# Monocular nasotemporal optokinetic asymmetry—unraveling the mystery

**Michael C. Brodsky, MD**

Pediatric ophthalmologists encounter a unique visuomotor phenomenon known as monocular nasotemporal optokinetic asymmetry (MNTA).[1,2] This condition is characterized by brisk nasalward and poor temporalward optokinetic responses under monocular conditions of viewing (Figure 1A-B). Babies with normal vision display this subcortical optokinetic bias within the first few months of life, until cortical binocular vision is firmly established.[1-3] When infantile strabismus precludes normal binocular development, however, this optokinetic asymmetry persists throughout life.[4-7] In this way, MNTA provides a diagnostic "footprint in the snow" of infantile strabismus. It also gives rise to latent nystagmus and underlies its unique association with infantile strabismus.[8-11]

What is the origin of MNTA, and how can we explain its unique association with infantile strabismus? MNTA is an ancestral visual reflex that is present in most lateral-eyed afoveate animals.[12,13] The afoveate retina in lateral-eyed animals, such as fish and rabbits, corresponds to the human nasal retina, because it views the ipsilateral (temporal) visual field. Its retinotectal pathways project contralaterally to the nucleus of the optic tract and the dorsal terminal nucleus of the accessory optic tract (NOT-DTN) of the midbrain,[14-17] which are sensitive to full-field motion and facilitate optokinetic stabilization of retinal slip during body movements. The subcortical circuitry for optokinetic asymmetry is shared by all vertebrates.[16]

During navigation, survival depends on distinguishing real-world motion from body motion.[15-17] To this end, the optokinetic system encodes full-field visual motion separately from labyrinthine signaling of head motion. MNTA enables the animal to see where it is going during both translational and rotational body movements.[15-17] Consider a fish with two eyes that each see the ipsilateral side of visual space. When it swims forward, it would be visually disruptive to have both eyes driven backward by optokinetic responses to radial optic flow as it passes stationary contours that move posteriorly relative to the body (Figure 1C). Because fish are buoyant, they may also need to use posterior optic flow to detect self-motion.[18] It is therefore evolutionarily advantageous to minimize temporalward optokinetic responses to a receding visual world.[15-18] As aquatic visibility can be low, fish swim closer to features that provide optokinetic input, enabling them to use temporal optic flow to judge self-motion.[18]

But this is only half the story. When the animal turns to one side, it needs to track the visual world that is moving nasally, because that is the area it is turning toward (Figure 1D). Because the nose and mouth are median (ie, nasal) to the eyes, accurate tracking of nasalward circumferential optic flow may help to stabilize the rapid visual motion of the approaching hemifield, as the animal rotates toward a salient nearby nutrient[17] to inspect it with the nose and ingest it with the mouth. At the same time, the opposite eye is seeing an area from which the animal is turning away, with objects likely at a further distance and therefore producing less retinal slip, so that the eye receiving nasalward visual input must dictate the optokinetic response for both eyes. So MNTA provides an ingenious evolutionary solution to enable the lateral-eyed animal to use optokinetic rotation to visually navigate both translation and rotation. In the zebrafish pretectum, monocularly and binocularly driven clusters of directionally selective horizontal motion detection cells are hierarchically organized to distinguish between translational and rotational optic flow.[19]

Ontegeny recapitulates phylogeny for primitive reflexes within the developing human visual system. In humans with infantile strabismus, the cortical optokinetic pathways reconfigure to the monocular subcortical template, so that MNTA can be modulated through the cortical pursuit centers as they establish ipsilateral connections to each NOT-DTN.[20-23] The resulting cortical pursuit asymmetry can now generate MNTA to foveated optokinetic targets.[24-26] In this setting, infantile esotropia confers the binocular advantage of restoring horizontal optokinetic bidirectionality in the presence of MNTA.[24] Horizontal visual motion can be followed in either direction simply by switching fixation to the eye receiving nasalward input.[27] In infantile esotropia, the human nasal retinas realign in the frontal plane but continue to function as they originally did in lateral-eyed afoveate animals. What an evolutionary wonder!

*Author affiliations: Departments of Ophthalmology and Neurology, Mayo Clinic, Rochester, Minnesota*

*Supported in part by a grant from the Knights Templar Eye Foundation.*
*Submitted April 1, 2019.*
*Revision accepted July 10, 2019.*
*Published online September 12, 2019.*

*Correspondence: Michael C. Brodsky, MD, Mayo Clinic, Department of Ophthalmology, 200 First St. SW, Rochester, MN 55905 (email: Brodsky.michael@mayo.edu).*

