Laser In Situ Keratomileusis Disrupts the Aberration Compensation Mechanism of the Human Eye

ANTONIO BENITO, MANUEL REDONDO, AND PABLO ARTAL

- PURPOSE: To study how changes induced on corneal optics by myopic and hyperopic laser in situ keratomileusis (LASIK) refractive surgery affect the aberration compensation mechanism.
- DESIGN: Intervventional case series and modeling theory.
- METHODS: We measured ocular, corneal, and internal aberrations for a 6-mm pupil in 15 myopic and 6 hyperopic eyes with similar age range before and 6 months after standard LASIK. Ocular aberrations were measured using our own developed Hartmann-Shack wavefront sensor, whereas corneal aberrations were calculated by using the elevation data obtained by corneal topography. Ocular, corneal, and internal root mean square (RMS), spherical aberration (SA), coma, and compensation factor were compared for each patient.
- RESULTS: After myopic LASIK, we obtained an average 1.6-fold increase in ocular RMS, mainly positive SA, and coma, associated with a similar increase in corneal aberrations. In the hyperopes, we found a higher (2.3-fold) induction of ocular aberrations after surgery, mainly negative SA and coma, but without net increases of corneal aberrations. Aberration compensation clearly decreased or even inverted after hyperopic LASIK, decreasing the ocular optical quality in a higher level than myopic LASIK.
- CONCLUSIONS: Although ocular aberrations after myopic LASIK usually were smaller than corneal aberrations because of partial compensation of SA, after hyperopic LASIK, because of induction of negative SA and change in coma, disruption of the compensation mechanism lead to a larger increase of ocular aberrations. Customized procedures should maintain the natural compensation to achieve improved visual outcomes. (Am J Ophthalmol 2009;147:424–431. © 2009 by Elsevier Inc. All rights reserved.)

The optical performance of the eye is set by both the aberrations of the cornea and lens. The first evidence of aberration compensation was the coupling between the with-the-rule corneal astigmatism and the astigmatism of the crystalline lens. More recently, different studies further described the compensation of the optical aberrations of the cornea and lens. In young eyes, this includes spherical aberration (SA), positive for the cornea and negative for the lens, although this compensation seemed to be decoupled in the aging eye because of a progressive change in the SA toward positive values of the lens. In addition to symmetric aberrations, such as SA, other aberrations such as coma were found to be compensated. This can be explained by the natural tilt of the eye's optical axis toward the temporal direction, commonly known as angle kappa (κ). This produces an increase of aberrations of both the cornea and internal media, especially lateral coma (c3), but with opposite sign. This compensation mechanism permits eyes with large anatomic differences, for instance, hyperopes and myopes, to have a similar optical quality.

Laser in situ keratomileusis (LASIK) is a successful technique for correcting myopia, hyperopia, and astigmatism. In myopes, the technique reduces the corneal curvature by subtracting a positive meniscus of corneal tissue creating a flatter optical zone. In hyperopes, LASIK ablates an outer ring to increase corneal curvature. In both cases, a transition zone is included to fit the optical zone with the peripheral untreated cornea, although the hyperopic correction may include an inversion from convex to concave shape to fit the corneal periphery. Although LASIK usually achieves good results correcting refractive errors, it usually increases corneal aberrations. Previous studies showed a relationship between the preoperative refractive error and the change in corneal SA after myopic and hyperopic LASIK. It has been suggested that an increase in corneal asphericity proportional to the amount of correction is not inherent to the Munnerlyn formula, probably because of losses of laser efficiency or corneal biomechanics. Differences in the ablation profile between myopic and hyperopic standard LASIK imposes significant differences between the surgically induced aberrations: induced positive SA after myopic LASIK and negative SA after hyperopic LASIK. The other type of aberration commonly induced by LASIK is coma, mainly because of decentration of the ablation profiles during the procedure. The amount and direction of corneal coma induced by surgery depends on several factors, among which ablation profile and corneal position play an important role. All these changes in corneal aberrations resulting from LASIK may disrupt the natural coupling with...
internal aberrations, inducing an additional decrease of the overall optical quality of the eye. The purpose of this study was to show how standard LASIK disrupts the aberration compensation presents in normal young eyes and the possible differences between the 2 types of surgeries.

