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Cataract & Refractive Surgery Today

Highlights From the EVO VISIAN ICL EXPERTS SUMMIT 2018

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EVO Visian ICL: The Ideal Treatment for Low and Moderate Myopia

The technology is predictable, reproducible, effective, and, if needed, removable.

BY ALAA ELDANASOURY, MD, FRCS

Refractive surgery has come a long way since it was first conceptualized in 1948 by Polish missionary and ophthalmologist Father Waclaw Szuniewicz and since the earliest surgical procedures were performed by Barraquer in 1964. In the early days of refractive surgery, when attempts were made to modify the cornea with keratomileusis and keratophakia, one could not predict surgical outcomes or promise patients the exceptional vision that can be achieved today with modern refractive surgery.

Fast forward to the current time, when we have a multitude of refractive surgery treatments that we can offer to our patients—among them LASIK, advanced surface ablation, small-incision lenticule extraction (SMILE), corneal inlays, refractive lens exchange, refractive cataract surgery, and phakic IOLs. With these new techniques and the advances in technology that have occurred throughout the years, we have achieved a reduced rate of complications associated with refractive surgery, such as patient discomfort and postoperative refractive regression.

But in this time, we have also learned that certain treatments are better geared toward certain patients. I believe that the EVO Visian ICL (STAAR Surgical) is the ideal treatment for the correction of low and moderate myopia, the reason being that the procedure is predictable, reproducible, effective, and, if needed, removable. As opposed to laser vision correction procedures that affect corneal biomechanics,[1] implanting the EVO Visian ICL does not alter the mechanical or optical properties of the central cornea, thereby allowing us to perform additional procedures in the future if and when necessary.

CRITERIA FOR EFFECTIVE SURGERY

There are three main requirements of an effective refractive surgery procedure: (1) efficacy, (2) safety, (3) removability and preserving the cornea for future surgical procedures. The EVO Visian ICL checks all three boxes.

Efficacy. With the EVO Visian ICL, patients can achieve excellent distance UCVA and manifest refraction.[2] This is repeatable, boosting my confidence that I can reproduce quality results in the vast majority of patients.

Safety. As mentioned previously, the EVO Visian ICL does not alter the mechanical or optical properties of the central cornea. Clinical outcomes demonstrate preservation of patients' distance BCVA and quality of vision.[2] The Collamer material used in the ICL has passed the test of time and proved to be biocompatible with intraocular structures.[3,4]

Removability and preserving the cornea. In the rare case a postoperative complication occurs, a patient is unhappy with his or her quality of vision postoperatively, or years down the road another procedure is required such as cataract surgery, the EVO Visian ICL can be easily removed. To provide some context, since implanting the versions of the Visian ICL with the KS-Aquaport—a tiny hole in the center of the ICL to promote a more natural flow of aqueous humor—I have not needed to explant a single ICL in more than 500 cases due to cataract formation. Prior to the availability of that feature, in the more than 1,000 ICLs I had implanted, I only removed four lenses due to peripheral touch. After phacoemulsification, all four patients were very satisfied with their vision.

In the rare case of the need for removal, the EVO Visian ICL can be removed and, because the cornea is preserved, outcomes with additional surgeries are not compromised.

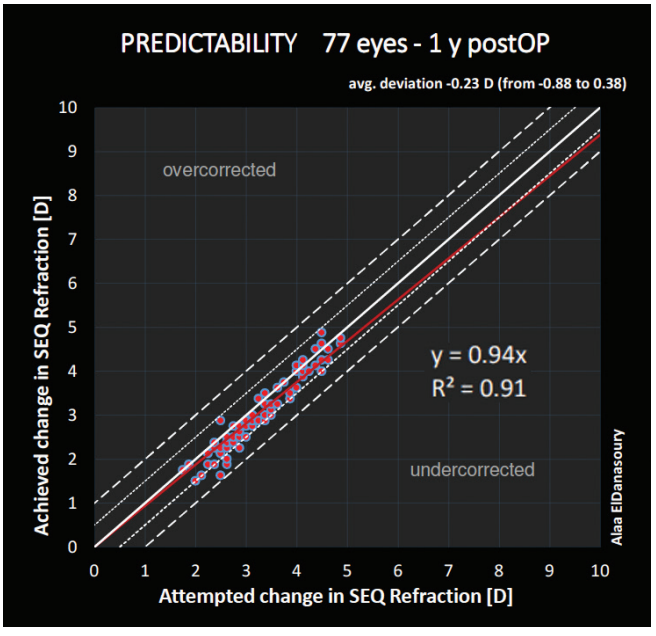

Figure 1. Attempted versus achieved MRSE at 1-year postoperative.

