiography did not show any abnormalities.

A follow-up examination 2 months later revealed no residual hemiparesis and significantly less ptosis and diplopia.

Comment. Previous investigators have found that intracranial penetrating injuries commonly occur via orbital roof fractures, the superior orbital fissure, or the optic foramen. None of our patients had obvious orbital fractures on computed tomographic scanning. Also, none of our patients suffered a visual field defect or relative afferent pupillary defect, which made entry through the optic foramen unlikely. The trajectory through the orbital apex, via the superior orbital fissure, points straight to the upper brainstem. A thin object entering in this direction would pass over the top of the clivus as it reaches the posterior fossa. The exact location of the entry wound is important in pathogenesis because lesions through the upper eyelid or above the globe angled through the posterior orbit would strike a location more inferior in the brainstem, which occurred in our second case.

None of our patients suffered serious globe injury. Furthermore, it is remarkable that none of our patients suffered a vascular injury to the internal carotid, basilar, or posterior cerebral arteries and that none of them suffered from delayed meningitis.

Although not unheard of, penetrating injury to the brainstem through the orbit is a rare occurrence and is often fatal. We recommend obtaining magnetic resonance imaging of the brain and intracranial vasculature in patients who develop diplopia, even after a minor injury to the region around the orbit. The lack of a significant entrance wound in the eyelid or conjunctiva should not dissuade imaging because external injuries may be very subtle, as was the case with our third subject. Patients should be observed closely for the appearance of subsequent meningitis. If patients survive the initial injury, then their prognosis is good for a near-complete recovery.

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Pharmacologic Treatment of Congenital Nystagmus

Pharmacologic treatment has been used in acquired nystagmus with mixed success. Treatments have included baclofen, sodium valproate, gabapentin, and memantine. However, in congenital nystagmus, little is known about the effect of drugs. We describe a patient with congenital nystagmus and corneal dystrophy who improved dramatically with gabapentin treatment.

Report of a Case. A 37-year-old man complained of difficulty crossing roads and reading since childhood due to his poor vision. The patient claimed that these symptoms were alleviated by the consumption of alcohol. He had no oscillopsia. He had congenital nystagmus from birth and was noted at the time to have bilateral corneal opacities. The left eye was amblyopic despite occlusion therapy, and a corneal graft had been performed 20 years previously. Histologic findings from the graft confirmed the diagnosis of congenital granular stromal corneal dystrophy.

The initial visual acuity was 20/80 OD, 20/600 OS, and 20/80 OU. He had a small esotropia in the left eye and a conjugate, horizontal, pendular, and jerk nystagmus. The null point was in pri-
Eye movement recorded with an infrared video pupil tracker (Eye-Link eye tracker; SensoMotoric Instruments, Berlin, Germany) confirmed a reduction in the amplitude of his nystagmus compared with pretreatment (Figure). Foveation time per second was estimated using the same criteria throughout (±2°/s position window and ±4°/s velocity window), and best-corrected visual acuity was predicted using the expanded nystagmus acuity function (NAF[X]) (www.omlab.org). The nystagmus was most pronounced on left gaze (eg, at 20° eccentricity). The peak-to-peak amplitudes and frequencies were measured as follows: 6.0° and 5.5 Hz before treatment and 1.5° and 6.0 Hz after treatment. In right gaze (20° eccentricity), the nystagmus was 3.5° and 3.0 Hz before treatment and 1.0° and 3.0 Hz after treatment. In primary position, the nystagmus was 2.0° and 3.0 Hz before treatment and 1.0° and 3.5 Hz after treatment. The foveation time per second increased from 0.12 second to 0.92 second in primary position, 0.008 second to 0.60 second in right gaze, and 0.027 second to 0.27 second in left gaze. Using the NAF[X] to predict best-corrected visual acuity, this corresponded to improvements from less than or equal to 20/50° to less than or equal to 20/25° in primary position, less than or equal to 20/240° to less than or equal to 20/25° in right gaze, and less than or equal to 20/180° to less than or equal to 20/85° in left gaze.

In the right (dominant) eye, the best-corrected visual acuity after gabapentin treatment was 20/40−3, whereas the predicted NAF[X] was 20/25−. The difference could be due to the corneal dystrophy or amblyopia acquired from early childhood nystagmus.

Comment. Gabapentin has been used in the treatment of acquired nystagmus in multiple sclerosis. It is thought to have several possible mechanisms of action, of which the most likely to be involved in nystagmus is its antiglutamatergic activity. Our study shows that gabapentin can reduce nystagmus in congenital nystagmus forms. This suggests that abnormalities in the glutamate and/or γ-aminobutyric acid system are involved in congenital nystagmus as well. Double-masked trials are needed to establish the effect of pharmacologic treatment on the various forms of congenital nystagmus.

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Figure. Horizontal eye movement recordings of the right and left eye during saccades of 10° and 20° to the left and right. A, Before treatment. B, After treatment.
Adduction on Attempted Abduction: The Opposite of Synergistic Divergence

Congenital fibrosis of the extraocular muscles (CFEOM) is a congenital ocular motility disorder that manifests as restrictive ophthalmoplegia with ptosis. Synergistic divergence (SD) is a deficit of adduction associated with simultaneous bilateral abduction on attempted gaze into the field of action of the affected medial rectus muscle. There has been no pathologic report of SD; however, magnetic resonance imaging showed that in 2 patients with CFEOM and SD, the oculomotor nerve was hypoplastic bilaterally and the abducens nerve was absent on the side exhibiting SD. To our knowledge, bilateral deficit of abduction associated with simultaneous bilateral adduction on attempted gaze into the field of action of the lateral rectus muscles, the counterpart of SD, has not been previously reported.

Report of a Case. A 39-year-old man was referred for the evaluation of ophthalmoplegia and ptosis since birth. He underwent a bilateral frontalis sling operation at age 29 years.

Figure 1. Axial magnetic resonance images of the normal control. A, Cisternal segments of the normal right and left abducens nerves (black single arrows) as a linear dark signal emerging from the pontomedullary sulcus. The normal acoustic nerve (white arrows) and facial nerve (double-stemmed arrow) are seen in the left cerebellopontine angle cistern. The mean diameter of the right and left acoustic nerves is 1.5 mm. B, Abducens nerves (arrows) course in a superior oblique direction at the level of the lower pons. C, The right and left abducens nerves (arrows) finally enter into the clivus. D, Cisternal segments of the normal right and left oculomotor nerves (arrows) are found coursing in an anterior lateral direction at the level of the lower midbrain. The oculomotor nerves appear thicker than the acoustic nerves. The mean diameter of the right and left oculomotor nerves is 2.1 mm.