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REPLY: We appreciate Dr Kim's point regarding our recent publication.¹ Although the addition of a nonsteroidal anti-inflammatory drug (NSAID) to topical prednisolone acetate

(PA) decreased the incidence of visually significant postphacoemulsification macular edema (i.e., with visual acuity of $\leq 20/40$ at diagnosis), the overall rate was low. The clinical significance of this rate, however, is still incompletely understood.

The effect of macular edema on visual acuity after recovery from phacoemulsification has not been examined in a controlled study with adequate follow-up, and our study did not have adequate resources to enable processing of late visual outcomes. Thus, the question of whether prophylactic NSAID improves late visual outcomes remains unresolved. However, Hunter et al² demonstrated ultrastructural changes in photoreceptors using ultra—high-resolution optical coherence tomography in patients' eyes with subnormal vision after phacoemulsification. Macular edema may have more of a permanent, long-lasting effect on vision than previously realized and merits further study.

Kim also suggests that NSAID plus PA in essence doubles the dose of a single-mechanism agent.³ This depends on how the drops were instilled by the patient. If both drops were instilled within a few seconds of each other, assuming no synergistic effect, then this is closer to a single instillation of 1 agent, given the small carrying volume of the eye surface.⁴ However, if the drops were instilled some minutes apart, substantial absorption of drug from the first drop would most likely occur before instillation of the second drug, allowing for an increase in overall drug concentration in the anterior chamber, and thus potentially doubling the dose of anti-inflammatory agent. Studies have shown that patients have a poor track record for separating drops and instilling them correctly,⁵ and we question whether "double dosing" can explain the 55% decrease in macular edema risk that we observed.

The question of prophylactic agent and the association with late, postoperative visual acuity requires further study. In addition, subgroup analysis examining ocular comorbidity and race is needed to evaluate whether prophylaxis is especially beneficial in these patients, and to examine the risks of NSAID. With an annual rate of 3 million cataract surgeries performed each year, and the widespread use of prophylactic NSAIDs in the United States, these questions have profound public health significance.

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Re: Wang et al.: Comparison of newer intraocular lens power calculation methods for eyes after corneal refractive surgery (Ophthalmology 2015;122:2443-9)

TO THE EDITOR: We read with interest the article on calculation methods for eyes after photorefractive surgery by Wang et al.¹ The authors explained that because of reduced accuracy of intraocular lens power prediction with methods using pre-LASIK/photorefractive keratectomy (PRK) keratometry values (Ks) in a previous study that they performed, they did not evaluate methods including that information. Effectively, some years ago they found that those methods using only pre-LASIK/PRK K values and surgically induced changes in manifest refraction performed the worst compared to methods using surgically induced change in refraction (but not K values), and methods using no previous data.² From a theoretical point of view, the clinical history method is ideal, and it is still considered the gold standard.³ However, its performance depends entirely on the quality of existing information. The problem is that

accurate and trustworthy data very rarely are available. Many times the post-LASIK/PRK refraction has been performed many years before the cataract onset. In such case, changes in corneal profile (possibly owing to epithelial hypertrophy) or in axial length (related to myopic progression) might have happened,⁴ making the refraction unreliable. On the other hand, a refraction after LASIK/PRK procedure, performed shortly before phacoemulsification, may have been done when the cataract has already generated refractive changes in the eye. In the original article from 2010, Wang et al recognized that because their historical data were typically acquired elsewhere they may not be accurate. In this new series, they did not evaluate the historical methods that use pre-LASIK/PRK K values; however, it would have been interesting to analyze the results predicting the intraocular lens power in a group of patients whose pre- and post-LASIK/PRK data (including K values) were of good quality. We are convinced that the performance of historical methods that use K values would improve significantly.

The authors explained that, in a subgroup of 28 eyes, they had the data of change in manifest refraction available and four methods using this information were assessed: adjusted effective refractive power, adjusted Atlas 0–3, Masket, and modified Masket. Again, it would be interesting to know the reliability of those postoperative data on refraction. For example, how a long time before the phacoemulsification were the data obtained? Did slit-lamp biomicroscopy confirm the absence of cataract at that moment? We wonder if, by selecting cases with high-quality prior data, there would have been significant differences among formulas using change in refraction compared with formulas using no prior data.