**METHODS**

- **SUBJECTS**: Ocular, corneal, and internal aberrations were measured before and 6 months after standard LASIK. Surgical procedures were performed using a VISX S2 (AMO, Santa Ana, California, USA) excimer laser platform at “Clinica Ircovisión” (Cartagena, Murcia, Spain). The measured population included 21 eyes (n = 11 subjects) classified according to their preoperative refractive error: 1 group including myopic eyes (n = 15 eyes; mean age ± standard deviation [SD], 28.0 ± 3.6 years; mean sphere, −4.08 ± 1.70 diopters [D]; mean cylinder, −0.68 ± 0.38 D), and other group including hyperopic eyes (n = 6; mean age ± SD, 30.7 ± 8.0 years; mean sphere, 3.63 ± 0.96 D; mean cylinder, −2.70 ± 1.83 D). Measurements were obtained under natural viewing conditions. The measured optical data were not used during the surgical procedures.

- **PROCEDURE**: Ocular wavefront aberrations were measured using our own developed near-infrared Hartmann-Shack wavefront sensor adapted to the clinical environment; the system has 300 microlenses sampling a 6-mm pupil diameter with a high dynamic range.\(^{18}\) Corneal aberrations were obtained for the same pupil diameter from the corneal elevation maps provided by videokeratography\(^{16}\) (Atlas; Carl Zeiss Meditec, Dublin, California, USA) by custom ray tracing using optical design software (Zemax software; Zemax Development Corp, San Diego, California, USA).

In every eye, the position of the pupil center with respect to the corneal reflection was obtained from the Placido disc images of the corneal topographer. In high myopic eyes (Figure 1, Top), the line of sight, and the visual and keratometric axes nearly coincide, as a consequence of small angle \(\kappa\), and pupil decentration decreases.
However, in hyperopic eyes, a larger angle \( \kappa \) supposes the optical axis to be tilted in the temporal side, and as a consequence, the line of sight and visual and keratometric axes are displaced laterally and a larger pupil decentration is observed. Pupil decentration was obtained first in polar coordinates and the horizontal (x-axis) and vertical (y-axis) coordinates were calculated.

Average ocular wavefront aberrations were computed from 4 different Hartmann-Shack images, whereas corneal wavefront aberrations were obtained from 3 consecutives videokeratographic measurements. Internal aberrations were obtained by subtracting corneal from ocular aberrations, using the pupil center as reference for both set of data to avoid errors when corneal aberrations were not measured at the line of sight.\(^{17}\) We compared ocular, corneal, and internal aberrations before surgery and 6 months after LASIK. Root mean square (RMS) of the higher-order aberrations, fourth-order SA (\( c_4 \); SA), and the magnitude of (coma = \( \sqrt{(c_3^1)^2+(c_3^2)^2} \)) were calculated for every case. We also obtained a compensation factor (CF), \( CF = 1 - (\text{RMS}_{\text{eye}}/\text{RMS}_{\text{cornea}}) \), as defined by Artal and Guirao, as a measure of the relative efficiency of the aberration compensation mechanism.\(^2\)

**RESULTS**

FIGURE 2 shows average ocular, corneal, and internal aberrations for a 6-mm pupil for the groups of myopic and hyperopic eyes. Before surgery, hyperopic eyes had more corneal and internal aberrations than myopes, mainly because of a higher coma for each component (cornea and lens). However, the overall ocular aberrations were similar to myopes because of the previously described coupling of corneal and internal SA and coma in young hyperopes. This scenario clearly was modified by the surgery: whereas ocular RMS showed a mean 1.6-fold increase after myopic LASIK (up to 0.49 ± 0.26 \( \mu \)m), we found a 2.3-fold increase (up to 0.57 ± 0.15 \( \mu \)m) after hyperopic LASIK. It should be noted that the mean attempted correction was lower for the hyperopes than for the myopes by approximately 0.5 D.

