the study to detect a difference between treatment groups would not be appropriate.

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Linear Relation between Structure and Function

The main limitation of the study by Danesh-Meyer et al.[1] was neither discussed nor mentioned. The time axis was not taken into account. The functional change after an acute event such as anterior ischemic optic neuropathy (AION) is immediate, but there is a delay before the anatomic changes take place. In this study, most of the patients with nonarteritic anterior ischemic optic neuropathy (NAION) or arteritic anterior ischemic optic neuropathy (AAION) were tested a short period after the acute event, as described by the authors.

In a similar study by Hood et al.,[2] the patients were tested at least 5 months after the AION event, with a median of 2.95 years. The reason was to allow sufficient time to minimize the effects of optic disc swelling and to allow the retinal ganglion cell (RGC) axons to degenerate. The results of the study in Hood et al. are obviously contrary to those presented by Danesh-Meyer et al. The relationship between a structure (optical coherence tomography [OCT]–determined retinal nerve fiber layer thickness) and function (standard automated perimetry [SAP]–determined sensitivity loss) is the same in patients with AION as in those with open angle glaucoma (OAG).

It is not adequate to use visual field perimetry results as a criterion for comparison in the population examined by Danish Meyer et al.,[1] as visual acuity and visual fields improve up to ~6 months from the onset of NAION.[3]

The only conclusion that can be made from the results of Danish-Meyer et al.[1] is that a few months after the acute event of NAION/AAION the optic disc and RNFL look different than they do in OAG.

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Author Response: Linear Relation between Structure and Function

We thank Wegner and Erben for their comments and draw their attention to the Discussion in our article.[1] In that section, we addressed their concern by stating that "most of our imaging studies were performed longer than 6 weeks after presentation, with 52 (91%) of 57 NAION and 16 (80%) of 20 AAION eyes imaged with HRT and/or OCT 3 months or more after the event." We further discussed that there may have been some additional thinning of the RNFL, which continues subsequent to this time point. However, when we limited the patients included to those with data recorded greater than 3 months after the acute AION event, there were no significant changes in the results.

We also disagree that the data of Hood et al.[2] are similar to ours. They did not evaluate optic disc topography, but rather modeled the relationship of peripapillary retinal nerve fiber layer thickness to visual field mean deviation. The conclusion we drew from our work was that there was a difference in the topography of the optic disc between open angle glaucoma and the anterior ischemic optic neuropathies, not that there were differences in nerve fiber layer thickness. We believe our conclusion about differences in disc topography is strengthened by the fact that we explicitly controlled for the total amount of damage in each condition, using either visual field mean defect or average nerve fiber layer thickness. Furthermore, Hood et al. did not differentiate between AAION and NAION in their study.

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Tobacco Smoking and Its Impact on Corneal Biomechanics

In their excellent study, Sahin et al.[1] investigated the effect of diabetes mellitus on various corneal biomechanical parameters, as measured by the Ocular Response Analyzer (ORA; Reichert, Inc., Depew, NY). The rationale of their study is very interesting. To my surprise, the authors showed a decrease in corneal hysteresis (CH) rather than the expected increase. Several factors suggest that diabetes mellitus would actually enhance corneal biomechanics by an increase in the cross-linking rate: First, an earlier retrospective study showed a lower incidence of keratoconus in diabetic patients, suggesting that corneal biomechanics are enhanced in diabetic corneas.[2] Second, the nonenzymatic glycosylation of proteins (Maillard reaction) that is prominent in diabetes mellitus, results in the formation of advanced glycosylation end products (AGEs). AGEs induce cross-links between connective tissue collagen and increase tissue rigidity, especially in the presence of glucose.[3,4]

Similar to diabetes, tobacco smoking represents a source of AGEs, and moreover, by-products of cigarette smoke, such as nitrogen oxides, nitrite, and formaldehyde, induce cross-links between collagen fibers.[5–7] A recent epidemiologic study showed that the incidence of keratoconus in smokers is considerably lower than in the nonsmoking population,[6] and we have recently performed a prospective comparative case series to investigate the effect of chronic tobacco smoking on corneal biomechanics using the ORA. Our results showed that chronic smoking increases corneal rigidity in a statistically significant manner.[8]

The study by Sahin et al.[1] shows the opposite and was performed in Turkey. From 1990 to 1999, Turkey had the second highest growth rate in cigarette consumption in the world, and in 1999, Turkey accounted for 2.2% of the total world cigarette consumption.[8,9] Therefore, accounting for the smoking status of the participants in this study would be essential for the outcome and might have significantly altered the results. The authors could not be aware of the influence chronic tobacco smoking might have on their results, because at the time of publication of their study our paper, now published, was in press.[8]

I therefore suggest that Sahin et al.[1] determine the smoking status of their patients and perform the statistical analysis in light of their findings.

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Lack of Statistical Power and Refractive Outcomes

We read with great interest the article by Raymond et al.[1] on a randomized controlled study comparing refractive outcomes after cataract surgery using applanation ultrasound (US) or partial coherence laser interferometry with the IOLMaster (Carl Zeiss Meditec, Dublin, CA). The purpose of the study was to assess whether these methods of measurement of axial length have a difference in precision of refractive outcomes. There are two aspects of the design of this study that compromise its conclusions.

The authors state that the trial was powered to detect a difference of 0.24 D in mean absolute error (MAE), without explaining the reasons or providing any evidence of why a difference of <0.24 D is not clinically significant. We can only assume that a level of 0.24 D was selected because of evidence supporting that a change of 0.25 D in spherical equivalent has an impact on unaided visual acuity.[2] A level of 0.24 D in MAE can actually have a big impact on refractive outcomes. For example, Olsen[3] discovered a difference at 0.23 D in MAE between applanation US and IOLMaster biometry (0.65 D vs. 0.43 D). This result translated to improved refractive outcomes from 45.5% and 77.3% for applanation US to 62.5% and 92.4% for IOLMaster for deviations of ± 0.5 and ± 1.0 D from the expected outcome ($P < 0.00001$).[3] According to the criteria set for the study by Raymond et al.,[1] this level of improvement in refractive outcomes is not clinically significant. There have been no clinical studies validating a specific level of clinical significance for MAE in the setting of refractive outcomes after cataract surgery.

MAE is a measure of the spread (precision) of a distribution assuming a mean numerical error (MNE) of 0. When the MNE is not 0, the MAE is increased, and it no longer quantifies spread (precision) alone but is also affected by inaccuracy. The authors' decision not to use optimized IOL constants but to use those recommended by the manufacturer (118.9 for IOLMaster and 118.7 for applanation US) could have introduced systematic errors from high MNEs and further compromised the