Incidence of Nonarteritic Anterior Ischemic Optic Neuropathy After Cataract Surgery: A Systematic Review and Meta-Analysis: Response

We thank Gupta et al for sharing their insightful observations drawn from the random-effects meta-analysis of 2 earlier studies comparing 6-month incidence rates of postcataract surgery nonanterior ischemic optic neuropathy (pcsNAION) compared with spontaneous nonanterior ischemic optic neuropathy (sNAION).[1–3]

The predominance of retrobulbar anesthesia in pcsNAION (18 of 19 cases) was highlighted with cases from the cohort used in the study by Al-Madani et al,[2] which only used retrobulbar anesthesia and from that in the study by Mansukhani 2020, which used a mixture of anesthetic techniques.[3] It has been postulated that delayed hematoma expansion may contribute to the potential association between retrobulbar anesthesia administration and pcsNAION.[1,4] Our review also identified that the use of retrobulbar compared with topical anesthesia may have partially contributed to the discordant trends reported by 2 studies investigating the risk of pcsNAION in patients with previous sNAION in the fellow eye.[5–7] However, only half of pcsNAION cases using retrobulbar anesthesia in the cohorts of Al-Madani and Mansukhani occurred within the first week of surgery,[2,3] suggesting that other mechanisms may predominate in triggering NAION. As we have discussed, another potential mechanism for delayed psNAION may involve postoperative inflammation triggering vasogenic disk edema. However, it is acknowledged that the impact of the route of anesthetic administration cannot be reliably discerned in the context of other significant interstudy methodological heterogeneities and confounding factors, including the differing proportion of cases undergoing phacoemulsification vs extracapsular cataract extraction.[5–7] Overall, we agree with the broader point raised by Gupta et al of the plausible role of retrobulbar anesthesia in the pathogenesis of pcsNAION and the need for future studies to reliably document the type of anesthesia administered.

Gupta et al[1] also reported that pcsNAION cases exhibited significant fewer cumulative systemic factors compared with the sNAION group, which contrasted with the nonsignificant trends reported in our review.[5] However, we agree with their view that the development of pcsNAION is likely multifactorial, and perioperative factors might lower the threshold in patients with preexisting systemic risk factors.[1] We look forward to further elucidation of the pathogenic mechanisms of pcsNAION in future research.

William Shew, MBChB, BOptom (Hons), Michael T. M. Wang, MBChB, PhD, Helen V. Danesh-Meyer, MBChB, MD, PhD, FRANZCO

Department of Ophthalmology, New Zealand National Eye Centre, The University of Auckland, Auckland, New Zealand

The authors report no conflicts of interest.

REFERENCES

1. **Gupta A**, Tejpal T, Ahemd IIK, Sharma R. Incidence of nonarteritic anterior ischemic optic neuropathy after cataract surgery: a systematic review and meta-analysis. J Neuroophthalmol. 2023 May 31 (epub ahead of print).
2. **Al-Madani MV**, Al-Raqqad NK, Al-Fgarra NA, Al-Thawaby AM, Jaafar AA. The risk of ischemic optic neuropathy post phacoemulsification cataract surgery. Pan Afr Med J. 2017;28:53.
3. **Mansukhani SA**, Chen JJ, Fairbanks AM, et al. A population-based study of anterior ischemic optic neuropathy following cataract surgery. Am J Ophthalmol. 2021;222:157–165.
4. **Gross A**, Cestari DM. Optic neuropathy following retrobulbar injection: a review. Semin Ophthalmol. 2014;29:434–439.
5. **Shew W**, Wang MTM, Danesh-Meyer HV. Nonarteritic anterior ischemic optic neuropathy after cataract surgery: a systematic review and meta-analysis. J Neuroophthalmol. 2023;43:17–28.
6. **Lam BL**, Jabaly-Habib H, Al-Sheikh N, et al. Risk of non-arteritic anterior ischaemic optic neuropathy (NAION) after cataract extraction in the fellow eye of patients with prior unilateral NAION. Br J Ophthalmol. 2007;91:585–587.
7. **Moradi A**, Kanagalingam S, Diener-West M, Miller NR. Post-cataract surgery optic neuropathy: prevalence, incidence, temporal relationship, and fellow eye involvement. Am J Ophthalmol. 2017;175:183–193.

Comments on: Partial Recovery of Amblyopia After Fellow Eye Ischemic Optic Neuropathy

I read with great interest the article entitled "Partial recovery of amblyopia after fellow eye ischemic optic neuropathy" by Resnick et al[1] The authors reported visual recovery in amblyopic eyes following ischemic optic neuropathy (ION) in the fellow eye.

Recovery from blindness is very rare. A few studies have shown significant visual improvement in the blind eye only after losing vision in the better eye.[1–3] Still, the underlying mechanisms are unknown.

I would like to share my ideas on this very interesting article. An animal study has shown that monocular deprivation may result in decreasing the acuity of the deprived pathway and improving the acuity of the nondeprived way.[4] Reed et al[5] showed better letter recognition in comparison to normally sighted controls in a group of unilaterally enucleated participants. Therefore, cortical response to monocular visual deprivation caused by ION may have resulted in with the reorganization of central and/or

peripheral mechanisms including still viable cells or removal of binocular inhibitory interactions along the visual pathway on the amblyopic side. Some aspects of vision in the amblyopic eye seem to be enhanced by the loss of binocularity as in the study by Resnick et al.[1]

Another issue is that some of the nonarteritic ION patients gain 3 or more lines within 6 months of event.[6] I wonder whether the authors met such an improvement and any changes occurred in the amblyopic eyes during the course.