*J AAPOS 2019;23:249-251.*



*1091-8531/$36.00*
*https://doi.org/10.1016/j.jaapos.2019.07.001*

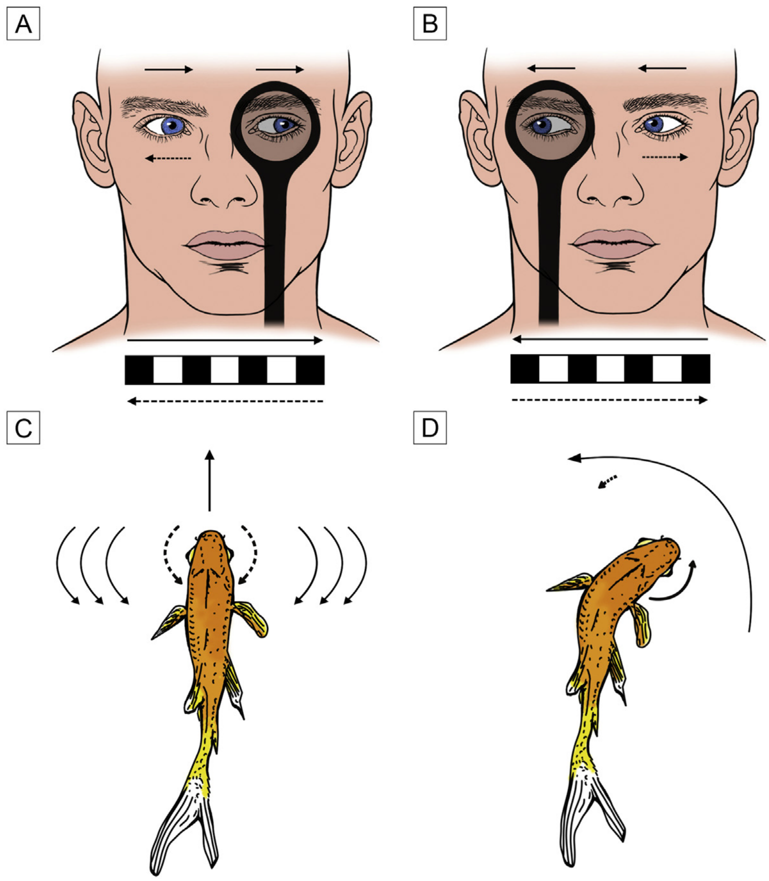


**FIG 1.** Monocular nasotemporal optokinetic asymmetry. A-B, In the human with infantile strabismus, monocular viewing results in strong nasalward optokinetic responses (solid lines) and poor temporalward optokinetic responses (dashed lines) relative the viewing eye. C, In a fish that is swimming forward, temporalward optokinetic responses (dashed lines) in response optic flow would rotate the eyes posteriorly and impede visualization of the oncoming visual scene. D, In a fish that is turning to the right, nasalward rotation of nearby contours in the right hemifield of visual space necessitates a strong and accurate optokinetic response in the right eye (solid curved line). As the fish turns away from the left hemifield of visual space, more distant visual contours seen by the left eye (dashed curved line) are moving at slower velocities; therefore, the nasalward visual input to the right eye dictates the optokinetic responses of both eyes.

## References


1. Atkinson J. Development of optokinetic nystagmus in the human infant and monkey infant: an analogue to development in kittens. In: Freeman R, ed. Developmental Neurobiology of Vision. New York: Plenum Press Nato Advanced Study Institute Series; 1979:277-87.
2. Naegele J, Held R. The post-natal development of monocular optokinetic nystagmus in infants. Vision Res 1982;22:341-6.
3. Flynn JT. Vestibulo-optokinetic interactions in strabismus. Am Orthop J 1982;32:36-47.
4. Mein J. The asymmetric optokinetic response. Br Orthop 1983;40:1-3.
5. Wright KW. Clinical optokinetic nystagmus asymmetry in treated esotropes. J Pediatr Ophthalmol Strabismus 1996;33:99-109.
6. Valmaggia C, Proudlock F, Gottlob I. Optokinetic nystagmus in strabismus: are asymmetries related to binocularity? Invest Ophthalmol Vis Sci 2003;44:5142-50.
7. Schor C, Fusaro R, Wilson N, McKee SP. Prediction of early-onset esotropia from components of the infantile squint syndrome. Invest Ophthalmol Vis Sci 1997;38:719-40.
8. Kommerell G. Ocular motor phenomena in infantile strabismus: asymmetry in optokinetic nystagmus and pursuit, latent nystagmus, and dissociated vertical divergence. In: Lennerstrand G, von Noorden GK, Campos EC, eds. Strabismus and Amblyopia: Experimental Basis for Advances in Clinical Management. London, England: Macmillan; 1988:99-109.
9. Kommerell G, Mehdorn E. Is an optokinetic defect the cause of congenital and latent nystagmus? In: Lennerstrand G, Zee DS, Keller EL, eds. Functional Basis of Ocular Motility Disorders. New York: Pergamon Press; 1982:159-67.
10. Schor CM. Subcortical binocular suppression affects the development of latent and optokinetic nystagmus. Am J Optom Physiol Opt 1983;60:481-502.
11. Brodsky MC. Latent nystagmus: Vestibular nystagmus with a twist. Arch Ophthalmol 2004;122:202-9.
12. Tauber ES, Atkin A. Optomotor responses to monocular stimulation. Relation to visual system organization. Science 1968;160:1365-7.
13. Masseck OA, Hoffmann KP. Comparative neurobiology of the optokinetic reflex. Ann NY Acad Sci 2009;1164:430-39.
14. Collewijn H, Noorduin H. Conjugate and disjunctive optokinetic movements in the rabbit, evoked by rotatory and translator motion. Pflügers Arch 1972;335:173-85.
15. Ohmi M, Howard IP, Everleigh B. Directional predominance in human optokinetic nystagmus. Exp Brain Res 1986;63:387-94.
16. Wallmann J. Subcortical optokinetic mechanisms. In: Miles FA, Wallman J, eds. Visual Motion and its Role in the Stabilization of Gaze. Amsterdam, The Netherlands: Elsevier Science; 1993:321-42.
17. Miles FA. The sensing of rotational and translational optic flow by the primate optokinetic system. In: Miles FA, Wallman J, eds. Visual

