Efficacy of laser in situ keratomileusis in correcting anterior and non-anterior corneal astigmatism: Comparative study

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PURPOSE: To compare the efficacy of conventional laser in situ keratomileusis (LASIK) in treating corneal astigmatism and in treating noncorneal ocular residual astigmatism.

SETTING: Private practice, Nashville, Tennessee, USA.

DESIGN: Retrospective case series.

METHODS: The records of dominant eyes of consecutive patients who had LASIK were retrospectively analyzed to compare the efficacy of LASIK in eyes with predominantly anterior corneal astigmatism with the efficacy in eyes with predominantly ocular residual astigmatism (ORA). The ORA was determined by vector analysis using refractive cylinder and topographic astigmatism. Preoperatively, the ratio of ORA to preoperative refractive cylinder (R) was used to divide the patients into 2 groups; that is, eyes with predominantly anterior corneal astigmatism (ORA/R ratio <1.0) and eyes with predominantly ORA (ORA/R ratio ≥1.0). Efficacy was determined by examining the magnitude of the remaining uncorrected astigmatism and comparing the index of success (proportion of preoperative refractive astigmatism that remained uncorrected by LASIK) between the 2 groups.

RESULTS: The study evaluated 61 eyes of 61 patients. Conventional LASIK was twice as efficacious in the low-ORA group as in the high-ORA group. The index of success was 0.24 and 0.50, respectively, and the difference between groups was statistically significant (P = .036).

CONCLUSION: The efficacy of astigmatic correction by LASIK was significantly higher in eyes in which the preoperative refractive astigmatism was located mainly on the anterior corneal surface than in eyes in which it was mainly located posterior to the anterior corneal surface.

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Laser in situ keratomileusis (LASIK) has been adopted worldwide as a safe and effective means of correcting low to moderate myopia, hyperopia, and astigmatism.1,2 However, LASIK is reported to be less effective in correcting astigmatism than in correcting sphere and even less effective in treating non-anterior corneal astigmatism.3–9 Non-anterior corneal astigmatism is also called intraocular astigmatism or lenticular astigmatism but is effectively quantified by magnitude and axis as the vector value ocular residual astigmatism (ORA). When correcting astigmatism based on refraction or wavefront, as in conventional or wavefront-guided LASIK, the amount of astigmatism created iatrogenically on the anterior cornea to compensate for the amount of ORA can affect postoperative visual quality.5–8

If it is less efficacious to treat astigmatism with an anterior corneal–confined procedure, such as LASIK, in eyes with significant ORA, it may be important to identify such cases preoperatively. These patients should be told that it is likely that LASIK will be less successful in correcting their astigmatism. It is also important that the surgeon be aware that LASIK may iatrogenically create an excessively astigmatic cornea that can reduce postoperative visual quality. Therefore, the appropriate treatment for eyes with high ORA may be to use vector planning, as proposed and studied by Alpins10 and Alpins and Stamatakis11,12 to treat both corneal and
refractive astigmatism, resulting in less induced corneal astigmatism. Alternatively, the more appropriate treatment in such eyes may be a lens-based procedure, thus avoiding corneal treatment altogether.

In recent years, wavefront-guided and wavefront-optimized treatments have gained wide popularity. However, both modalities are based on an important implicit assumption; that is, that the location of an aberration (anterior corneal versus non-anterior corneal) is not important and all aberrations, regardless of axial location, can be adequately treated on the corneal surface. For all aberrations detected by wavefront mapping (in the case of wavefront-guided ablation) or manifest refraction (in the case of wavefront-optimized and conventional ablations), the location of treatment is confined to the anterior cornea, regardless of whether the aberration arises from the anterior cornea or from intraocular structures such as the lens. Considerable progress has been made in recent years in developing topographic-guided technology designed to treat corneal aberrations at their source—the cornea—as well as a hybrid approach combining topographic-guided treatment with wavefront-guided treatment. Although it is now commonly accepted that topography-guided treatment should be considered in eyes with irregular corneal astigmatism (higher-order aberrations [HOAs]), the importance of detecting the location of regular astigmatism (lower-order aberration [LOA]) preoperatively in planning routine laser-vision correction treatment is often overlooked.

