
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° 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).³ 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 20/25 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 20/40³, 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.⁴ 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. Otherwise, medical and family histories were noncontributory.