Motion and its Role in the Stabilization of Gaze. Amsterdam, The Netherlands: Elsevier Science; 1993:393-403.
18. Scholtyssek C, Dacke M, Kröger R, Baird E. Control of self-motion in dynamic fluids: fish do it differently from bees. Biol Lett 2014;10:20140279.
19. Kubo F, Hablitzel B, Dal Maschio M, et al. Functional architecture of an optic flow responsive area that drives horizontal eye movements and zebrafish. Neuron 2014;81:1344-59.
20. Hoffman KP. Control of the optokinetic reflex by the nucleus of the optic tract in primates. Prog Brain Res 1989;80:173-82.
21. Hoffmann K-P. Neural basis for optokinetic defects in experimental animals with strabismus. In: Kaufmann H, ed. Transactions of the 16th Meeting of the European Strabismological Association. Giessen, September 1987. Giessen, Germany: Gahmig Druck Giessen; 1987:35-6.
22. Distler C, Hoffmann K-P. Development of the optokinetic response in macaques: A comparison with cat and man. Ann NY Acad Sci 2003;1004:10-18.
23. Braddick O. Where is the naso-temporal asymmetry? Current Biol 1996;6:250-53.
24. Brodsky MC. Is infantile esotropia subcortical in origin? Prog Brain Research 2019;248:183-93.
25. Tychsen L, Hurtig RR, Scott WE. Pursuit is impaired but the vestibulo-ocular reflex is normal in infantile strabismus. Arch Ophthalmol 1985;103:536-9.
26. Joshi AC, Aqaoglu MN, Das V. Comparison of naso-temporal asymmetry during monocular smooth pursuit, optokinetic nystagmus, and ocular following response in strabismic monkeys. Strabismus 2017;25:47-55.
27. Brodsky MC, Klaehn LD. An optokinetic clue to the pathogenesis of crossed fixation in infantile esotropia. Ophthalmology 2017;124:272-3.

## CLINICAL PEARLS

### A possible clinical sign for orbital cellulitis

Distinguishing orbital from preseptal cellulitis is important for determining the best therapeutic course for the patient. Signs suggestive of orbital cellulitis include proptosis, strabismus, restriction of eye movement, diplopia, orbital pain, decreased vision, afferent pupillary defect, conjunctival injection, and chemosis. A sharp demarcation at the orbital rim may be a further sign of orbital cellulitis. We examined photographs from several ophthalmology units of individuals with orbital and preseptal cellulitis. Of 7 eyelids with orbital cellulitis, 4 had a sharp demarcation line running along the superior rim where the septum inserts. Only 1 of 17 eyelids with preseptal cellulitis had a demarcation line; the other 16 showed a diffuse pattern of erythema. The possible anatomic explanation for this finding is that preseptal inflammation due to some inflammatory process is not limited in the horizontal or vertical planes of the subcutaneous tissue by the septum and thus demonstrates a more diffuse erythema. By contrast, the septum prevents the spread of orbital inflammation superiorly, thus creating a sharp demarcation line. This distinction is also present in patients with an orbital hematoma, where the dark ecchymosis may have a sharp demarcation line where the septum prevents the posterior hematoma from spreading superiorly. In the Figure, on the left is a child with preseptal cellulitis and diffuse erythema; in the middle, a child with orbital cellulitis and a sharp demarcation line along the superior orbital rim; and on the right, a child with an orbital hematoma showing a sharp superior demarcation line.

*Contributed by Gonzalo Vike Vicente, Bethesda, Maryland; Kara M. Cavuoto, MD, Miami, Florida; Saul N. Rajak, PhD, FRCOphth, Sussex, United Kingdom; Lilly H. Wagner, MD, Rochester, Minnesota; and Linn M. Mangano, MD, Cincinnati, Ohio*