To our knowledge, there are no published clinical studies comparing the efficacy of conventional LASIK in treating anterior corneal astigmatism and its efficacy in treating non-anterior corneal astigmatism. If a difference exists, as suggested by Alpins and Alpins and Stamatelatos, using a combined approach that takes into account the total refractive cylinder and corneal astigmatism may be beneficial. The present study was designed to answer this basic question by comparing 2 groups of eyes treated with conventional LASIK; 1 group comprised eyes with astigmatism arising predominantly from the anterior corneal surface (low ORA) and the other, eyes with astigmatism arising mainly from non-anterior corneal sources (high ORA). Our goal was to determine treatment efficacy by comparing the magnitude of uncorrected remaining refractive astigmatism postoperatively in the 2 groups. In the analysis, we used the Alpins index of success method for astigmatic correction analysis.

PATIENTS AND METHODS

Data were gathered from charts of consecutive patients who had successful primary LASIK. Patients with conditions that were common contraindications to LASIK were excluded from surgery; these included inadequate anticipated residual corneal bed, forme fruste keratoconus, irregular astigmatism, anterior basement membrane dystrophy, and severe dry-eye disease. Patients with purely spherical refractive errors preoperatively were also excluded from the study.

To eliminate a possible statistical correlation between 2 eyes of the same patient and exclude the effect of intentional undercorrection in the nondominant eye, only the dominant eye of each patient was included in the study. Data were collected from the initial preoperative examination and at the 3-, 6-, and 12-month postoperative visits. The following were recorded: patient age and sex; preoperative data, including uncorrected (UDVA) and corrected (CDVA) distance visual acuity, manifest and cycloplegic refractions, topography, and keratometry; and postoperative data, including UDVA, CDVA, manifest and cycloplegic refractions, topography, and keratometry.

Surgical Technique

The same surgeon (M.W.) performed all LASIK procedures using an identical laser and microkeratome. After providing informed consent, patients received 0.5 mg of diazepam (Valium) 30 minutes before surgery. In patients with 1.5 diopters (D) or higher refractive astigmatism, the 180-degree meridian was marked at the slitlamp. The patients were taken to the laser suite reclined with their head supported and given 1 drop of proparacaine in each eye. The operative eye was draped and the speculum inserted. An LSK1 microkeratome (Moria) was used to create a 130 μm flap in all cases. The flap was lifted to expose the stromal bed. If necessary, the patient’s head was rotated to align the 180-degree meridian with the corresponding 180-degree marker of the laser reticule. A Visx laser was used for refractive treatment. After the ablation, the flap was repositioned and 1 drop each of diclofenac or ketorolac, ofloxacin or levofloxacin, and prednisolone acetate was administered. The flaps were checked 5 minutes later at the slitlamp, and the patient received verbal and written instructions on the use of postoperative medications as follows: ofloxacin or levofloxacin 4 times a day for 3 days and prednisolone acetate 4 times a day for 3 days.

Patients were examined postoperatively at 1 day and 1, 3, 6, and 12 months.
Determination of Ocular Residual Astigmatism

To determine the amount of ORA before LASIK, standard double-angle vector analysis was performed using a method (Cartesian coordinates) similar to that used by Alpins4 and Jaffe and Clayman.22 As shown in Figure 1, the magnitude and direction of ORA was determined in the double-angle vector diagram by the vector difference between the preoperative refractive astigmatism (R) (corneal plane) and the topographic (simulated keratography) astigmatism (K). The R value was obtained from the manifest refraction. The K value was calculated from corneal topography (Orbscan, Bausch & Lomb) based on the difference between the steepest meridian and the flattest meridian oriented 90 degrees from each other. The ORA is the amount of the vector difference between R and K, or [R – K] with its orientation directed to refractive astigmatism value from cornea, and was calculated using the trigonometric law of cosines6 as follows:

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ORA = \sqrt{K^2 + R^2 - 2KR \cos 2\theta_1 \cos 2\theta_2 + \sin 2\theta_1 \sin 2\theta_2}
\] (1)

where K is the magnitude of corneal astigmatism, R is the magnitude of refractive astigmatism, ORA is the vectorial value arising from non-anterior corneal sources, and 2\(\theta_1\) and 2\(\theta_2\) are the axes (doubled) from K to R, respectively, on the double-angle vector diagram. The amount of contribution to the total refractive astigmatism R by the ORA preoperatively is determined by the magnitude ratio ORA/R. Because R and K are both astigmatism, the magnitude of ORA may be greater than the magnitude of R, resulting in the ratio ORA/R being greater than 1 in eyes with a high amount of ORA.

