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SUBMISSION ROLE: Abstract Submission

## AUTHORS

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**Commercial Relationships Disclosure (Abstract):** Mehmet Agaoglu: Commercial Relationship: Code N (No Commercial Relationship) | Myhtri Pallela: Commercial Relationship: Code N (No Commercial Relationship) | Anand Joshi: Commercial Relationship: Code N (No Commercial Relationship) | Sevda Agaoglu: Commercial Relationship: Code N (No Commercial Relationship) | David Coats: Commercial Relationship: Code N (No Commercial Relationship) | Vallabh Das: Commercial Relationship: Code N (No Commercial Relationship)

### **Study Group:**

## ABSTRACT

**TITLE:** Surgical correction of strabismus in monkeys: III. Longitudinal evaluation of neuronal responses in the Abducens nucleus

### **ABSTRACT BODY:**

**Purpose:** Strabismus correction surgery is well documented in both the literature and in practice with varying levels of success and permanence. Potentially, muscle remodeling and/or central neural adaptation affects the final state of misalignment after treatment. Our goal was to assess central adaptation by examining Abducens neuron (ABN) responses in strabismic monkeys following surgical correction.

**Methods:** The study included one rhesus monkey with an exotropia (strabismus angle: OD: ~30°, OS: ~15°) that was induced in infancy using an optical prism-viewing paradigm. Surgical treatment when animal was ~6years old involved recession of the lateral rectus (LR) and resection of the medial rectus (MR) of the left eye only. We recorded from 75 ABNs prior to treatment and from 92 ABNs over the first 6 months following treatment. ABN firing rates (FR) and horizontal eye position and velocity acquired during a horizontal smooth pursuit task (0.3Hz, ±15°) were used to identify regression coefficients in a first-order model ( $FR = K * Epos + R * Evel + C$ ). K and C coefficients were then used to compute the population LR neuronal drive (ND) necessary to produce static deviation of the non-fixating eye before surgery (pre), <1 month after surgery (post1), ~6 months after surgery (post6).

**Results:** Strabismus angle (SA) was reduced by ~35% at post1. SA during OS view gradually increased back to its pre-surgery value while SA during OD view was still reduced by ~28% of its pre-surgery value at post6. Analysis of Left ABN cells showed that the ND to the LR of the treated left eye did not change at post1 although SA was reduced significantly (pre: 177±81 sp/s, post1: 173±79 sp/s). Analysis of Right ABN cells indicated that the ND to the LR of the untreated right eye was reduced at post1 (pre: 115±78 sp/s, post1: 80±57). At post6, the ND from Left ABN showed a significant drop (138±47 sp/s); the ND from Right ABN reverted to pre-surgery levels (107±26 sp/s).

**Conclusions:** The unchanged ND to the treated eye immediately after surgery suggests that alterations in muscle strength of the treated eye determined the improvement in strabismus angle; post1 reduction in ND to the untreated eye simply reflects Hering's law. The post6 changes in the NDs to both treated and untreated eyes suggest a significant role of neural adaptation in addition to muscle remodeling in setting the steady-state strabismus angle.  
(No Image Selected)

**Layman Abstract (optional):** Provide a 50-200 word description of your work that non-scientists can understand.

**Describe the big picture and the implications of your findings, not the study itself and the associated details.:**

Strabismus is often treated using surgical methods wherein the strategy is to alter strength of eye muscles (EM) to realign the eyes. However, the outcome of strabismus surgery is often not as predicted and patients undergo additional surgical procedures. Why does surgical treatment succeed fully in some patients, succeed partially in others

and fail in yet some others? One possibility is that there is plasticity that occurs following surgery that either 'helps' (i.e., improves upon) or 'fights' (i.e., counteracts) the effect of surgical manipulation of EM. The source of plasticity could be intrinsic to EM such as a change in contractility due to local muscle remodeling triggered by the surgical treatment. Alternatively, plasticity may involve a post-surgical change in neural drive to EM that may be triggered by the change in muscle strength due to surgery. The goal of our study was to investigate alterations in neural drive to horizontal EM by recording from abducens nucleus (an area in the brain which controls some of the eye muscles) in monkey models for strabismus before and after the correction surgery. Our data point to a significant role of neuronal adaptation in addition to muscle remodeling in setting the steady-state strabismus angle.

## **DETAILS**

**PRESENTATION TYPE:** Poster Only

**CURRENT REVIEWING CODE:** 3640 strabismus: basic mechanisms and animal models - EY

**CURRENT SECTION:** Eye Movements/Strabismus/Amblyopia/Neuro-ophthalmology

**KEYWORDS:** 725 strabismus: treatment, 525 eye movements: saccades and pursuits, 508 electrophysiology: non-clinical.

**Clinical Trial Registration (Abstract):** No

**Other Registry Site (Abstract):**

**Registration Number (Abstract):**

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**Grant Support (Abstract):** Yes

**Support Detail (Abstract):** NIH R01 EY022723; P30 EY07551

## **TRAVEL GRANTS and AWARDS APPLICATIONS**

**AWARDS:** ARVO 2015 Members-in-Training Outstanding Poster Award