The range of intraocular lens prediction error was rather large with all the methods using no prior data and their combinations, on average between -2.03 and 1.83 diopters. An additional analysis of risk factors related to this outcome in eyes with a postoperative refractive prediction error of >1.00 diopter would have been enlightening.

Recently, Fram et al⁵ found that mean absolute refractive prediction errors were lower for Optiwave Refractive Analysis (ORA) device (Alcon Labs, Fort Worth, TX) than for optical coherence tomography, and for Haigis-L, but without reaching significant difference. The percentage of eyes within ± 0.50 , ± 0.75 , and ± 1.00 diopters of refractive intraocular lens power prediction errors were higher using the ORA than optical coherence tomography and Haigis-L methods. It seems that intraoperative aberrometry shows promising results. In a given case, the system would confirm the calculations previously done using other methods. If the results of ORA do not match those previously obtained, the surgeon can assess the conditions in which they are performing intraoperative aphakic refraction, which might be causing alterations in the results, such as intraocular pressure outside the appropriate range or eyelid speculum pressure causing globe distortion. If a new measurement made with the ORA consistently yields a different result than previous estimates by other methods, the surgeon will face the dilemma of which one to use. That question remains unsolved, but with the ability to perform intraoperative pseudophakic refraction immediately after the implantation, there is the possibility of solving a refractive surprise in the same surgery. The advent of the latest ORA should lead to a change in the way cataract surgery is performed in patients with a history of corneal refractive surgery.

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REPLY: We thank Drs Holladay, Galvis, and Tello for their thoughtful comments about our paper.¹ First, we want to acknowledge Dr Holladay's initial contribution to this area.



His seminal article in 1989² was the first theoretical recognition of this problem and, by recommending the clinical history method, he offered the first solution for improving the accuracy of intraocular lens calculations in these challenging cases. We agree with them that, theoretically, the clinical history method should be accurate; however, as they nicely outlined, its performance depends excessively on the quality of preoperative data. There is essentially a one-to-one diopter error if any of the historical data are incorrect. Historical data are often obtained from another office where one cannot verify the calibration of the keratometer, accuracy of the person obtaining the measurements, and so on. Also, it is often difficult to accurately determine when the post-LASIK refraction has stabilized before the cataract has begun to alter the refractive error.

Several methods modify corneal power measurements or intraocular lens powers based on the amount of refractive change induced by the LASIK/photorefractive keratectomy surgery. The advantages of these approaches are that they use corneal data obtained at the time the patient presents for cataract surgery and, by multiplying the change in manifest refraction by some fraction, typically <0.3, they avoid the one-for-one error involved in the approaches that rely entirely on historical data.

In our study,³ we had data for change in manifest refraction for only 28 eyes. The patient characteristics in this recent study were similar to these in our earlier study in 2010⁴ in that the historical data were typically acquired elsewhere. We agree with the authors that it is desirable to evaluate the performance of the clinical history method in selective cases with known accurate preoperative data. That said, in the real world of clinical practice, these patients are uncommon as we see many patients who do not even remember who performed their surgery.

They raise an excellent point regarding the value of exploring factors that might predispose to refractive prediction errors of >1.0 diopter (D). Of the 104 eyes included in our study,³ depending on the methods requiring no prior data, 5-12 eyes had refractive prediction errors of >1.0 D. The sample size is too small for further risk factor analysis. We will collect cases with refractive prediction errors of >1.0 D and hope to explore this in a future study.

We did not evaluate the performance of Optiwave Refractive Analysis device (Alcon Labs, Fort Worth, TX) in this study. However, the authors misquote the paper by Fram et al,⁵ who in fact found no difference between outcomes using Optiwave Refractive Analysis versus other methods. We do agree that further studies comparing outcomes using the Optiwave Refractive Analysis versus other devices/methods are needed.

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