Preoperatively, patients were divided into 2 groups: low ORA (ORA/R ratio <1.0) and high ORA (ORA/R ratio ≥1.0). In eyes in the low-ORA group, the total refractive astigmatism R arose principally from the anterior corneal surface. In eyes in the high-ORA group, a significant proportion of the total refractive astigmatism R arose from non-anterior corneal sources.

The efficacy of astigmatic correction with LASIK was assessed using an established method of Alpins3,4 and the index of success was calculated. The index of success is the ratio of the magnitude of the remaining uncorrected astigmatism R' to that of the initial preoperative astigmatism R (R/R). This method of analysis is in accordance with the vector analysis method of Alpins3,4 in which the index of success is the ratio of the difference vector to the target-induced astigmatism vector (TIA) (Figure 2). The difference vector is the vectorial difference between the achieved and the intended astigmatism and represents the remaining uncorrected (difference) vector, similar to the remaining uncorrected astigmatism R' in the present study. The TIA represents the intended astigmatic change and is equivalent to preoperative astigmatism R in the present study with an orientation 90 degrees away. The surgically induced astigmatism (SIA) vector is the difference vector between the difference vector and TIA and is the vector between the postoperative astigmatism and the preoperative astigmatism that the surgery actually achieves. In an ideal case in which all preoperative refractive astigmatism is corrected, the difference vector or index of success should be zero.
or, analogously, the remaining uncorrected astigmatism R' should be zero, which is the goal of LASIK treatment. As shown in Figure 2, by plotting the preoperative astigmatism R and the residual uncorrected postoperative refractive astigmatism R' on the double-angle diagram, the index of success can be calculated (index of success = $R'/R$, similar to difference vector/TIA described by Alpins).3

**Statistical Analysis**

The mean values of preoperative parameters (spherical equivalent [SE], astigmatism, and patient age) were analyzed using t tests and Statistical Analysis System software (SAS Institute, Inc.). A P value less than 0.05 was considered statistically significant.

**RESULTS**

The study evaluated 61 eyes of 61 patients, 30 in the low-ORA group and 31 in the high-ORA group. The refraction was stable in all patients by 12 months postoperatively.

Table 1 shows the mean age of the patients, mean preoperative SE, mean preoperative refractive cylinder, and the fraction of remaining untreated cylinder (R2/R) in the low-ORA group and the high-ORA group. There was no statistically significant difference in age or SE between the 2 groups. In all eyes, the preoperative refractive SE at the corneal plane ranged from $-4.16$ to $-6.71$ D and the cylinder from $+0.59$ to $+1.60$ D. The mean preoperative refractive cylinder was statistically higher in the low-ORA group than in the high-ORA group ($P = .0001$).

The mean index of success in the low-ORA group was 0.24, indicating that approximately 75% of the preoperative total refractive astigmatism was successfully treated. In contrast, the mean index of success in the high-ORA group was 0.50, indicating that 50% of the refractive astigmatism was successfully treated. The lower index in the high-ORA group suggests that LASIK was significantly less effective in treating non-anterior corneal astigmatism. The 95% confidence interval of index of success was 0.12 to 0.35 in the low-ORA group and 0.30 to 0.71 in the high-ORA group; the difference between the 2 groups was statistically significant (Table 2).

**DISCUSSION**

Astigmatism is a refractive error that mainly originates from anterior corneal surface toricity. However, in some patients, non-anterior corneal elements in the eye’s optical system can also contribute to the total refractive astigmatism. A discrepancy between anterior corneal astigmatism and refractive astigmatism is a common clinical finding.3,5–8 The vector ORA is a gauge to assess external (anterior cornea) versus internal (non-anterior cornea) LOAs. Alpins’ initial study of 100 patients3 found a mean ORA value of 0.81 D; 34% of patients had a value greater than 1.00 D, and a second study21 found a mean value of 0.73 D. A subsequent study of 220 eyes by Srivanna-boon23 found ORA values higher than 1.00 D in one third of eyes. These studies show that non-anterior corneal astigmatism can be a significant contributor to overall refractive astigmatism. Although the lens may contribute to a large portion of ORA, it is inaccurate to equate lenticular astigmatism with ORA because other elements may also play a role.5–8 These elements include the posterior cornea, vitreous, retina, and nonoptical components, such as the visual cortex.20