CLINCAL OUTCOMES

We recently evaluated the EVO Visian ICL for myopic correction below -5.00 D against the criteria for an ideal refractive surgery procedure. The retrospective study included 77 consecutive eyes that received either the EVO+ Visian ICL or the EVO+ Toric Visian ICL, both of which have an increased optical diameter. Preoperatively, all patients had a distance BCVA of 20/20 or better, were good candidates for laser vision correction, and were available for 1-year follow-up. The refractive goal in all cases was emmetropia. Using the three criteria for effective refractive surgery outlined above, the clinical outcomes of this study are discussed.

Efficacy. Preoperatively in this cohort, the mean refractive spherical equivalent (MRSE) was -3.51 ±0.85 D (-5.00 to -1.63 D). By 1-year postoperative, MRSE improved to -0.23 ±0.23 D (-0.88 to 0.38 D). The attempted versus achieved change in MRSE is shown in Figure 1; the average deviation was -0.23 D (range, -0.88 to 0.38 D). Furthermore, 61% of eyes were within ±0.25 D of intended correction, 95% were within ±0.50 D of intended correction, and 100% were within ±1.00 D.

With regard to predictability of astigmatic correction, the mean refractive cylinder improved from -1.80 ±0.66 D (range, -4.25 to 0.00) preoperatively to -0.41 ±0.34 D (range, -1.25 to 0.00) at 1 year postoperatively for all subjects (Figure 2). When looking at the 25 subjects with the Toric ICL, the mean refractive cylinder improved from -2.6 D ± 0.91 D (range, -4.25 to -1.00) preoperatively to -0.32 D ± 0.42 D (range, -1.25 to 0.00) at 1 year postoperatively (Figure 2).

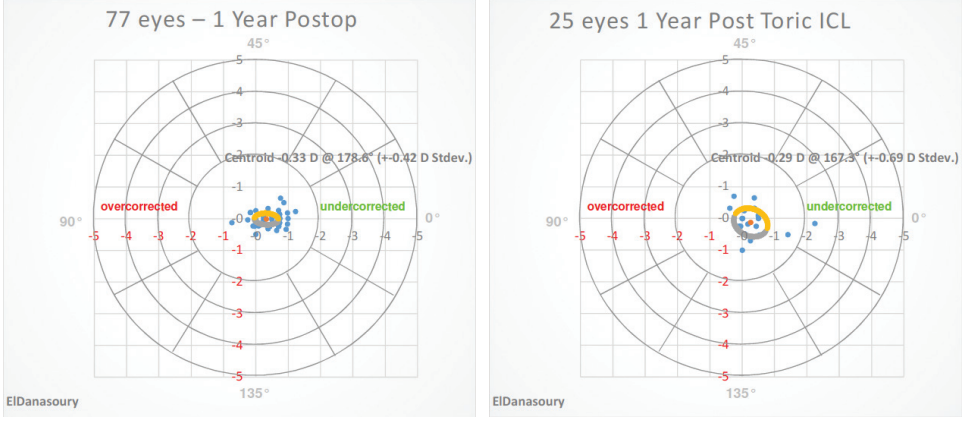

Figure 2. Double-angle cylinder plot (axis normalized), depicting the predictability of astigmatic correction with the EVO Visian ICL (left) and the EVO Visian Toric ICL (right).

