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## Myelin damage after NAION impacts visual function and recovery

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Nonarteritic anterior ischemic optic neuropathy (NAION) is a sudden ischemic episode that causes permanent visual debilitation. While vision can improve somewhat following NAION, it never returns to baseline function, and many individuals suffer additional visual loss. The reason for these post-ischemic functional changes remains unknown. We hypothesized that these variations result from changes in post-infarct myelination and repair. We obtained human material from a NAION-bilaterally affected human donor who experienced NAION at distant (>20y) and near (~1-1/2y) times. We also generated tissue from the primate NAION (pNAION) and rodent NAION (rAION) models, and evaluated affected ON tissue functionally (visual evoked potentials: VEPs, compound action potentials: CAPs) and histologically using immunohistochemistry and ultrastructural analysis. Considerable post-infarct myelin damage of intact axons was found in both human and animals by confocal analysis, particularly in the human specimen with recent NAION. Ultrastructural analysis revealed myelin damage in both clinical and experimental groups. While *in vivo* VEPs did not show typical demyelination changes, *ex vivo* CAPs demonstrated slowing of axonal conduction, consistent with demyelination. Myelin damaged areas with intact axons were associated with significant cellular inflammation. Our results suggest that NAION results not only in isolated retinal ganglion cell and axonal loss, but also post-infarct demyelination and myelin damage that may be responsible for much of the post-infarct variability in visual function, late improvement and decline. Effective NAION treatments may focus on immunomodulation to improve the inflammatory response, reduce long-term, post-infarct demyelination and damage, and improve clinical functional outcomes.

## Biography

Steven Bernstein received his Ph.D. in Neurobiology from Cornell University, and M.D. from SUNY-Downstate. His Ophthalmology residency training was at the Medical College of Virginia, followed by an Ophthalmic Genetics Fellowship at the NEI. Bernstein's postdoctoral research training was in retinal biology. Bernstein is currently Professor and Vice Chair of Ophthalmology at the University of Maryland at Baltimore. He is an author of more than 70 peer reviewed papers. His laboratory focuses on characterization and treatment of ischemic optic neuropathies. He serves as an editorial board member of Molecular Vision.

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