Our study was designed to compare the efficacy of conventional LASIK in treating anterior corneal astigmatism and its efficacy in correcting ORA. The results suggest that treating astigmatism with LASIK based on manifest refraction results in a successful outcome only if the preoperative refractive astigmatism arises primarily from the anterior corneal surface. In these eyes, the keratometric, topographic, and refractive astigmatism approximate each other more closely in magnitude and orientation. This result is what one would intuitively expect because the location of the astigmatism—the anterior cornea—is the same as the location of the treatment.

We reviewed several clinical studies9–11,24–30 evaluating the success of PRK and LASIK in treating

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<td>Parameter</td>
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<td>Mean age (y)</td>
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<tr>
<td>Mean preop SE (D)</td>
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<tr>
<td>Mean preop cylinder* (D)</td>
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<td>Fraction of remaining untreated cylinder† (D)</td>
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| ORA = ocular residual astigmatism; SE = spherical equivalent |

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<th>Table 2. Confidence intervals.</th>
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<tr>
<td>Parameter</td>
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<tr>
<td>Age</td>
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<td>Preoperative SE</td>
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SE = spherical equivalent
astigmatism (Table 3). In our study, the index of success was 0.24 in the low-ORA group and 0.50 in the high-ORA group. Our study is unique in that it differentiates the efficacy of astigmatic correction based on the location of the astigmatism on the visual axis and derives an index of success for each type. Several studies have assessed the effectiveness of correcting HOAs measured by wavefront and the secondary impact on anterior corneal astigmatism\(^{10-16}\); however, to our knowledge, ours is the only study to assess the effect of treating only LOAs on the anterior cornea and the relationship between the treatment’s efficacy and the origin and location of the astigmatism preoperatively. The results are equally relevant to wavefront-guided treatments. A wavefront-guided refractive treatment also fails to account for the location along the visual axis (Z) and the source of the regular and irregular astigmatism. Wavefront-guided treatment will, therefore, create a set of aberrations on the anterior corneal surface to compensate for the non-anterior corneal aberrations. These iatrogenically created corneal aberrations may increase the irregularity of the cornea and reduce visual performance.

The amount of postoperative astigmatism in the low-ORA group was 24% of the preoperative value, while it was as high as 50% in the high-ORA group. In other words, as much as 50% of the existing astigmatism in the high-ORA group was not successfully treated by LASIK (Figure 3). We attribute the difference in efficacy in the 2 groups mainly to the nature and origin of the astigmatism and the excess astigmatism remaining on the cornea postoperatively in eyes in which the preoperative ORA was higher. We recognize that there is a difference in the absolute value of the preoperative astigmatism in the 2 groups. This statistically significant difference was expected because eyes with a larger percentage of astigmatism from ORA tend to have a smaller magnitude of net refractive cylinder as a result of the natural compensatory mechanism between the cornea and lens.\(^{5-8}\) The amount of ORA in each group is representative of the natural distribution of ORA and total astigmatism. In other words, if one gathers a group of patients with high ORA, the total refractive astigmatism in that group will be low, or at least lower than the ORA.\(^{5-8}\) Therefore, we believe our cohort is an accurate representative sample of the general population. Interestingly, the group with more corneal astigmatism preoperatively achieved better results. Typically, as the refractive error increases, the remaining postoperative error increases. We found the opposite; that is, a higher percentage of the astigmatism was corrected in patients with a higher magnitude of manifest refractive astigmatism preoperatively. This observation further confirms that correcting astigmatism at its source improves treatment efficacy.

