

Author Response: Motion Responses in Human Strabismus: What Optokinesis in the Deviating Eye Is Telling Us

We thank Michael Brodsky¹ for his interest in our study² and for drawing our attention to a paper by Brodsky and Klaehn³ that examined optokinetic responses in humans with infantile esotropia using what the authors labeled an “optokinetic uncover test.”³ We commend Brodsky and Klaehn on the observations and conclusions that they draw from their study. However, we would like to point out some key differences between their study and our study. A major difference is in the construction and delivery of the stimulus. Brodsky and Klaehn presented the patients with an optokinetic stimulus, first monocularly to the fixating eye and then binocularly. Further, the stimulus itself was full field, that is, not localized to any part of the visual field of the deviated eye. In our study, we utilized a dichoptic presentation in which the optokinetic stimulus is presented only to the deviated eye (never binocularly) and the fixating eye sees only a stationary target. Moreover, the optokinetic stimulation is restricted to a 10° patch, and we observed optokinetic nystagmus (OKN) only when the patch occupied the central 10° of the deviated eye that included the fovea. We also made quantitative evaluations of the strength of the OKN response and its gradation with spatial location and contrast of the OKN stimulus, whereas the Brodsky and Klaehn study was essentially a qualitative evaluation of the OKN response. Therefore, in our view, the two studies are not directly comparable. However, our results do agree with one of the conclusions that they reached, which was that information is being processed via both eyes.

In his letter, Brodsky does not question our results but perhaps suggests two additional discussion points with regard to our study: (1) The OKN responses that we observed are driven by a subcortical optokinetic circuit, that is, no cortical involvement, and (2) cortical suppression of the fovea of the deviating eye might still have been present because the OKN leaked through via the subcortical pathway. In response to his two points, we make the following arguments. (1) In strabismus, nasotemporal asymmetry is observed in motion detection, visually evoked potential (VEP) response, smooth pursuit, and OKN.^{4,5} Neurophysiological investigation in strabismic monkeys has shown loss of binocularity in cortical areas V1, MT, MST, and also in brainstem area nucleus of the optic tract (NOT).^{4,6-8} Therefore, as proposed by models in the literature,^{9,10} the loss of binocular connections in the pathway from V1 → MT → MST → NOT could lead to asymmetric visual or oculomotor response to monocular motion stimuli. Subcortical projections (direct retina-NOT projections) may also play a role, but it is not clear that they play an exclusive or primary role in generating nasalward OKN in strabismus. (2) Our study was focused on identifying areas of retinal suppression in strabismus, and OKN was simply used as a readout to identify suppressed versus unsuppressed retina. Our data and conclusions fit in nicely with the previous work in the literature. For instance, Economides, Adams, and Horton^{11,12} used a visual psychophysical paradigm, and showed that the fovea of the deviated eye was not suppressed. In addition, other work from our lab in which we used a saccade paradigm to examine spatial patterns of fixation switch behavior (presumably driven by suppression) also revealed similar results.¹³ Taken together with these other studies, our current study does indeed support the idea of lack of suppression of the fovea of the deviated eye in exotropia.

Finally, we would like to point out that the discussion points above are fundamentally speculative because neither Brodsky's previous work nor our current study directly examined neural responses, and therefore cannot ascribe the optokinetic responses to cortical/subcortical pathways. As always in science, the best way to settle the issue would be to design and perform an appropriate experiment and quantitatively evaluate the data.

Sevda Agaoglu
Mehmet N. Agaoglu
Vallabb E. Das

College of Optometry, University of Houston, Houston, Texas, United States.

E-mail: vdas@central.uh.edu

References

1. Brodsky MC. Motion responses in human strabismus: what optokinesis in the deviating eye is telling us. *Invest Ophthalmol Vis Sci.* 2016;57:2990.
2. Agaoglu S, Agaoglu MN, Das VE. Motion information via the nonfixating eye can drive optokinetic nystagmus in strabismus. *Invest Ophthalmol Vis Sci.* 2015;56:6423-6432.
3. Brodsky MC, Klaehn L. The optokinetic uncover test: a new insight into infantile esotropia. *JAMA Ophthalmol.* 2013;131:759-765.
4. Tychsen L. Causing and curing infantile esotropia in primates: the role of decorrelated binocular input (an American Ophthalmological Society thesis). *Trans Am Ophthalmol Soc.* 2007;105:564-593.
5. Bosworth RG, Birch EE. Direction-of-motion detection and motion VEP asymmetries in normal children and children with infantile esotropia. *Invest Ophthalmol Vis Sci.* 2007;48:5523-5531.
6. Tychsen L, Burkhalter A. Nasotemporal asymmetries in V1: ocular dominance columns of infant, adult, and strabismic macaque monkeys. *J Comp Neurol.* 1997;388:32-46.
7. Mustari MJ, Tusa RJ, Burrows AF, Fuchs AF, Livingston CA. Gaze-stabilizing deficits and latent nystagmus in monkeys with early-onset visual deprivation: role of the pretectal not. *J Neurophysiol.* 2001;86:662-675.
8. Kiorpes L, Walton PJ, O'Keefe LP, Movshon JA, Lisberger SG. Effects of early-onset artificial strabismus on pursuit eye movements and on neuronal responses in area MT of macaque monkeys. *J Neurosci.* 1996;16:6537-6553.
9. Tusa RJ, Mustari MJ, Burrows AF, Fuchs AF. Gaze-stabilizing deficits and latent nystagmus in monkeys with brief, early-onset visual deprivation: eye movement recordings. *J Neurophysiol.* 2001;86:651-661.
10. Tychsen L, Richards M, Wong A, Foeller P, Bradley D, Burkhalter A. The neural mechanism for latent (fusion maldevelopment) nystagmus. *J Neuroophthalmol.* 2010;30:276-283.
11. Economides JR, Adams DL, Horton JC. Perception via the deviated eye in strabismus. *J Neurosci.* 2012;32:10286-10295.
12. Economides JR, Adams DL, Horton JC. Eye choice for acquisition of targets in alternating strabismus. *J Neurosci.* 2014;34:14578-14588.
13. Agaoglu MN, LeSage SK, Joshi AC, Das VE. Spatial patterns of fixation in monkeys with strabismus. *Invest Ophthalmol Vis Sci.* 2014;55:1259-1268.

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