Results for corneal aberrations showed an average two-fold increase of RMS after myopic LASIK, mainly for SA and coma, although the later showed a larger intersubject variability (±0.32 \( \mu \)m, SD). Mean corneal RMS after hyperopic LASIK showed little variation from preoperative RMS. This could be explained in terms of previous
corneal aberrations and surgically induced aberrations: mean corneal SA changed on average $-0.356$ μm after hyperopic LASIK, whereas after myopic LASIK, the difference with mean preoperative values was $0.173$ μm. On average, internal SA values after myopic or hyperopic LASIK were similar to preoperative values. Then, whereas myopic LASIK increased positive corneal SA, still keeping the compensation with the internal media, after hyperopic LASIK, corneal SA moved toward negative values at a 2.5-times higher rate than for the myopes. These overall changes in corneal aberrations were in good agreement with those of other previous studies. In addition, whereas myopic LASIK increased corneal coma as a function of the amount of the treated myopia ($r^2 = 0.31; P < .001$), after hyperopic LASIK, an average $0.257$-μm reduction of corneal coma was found. The Table summarizes the average ocular, corneal, and internal aberrations before and after myopic and hyperopic LASIK.

![Figure 3](image-url)  
**Figure 3.** Scatterplots showing lateral coma for the cornea (circles) and the internal media (triangles) for all eyes (Left) before and (Right) after LASIK as a function of measured horizontal pupil decentration (X). Regression lines also are shown. Although preoperative results show a coupling between corneal and internal lateral coma, the induced changes of corneal lateral coma resulting from LASIK produced a disruption of the coma compensation.

![Table](image-url)  
**Table.** Preoperative and 6-Month Postoperative Average Results for Aberration Coefficients Root Mean Square, Coma, and Spherical Aberration for the Eye, the Cornea, and Internal Media for a 6-mm Pupil

<table>
<thead>
<tr>
<th></th>
<th>Myopes Preoperative*</th>
<th>Postoperative*</th>
<th>Hyperopes Preoperative*</th>
<th>Postoperative*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ocular RMS</td>
<td>0.297 ± 0.061</td>
<td>0.485 ± 0.264</td>
<td>0.248 ± 0.077</td>
<td>0.568 ± 0.153</td>
</tr>
<tr>
<td>Ocular Coma</td>
<td>0.158 ± 0.071</td>
<td>0.313 ± 0.305</td>
<td>0.176 ± 0.089</td>
<td>0.269 ± 0.088</td>
</tr>
<tr>
<td>Ocular SA</td>
<td>0.064 ± 0.072</td>
<td>0.189 ± 0.109</td>
<td>0.012 ± 0.048</td>
<td>$-0.306$ ± 0.166</td>
</tr>
<tr>
<td>Corneal RMS</td>
<td>0.405 ± 0.085</td>
<td>0.745 ± 0.291</td>
<td>0.562 ± 0.132</td>
<td>0.571 ± 0.102</td>
</tr>
<tr>
<td>Corneal Coma</td>
<td>0.202 ± 0.098</td>
<td>0.473 ± 0.319</td>
<td>0.463 ± 0.158</td>
<td>0.206 ± 0.094</td>
</tr>
<tr>
<td>Corneal SA</td>
<td>0.224 ± 0.069</td>
<td>0.377 ± 0.114</td>
<td>0.095 ± 0.028</td>
<td>$-0.261$ ± 0.133</td>
</tr>
<tr>
<td>Internal RMS</td>
<td>0.362 ± 0.075</td>
<td>0.461 ± 0.147</td>
<td>0.455 ± 0.059</td>
<td>0.537 ± 0.135</td>
</tr>
<tr>
<td>Internal Coma</td>
<td>0.222 ± 0.093</td>
<td>0.258 ± 0.166</td>
<td>0.379 ± 0.087</td>
<td>0.391 ± 0.135</td>
</tr>
<tr>
<td>Internal SA</td>
<td>$-0.138$ ± 0.075</td>
<td>$-0.139$ ± 0.108</td>
<td>$-0.083$ ± 0.048</td>
<td>$-0.046$ ± 0.151</td>
</tr>
</tbody>
</table>

RMS = root mean square; SA = spherical aberration.