We controlled our study so that the effect of any confounding factor was minimized. The same technician took all measurements, and the same surgeon performed all LASIK procedures using the same microkeratome, excimer laser, and nomogram. Other potential confounding factors must be considered, however. First, LASIK itself can induce astigmatism\(^{30,32}\), and postoperative astigmatism is the vectorial sum of the

<table>
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<th>Parameter</th>
<th>Index of Success</th>
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<tr>
<td>Myopic astigmatism with LASIK(^{9,24,25,27-29,31})</td>
<td>0.12-0.53</td>
</tr>
<tr>
<td>Myopic astigmatism with PRK(^{31})</td>
<td>0.38</td>
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LASIK = laser in situ keratomileusis; PRK = photorefractive keratectomy

Figure 3. Ratio of postoperative cylinder to preoperative cylinder in each group (ORA = ocular residual astigmatism; R’/R = index of success).
resulting untreated astigmatism and new surgery-induced astigmatism. Consider an eye with 1.00 D of preoperative astigmatism; in this case, a postoperative astigmatic value of 0.30 D does not necessarily mean a 70% reduction in astigmatism and 70% treatment efficacy. The ablation itself could have treated all the preoperative astigmatism, and the resulting astigmatic value is due to the architecture of the flap, the patient’s individual healing pattern, or both. Another intraoperative factor that can affect the efficacy of astigmatism treatment is cyclotorsion.33 However, in our study, the same surgical techniques and settings, including limbal marking and head positioning, were used in all eyes. Hence, cyclotorsion and the effect of the flap or hinge on astigmatism did not likely compromise our data in a systematic manner. If they did, both groups would have been equally affected. Furthermore, recent studies conclude that iris-registration technology has no significant benefit in the treatment of astigmatism.34

Figures 4 and 5 are schematic conceptual representations of the ray-tracing principles used to simulate optical patterns before and after LASIK in patients with astigmatism. We show them in an attempt to explain the clinical difference we observed between the low-ORA group and the high-ORA group. The
figures are mainly conceptual with the goal of illustrating the principles involved here and are not intended as an actual representation of the exact ray-tracing numerical result. Figure 4 shows the LASIK astigmatic correction in an eye with low ORA preoperatively; the treatment on the anterior cornea successfully removed all astigmatism. In contrast, Figure 5 shows an eye with high ORA. The LASIK treatment in this case resulted in the creation of “reverse” astigmatism on the cornea. Because the new corneal astigmatism and the preexisting ORA are not located on the same z point along the visual (z-axis), the astigmatism is not removed entirely and ray tracing an object resulted in a distorted and blurred retinal image.3–8,35
The size, shape, and orientation of the retinal image can significantly affect visual quality in these eyes. A postoperative eye with plano refraction may still have a distorted and rotated image as a result of residual irregular astigmatism.

This study has implications for cataract surgery as well. Cataract surgery with intraocular lens (IOL) implantation attempts to treat the entire refractive error at a single point along the visual axis—the IOL—regardless of the source of the error. In an eye with significant corneal aberrations, either lower order or higher order, this can be problematic. The current standard of care is to neutralize corneal astigmatism with a toric IOL. However, it is not uncommon to encounter eyes in which refractive cylinder remains after toric IOL implantation despite the IOL’s perfect alignment with the corresponding corneal astigmatism. Conversely, eyes with high refractive cylinder but low corneal astigmatism in which a monofocal IOL rather than a toric IOL was implanted may have postoperative refractive cylinder despite low corneal astigmatism. In both post–cataract surgery scenarios, other sources of ORA become clinically relevant. Perhaps the best approach to the management of astigmatism in a cataract surgery patient is to balance the treatment based on the cornea and the ORA. Further work in this area is needed.

In conclusion, anterior cornea–based astigmatic correction procedures, such as LASIK, treat anterior corneal astigmatism much more effectively than astigmatism arising from non-anterior corneal sources. When the astigmatic correction necessary to counter the ORA is applied to the anterior cornea, it is not located on the same point along the visual (z) axis as the source of the ORA, resulting in less effective management of the ORA. When significant ORA is identified preoperatively, it may be prudent to incorporate vector planning into the surgical plan or to recommend against corneal refractive surgery and consider a lens-based procedure. If LASIK is performed without regard to the location of the z-axis and the source of preoperative astigmatism, the compensatory astigmatism created on the cornea to neutralize the internal ORA may result in unsatisfactory visual outcomes by creating excessive corneal aberrations. Such aberrations may contribute to further problems after subsequent cataract surgery or lens extraction later in life.

As the fields of refractive surgery and cataract surgery merge and corneal topography and wavefront aberrometry become complementary tools, treating an aberration at its source along the visual axis may be the direction of the future.

REFERENCES