Kemal Örnek, MD
Department of Ophthalmology, Kırşehir Ahi Evran University School of Medicine, Kırşehir, Turkey

The author reports no conflicts of interest.

REFERENCES

1. **Resnick HH**, Bear MF, Gaier ED. Partial recovery of amblyopia after fellow eye ischemic optic neuropathy. J Neuroophthalmol. 2023;43:76–81.
2. **Bissell AJ**, Yalcinbayir O, Akduman L. Bilateral geographic atrophy: spontaneous visual improvement after loss of vision in the fellow eye. Acta Ophthalmol Scand. 2005;83:514–515.
3. **Fronius M**, Bachert I. Spontaneous improvement of visual acuity in adult amblyopia after functional loss in the non-amblyopic eye? Klin Monbl Augenheilkd. 2009;226:634–639.
4. **Fischer QS**, Graves A, Evans S, Lickey ME, Pham TA. Monocular deprivation in adult mice alters visual acuity and single-unit activity. Learn Mem. 2007;14:277–286.
5. **Reed MJ**, Steeves JK, Kraft SP, Gallie BL, Steinbach MJ. Contrast letter thresholds in the non-affected eye of strabismic and unilateral eye enucleated children. Vis Res. 1996;36:3011–3018.
6. **Mathews MK**. Nonarteritic anterior ischemic optic neuropathy. Curr Opin Ophthalmol. 2005;16:341–345.

Comments on: Partial Recovery of Amblyopia After Fellow Eye Ischemic Optic Neuropathy: Response

We thank Dr. Örnek for his interest in our work on this important topic.

We agree that elimination of monocular inputs imparts a strong drive for neuroplastic expansion of cortical responsiveness to the unaffected eye that defies the traditional concept of the critical period. Since publishing this article, we have extended our examination of this phenomenon to all published cases to identify clinical factors that facilitate recovery from amblyopia.[1] As Dr. Örnek points out, there are several potential mechanisms at play as we have outlined in a recent review.[2] We are committed to elucidating these mechanisms which hold translational potential for visual recovery beyond amblyopia.

To address the question of visual acuity recovery in ischemic optic neuropathy (ION)–affected eyes and the potential influence on amblyopic eye visual acuity, we provide detailed information about the fellow eye best-corrected visual acuity (FEVA) across the post-ION follow-up period (Fig. 1). The degree of FEVA vision loss, worsening over time, and recovery do not systematically influence the degree of amblyopic recovery. Among those showing improvement, there were 3 of 9 cases (5, 7, and 12) that showed 1–2 lines of amblyopic eye best-corrected visual acuity (AEVA) regression between the best AEVA and the final follow-up visit (Fig. 1; red), although with the caveats that Case 5 had macular pathology in the amblyopic eye that may have progressed and the final examination was without glasses for Case 12. Nevertheless, these cases do not consistently demonstrate prominent ION/FEVA recovery. Overall, the relatively small sample size precludes us from drawing definitive conclusions regarding this question.

In our meta-analysis of 101 reported cases with AEVA improvement after FE vision loss (which included this ION data set), multiple regression analysis revealed that FEVA nadir was independently associated with greater grains AEVA.[1] We did not examine durability of AEVA because

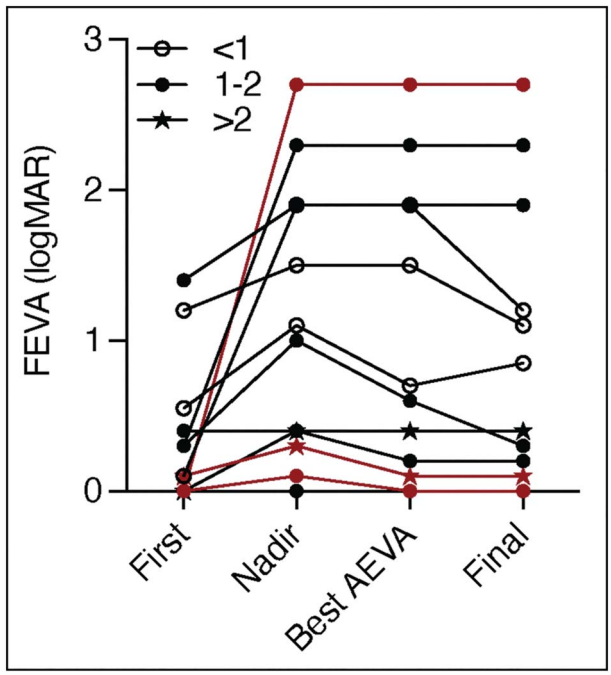

FIG. 1. Fellow eye best-corrected visual acuity (FEVA). FEVA plotted for each relevant post-ION visit: Initial, nadir (worst FEVA documented), best amblyopic eye best-corrected visual acuity (AEVA), and the final follow-up visit. Cases are represented by symbols according to the category of AEVA recovery as indicated by legend (lines of AEVA improvement). Cases in red showed 1–2 lines of AEVA regression between the best-recorded time point and the final follow-up visit. ION indicates ischemic optic neuropathy.