*Values are in micrometers (μm) and represent mean ± standard deviation.
These changes in both corneal SA and lateral coma suppose a complete disruption of the compensation mechanism after hyperopic LASIK. Figure 4 shows the CF for all eyes as function of the preoperative refractive error before (solid) and after (clear) standard LASIK. On average, hyperopic eyes (Figure 4, Right) had better compensation before surgery (mean, 0.41 ± 0.18) than myopic eyes (Figure 4, Left; mean, 0.25 ± 0.16). This scenario completely changed after surgery: whereas myopic LASIK increased the average aberration compensation (up to 0.37 ± 0.14), mainly because of SA, after hyperopic LASIK, all eyes showed a reduction compared with the previous CF (down to −0.12 ± 0.41). For half of the hyperopic patients, we even found an inversion on the CF toward negative values, which indicates a complete disruption of the compensation mechanism. An example illustrating this phenomenon is shown in Figure 5. Before surgery, a myopic eye (Figure 5, Left) showed a slight compensation of corneal and internal aberrations (mainly SA); the hyperopic eye (Figure 5, Right) showed higher corneal and internal RMS, but because of compensation, ocular RMS was similar to that in the myopic eye. This situation was modified by the surgery: myopic LASIK increased corneal RMS (mainly positive SA and coma) by nearly 1 μm, but some compensation still remained (ocular RMS increased by 0.70 μm). For the hyperopic eye, LASIK induced an inversion of corneal SA toward negative values and a reduction of preoperative corneal coma, leading to a similar corneal RMS. Postoperative corneal aberrations (negative SA and little coma) did not couple with that of the internal media (negative SA and large coma), leading to a three-fold increase in ocular RMS.

DISCUSSION

We estimated the corneal aberrations by considering a simplified cornea with one single surface, the measured corneal anterior surface, and by using an effective refractive index for corneal aberration calculations. By doing so, we did not completely consider the effect of the corneal posterior surface. This is probably a minor issue in normal eyes, but this may not be the case after LASIK. The behavior of the corneal posterior surface after LASIK and its role in the increment of aberrations has been subject of some controversy during the last years. LASIK disrupts stromal fibers, even in the flap creation process, which tend to relax, inducing a bulge in the periphery of the treated area that affects corneal asphericity, increasing positive SA. A relation between the surgically induced variation of internal SA and attempted correction resulting from the presence of a systematic biomechanical effect over the corneal posterior surface related to LASIK has been suggested. It is not totally clear if the biomechanical changes of the corneal posterior surface after LASIK are large enough to prevent use of a corneal simplified model. In any case, the possible changes in the posterior corneal surface would have affected our estimates of the internal optics aberrations. We found some patients with differences between preoperative and postoperative internal aberrations, although the average difference for the measured population was less than 0.1 μm, which does not support the idea of systematic changes of the aberrations induced by the posterior corneal surface resulting from LASIK. However, because this controversy remains, we considered the difference between ocular and corneal aberrations as internal media aberrations, then included all possible changes of the posterior corneal surface because of the surgery.

Our results on increasing positive SA and coma for both the entire eye and the cornea after LASIK were in good agreement with previously reported results. We also compared our data for the hyperopic eyes with that of reports about optical outcomes after refractive surgery for hyperopia, including those of photorefractive keratectomy. There were similarities about induction of negative SA, although there were many differences about values of postoperative coma; whereas Oliver and associates found large increases in corneal coma after hyperopic photorefractive keratectomy, Wang and Koch found only a small increase in corneal coma after hyperopic LASIK, in this case using the VISX S2 (AMO). Another study compared ocular, corneal, and internal media aberrations after myopic or hyperopic LASIK. Similar to our study, both populations were comparable in terms of age, measured
pupil size, and refractive error ranges, although we considered only subjects measured 6 months after surgery. They found a higher increase of ocular and corneal aberrations resulting from hyperopic LASIK compared with myopic LASIK, mainly corneal negative SA, and an increase in both corneal and ocular coma. Our results revealed that an increase of ocular coma in subjects with previous compensation of aberrations is possible simply by changing the nature of corneal aberrations. This is especially important in hyperopic LASIK. First, the ablation profile itself usually changes the corneal SA toward negative values, decoupling with that of the internal media. Second, hyperopic eyes usually show a large value of angle $\kappa$ and a better compensation between corneal and internal coma. Most studies suggested that hyperopic LASIK tends to increase corneal coma, and then possibly increases that of the eye. However, we showed herein that because of the natural fine coupling of lateral coma within the eye previous to the surgery, not only an increase but also a reduction of corneal coma may cause an increase of coma for the entire eye.