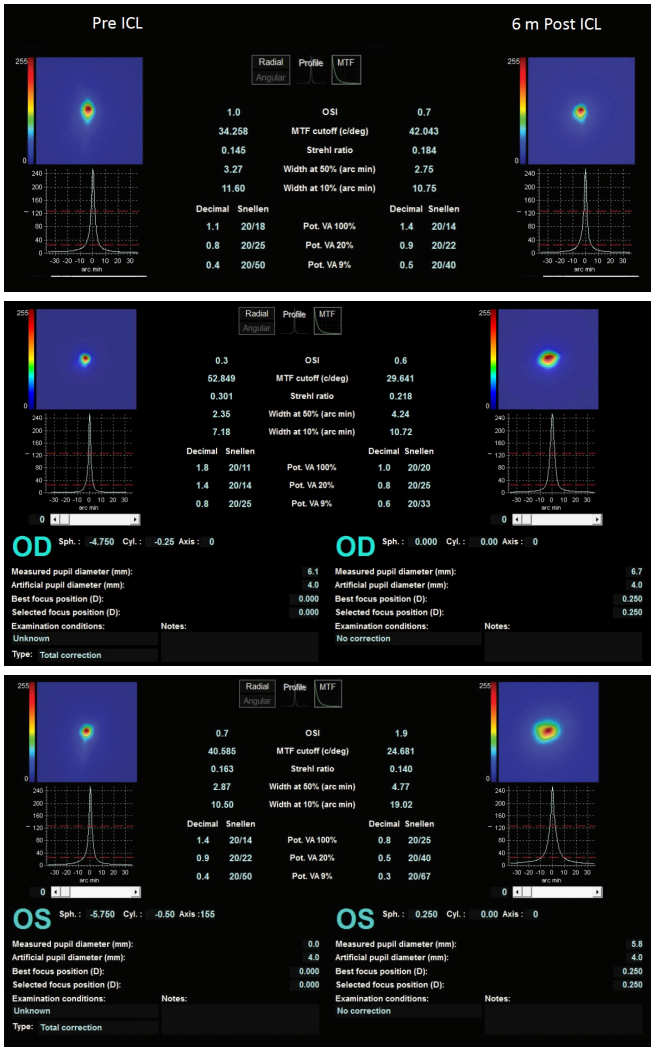

Figure 3. Scatter plots preoperatively and 6-months postoperatively with the EVO Visian ICL (top), compared with pre- and postoperative scatter plots with LASIK (middle), and SMILE (bottom).

In 96% of patients, the 1year postoperative distance UCVA was 20/20 or better and in 32% it was 20/16 or better. This improved from DCVA of 100% and 16% of patients, respectively, prior to implantation of the EVO Visian ICL.

Safety. The safety index in our clinical evaluation, with regard to changes in distance BCVA, was 1.2. In total, 55% of patients remained unchanged and 36% gained 1 line. Only 9% lost 1 line. We also evaluated the preservation of quality of vision by looking at light scatter (Figure 3) and by evaluating the root-mean-square higher-order aberrations, and Coma over time as well as the mean higher-order MTF over time (Figure 4).

Removability and preserving the cornea. At 1 year postoperative, there were no visually threatening complications in this cohort, the cornea has been preserved. Should the EVO

