iodine (PI) and low fluoroquinolone (FQ) dose might increase resistance to antibiotics," but we believe that it may not be appropriate to attribute the development of resistance against fourth-generation FQ to PI. PI was used just once every 4 weeks just before changing the contact lens. We wonder if this frequency could play any role in the development of resistance against a completely different class of drug that is fourth-generation FQ. Moreover, the authors themselves have mentioned in their discussion part that "Furthermore, so far there is no report of resistance induced by PI in ophthalmologic use.[2] Hence, this reinforces that the resistance induction in our series is most likely attributed to the daily use of antibiotic." The conclusion in the abstract looks contradictory to what has been discussed in the text.

The study included 31 samples from 19 eyes of 18 patients with Boston keratoprosthesis. This suggests that few of the samples were taken from the same patient's eye, although at different points of time. Local microorganism flora could play an essential role in the development of infection as well as colonization of the contact lenses used. Taking multiple samples from the same eye or patient, even at different points in time, could affect the study results. It would be interesting to know the microorganism and resistance pattern without the inclusion of multiple samples from a single patient.

Finally, in addition to the relatively smaller sample size for making any conclusive statement, the study included 3 different groups of patients: multiple graft failure in 9 patients (50%), chemical burn in 8 patients (44.4%), and Stevens–Johnson syndrome in 1 patient (5.6%). The 3 groups differ significantly in their pathogenesis as well the chronic changes produced in the eye, which again could have an impact on colonization of contact lenses and the development of antibiotic resistance. It would be interesting to see if there were any differences in the antibacterial resistance and microbiota patterns between these 3 groups.

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REPLY

WE READ WITH INTEREST THE COMMENTS ABOUT OUR article by Sahay and associates.[1] We agree with the authors that it is not appropriate to attribute the development of antibiotic resistance to povidone-iodine (PI). While we understand the authors' concern, their interpretation must be analyzed with discretion. Even though in the discussion of our article it is clear that resistance was probably attributed to fourth-generation fluoroquinolone, it is necessary to disclose that all patients used a prophylactic regimen that not only included antibiotic therapy, but also PI. As the authors have noted, we have already said that there is no report of resistance induced by PI in ophthalmologic use.[2]

The ocular flora undoubtedly could have had a role in corneal infection. However, our study did not aim to analyze the flora of a specific patient in a single time point, but to investigate the antibacterial resistance under a given chronic prophylactic scheme.[3] Moreover, sampling the same patient at different time points may potentially confirm resistance in a given eye and also allow genotypic investigation to characterize microbial resistance development.

We are aware that the natural flora could differ depending on the preoperative diagnosis. Nonetheless, because contact lenses were only used during the postoperative period, microbiologic investigations on contact lenses were not possible before Boston type I Keratoprosthesis implantation. In addition, soon after the surgery all eyes were placed on the same prophylactic regimen, and therefore one could assume that the flora was no longer virgin and that this would not interfere in the proposed analysis. Antibacterial resistance and microbiota pattern in patients with different preoperative diagnoses can be checked in the table in our article. Although resistance to fourth-generation fluoroquinolone was identified in 6 different eyes, we do not believe that an analysis between groups would add relevant information given our sample size. Ideally, multicenter studies with larger samples may bring about more conclusive knowledge. We reinforce that recognizing the pattern of resistance to antibiotics is critical to outline future changes of prophylactic guidelines and therapeutic strategies.

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Re: Magnetic Resonance Imaging of the Globe–Tendon Interface for Extraocular Muscles: Is There an "Arc of Contact"?

EDITOR:

EACH EXTRAOCULAR MUSCLE NORMALLY LEAVES ITS INSERtion, wraps around the globe along a contact arc, and departs, tangent to the globe (perpendicular to its radius, departure angle = 0), at a departure point en route to its connective tissue pulley or trochlea. Recessed muscles, muscles with posterior sutures, and muscles in extreme ipsiversive gaze can unwrap from the globe, lose tangency (departure angle >0), and suffer their oculorotary force reduced by the cosine of the departure angle.

Clark and Demer[1] mean to cast doubt on the contact arc notion. Accordingly, they looked for departure angles >0, but only with eyes in extreme ipsiversive gaze where muscles were unwrapped from the globe and in abnormal and operated eyes—all cases for which contact arc models would also predict loss of tangency.[2] Their study therefore does not bear on the existence of contact arcs.

Instead of simply measuring departure angles relative to globe tangents, Clark and Demer[1] wrongly assert that contact arcs require muscles to take straight paths to their anatomic origins, as though pulleys did not exist, and compare their measurements to "predicted" departure angles, determined by globe center (their white pixel "1"), insertion ("2"), and anatomic origin ("4"). Their magnetic resonance imaging analysis is consequently spurious.

Looking away from these conceptual errors, one can ask how large the claimed effects were. The largest deviation from tangency reported for normal eyes is 6.2°. The cosine of 6.2° is 0.995, which means that the reduction in oculorotary torque related to a loss of tangency is 0.5%. Far from "fundamentally alter[ing] the globe–tendon interface," effects of this size would best be described as "negligible."

Nothing in their article in any way discredits existing modeling.

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REPLY

WE THANK DRS. MILLER AND SCOTT FOR REVIEWING OUR work and enabling us to elaborate. As they succinctly state, the "arc of contact" model predicts an extraocular muscle departure angle of 0°—a perfect tangent to the globe whereby all extraocular muscle force parallels globe circumference at the insertion. Given this defining prediction, any data that convincingly demonstrate a significantly nonzero departure angle under appropriate conditions is inconsistent with the "arc of contact" model. Any such inconsistency, if observed, should be interpreted within the context of 2 additional considerations. First, nonzero departure angles are predicted by the arc of contact model when globe rotations exceed an angle where tendon tangency is lost and the only tendon contact with the globe is at the scleral insertion. In other words, the arc of contact predicts a 0° departure angle only when at least some of the extraocular tendon remains wrapped around the globe. Second, a nonzero departure angle would only be problematic for the arc of contact theory were it sufficiently different from 0° to materially affect the mechanics of ocular rotation. We agree that a slightly nonzero departure angle, even if statistically significantly so, may have too small a mechanical effect for its existence to compromise the arc of contact model for practical purposes. We interpret the letter by Miller and Scott as arguing for the second consideration. We maintain that we have provided evidence that nonzero tendon departure angles are often too large to be neglected.