It is widely accepted that myopic LASIK tends to increase both corneal SA, mainly because of the ablation profile and corneal coma related to decentration, and the increase of these aberrations also suppose an increase of ocular SA and coma. Studies considering corneal aberrations after refractive surgery for hyperopia agree in the induction of negative SA, but differ in the amount of corneal coma induced. We should point out that, as is shown on Figure 1, the pupil is usually more decentered in the hyperopic eye because of larger angle $\kappa$. Differences in ablation centration among studies may explain differences in the amount of coma measured after refractive surgery. Figure 6 shows the mean lateral coma after LASIK for 3 groups. Ten myopic eyes with horizontal pupil decentration lower than 0.15 mm (n = 10; black circle); higher than 0.25 mm (n = 5; white circle); and hyperopes (n = 6; black triangle).
gles; mean x-value, 0.44 ± 0.18 mm) showed an important average decrease of lateral coma (−0.31 ± 0.09 μm) after hyperopic LASIK. The myopic eyes with lower pupil decentration (black circles) were those with higher preoperative myopia and those that showed a larger increase in corneal coma. The group of myopic eyes with the largest pupil decentrations (clear circles) did not present any change in corneal lateral coma. But hyperopic eyes with similar pupil displacement showed a large reduction of corneal lateral coma, which may suggest that the observed changes in corneal coma were the result of differences between myopic and hyperopic ablation profiles. The hyperopic LASIK ablation profile have a more complicated transition zone, which possibly induced a less uniform topographic functional optical zone than typical myopic LASIK treatments. Another reason may be the biomechanical changes related to the release of tension on collagen bundles after midperipheral hyperopic ablation, although this is less predictable. A smaller functional optical zone resulting from the intrinsic inversion in the outer blending zone of the hyperopic corrections would made them more suitable to increasing corneal aberrations even in cases of low hyperopia and may be one of the reasons for the comparable low limit of maximum permissible hyperopic correction.

We analyzed the nature of the aberration changes after standard LASIK. We compared the results of ocular, corneal, and internal aberrations after both myopic and hyperopic LASIK. Myopic LASIK increment in corneal SA and coma is followed by a similar increase of ocular SA and coma. The scenario is different for the hyperopic eyes because of a larger coupling between corneal and internal aberrations. Hyperopic LASIK usually disrupts the aberration coupling by modifying the type of corneal aberrations. One reason is that after hyperopic LASIK, the postoperative corneal negative SA adds to that of the lens, resulting in a more negative ocular SA. This may be managed by using new optimized ablation profiles. The other reason to explain the large increase in ocular aberrations after hyperopic LASIK is the disruption of coma compensation. This is a more delicate issue related somehow to centration strategies of the ablation profiles. In this case, customized ablation profiles should maintain the aberration balance between corneal and internal optics. This study recognizes the critical importance of considering the eye as a two-element optical system that is naturally well balanced in terms of its aberrations. Modifying the optical properties of one the components, either the cornea or the lens, should consider the aberration compensation to obtain improved final optical results and in consequence optimum visual outcomes.

REFERENCES


Biosketch

Antonio Benito, after finishing his degree in Optics and Optometry, joined the Pablo Artal’s “Laboratorio de Óptica de la Universidad de Murcia”, Murcia, Spain in 1998. He has recently completed an MSc course at the Department of Physics at the Universidad de Murcia, Spain. His research interests have been focused on aberrations of the human eye, especially on ocular aberration compensation mechanism and optical effects of laser in situ keratomileusis on the aberrations of the eye and its components.