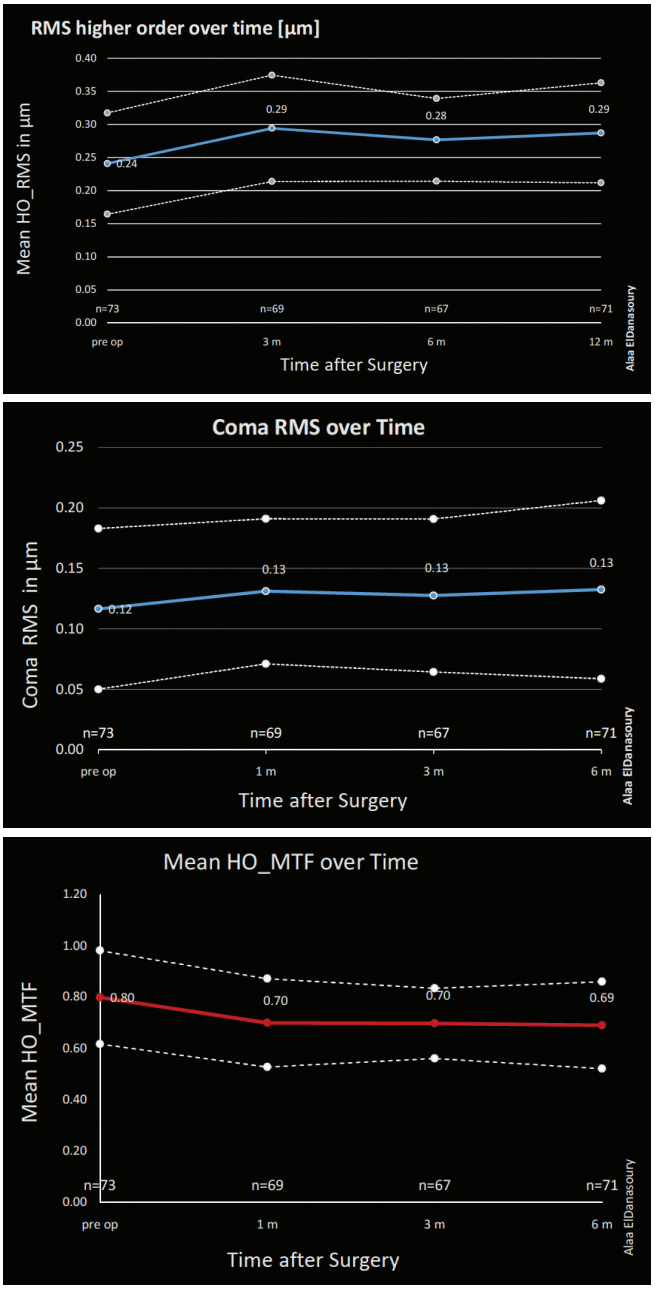

Figure 4. Root-mean-square higher-order aberrations over time (top) and root-mean-square coma over time (center) and mean higher-order aberrations MTF over time (bottom).

Visian ICL need to be removed in the future for additional surgeries, future treatment options remain open because the optical properties of the central cornea have not been altered.

MAKE THE COMPARISON

So why is the EVO Visian ICL the best choice for my patients with low to moderate myopia? In addition to the excellent visual and refractive results that patients

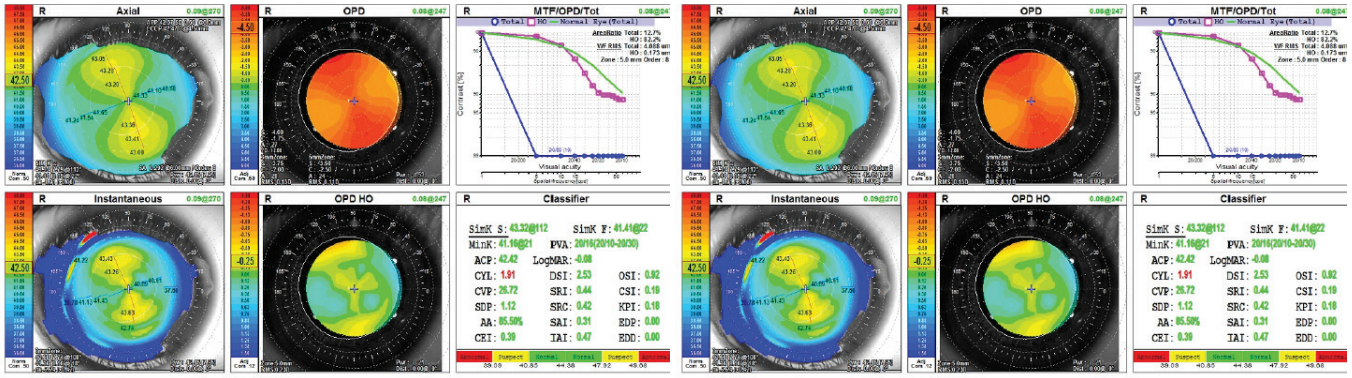

Figure 5. Preoperative (left) and postoperative (right) topography maps from a patient who had undergone LASIK. Note the presence of ectasia in the 3-year postoperative assessment.

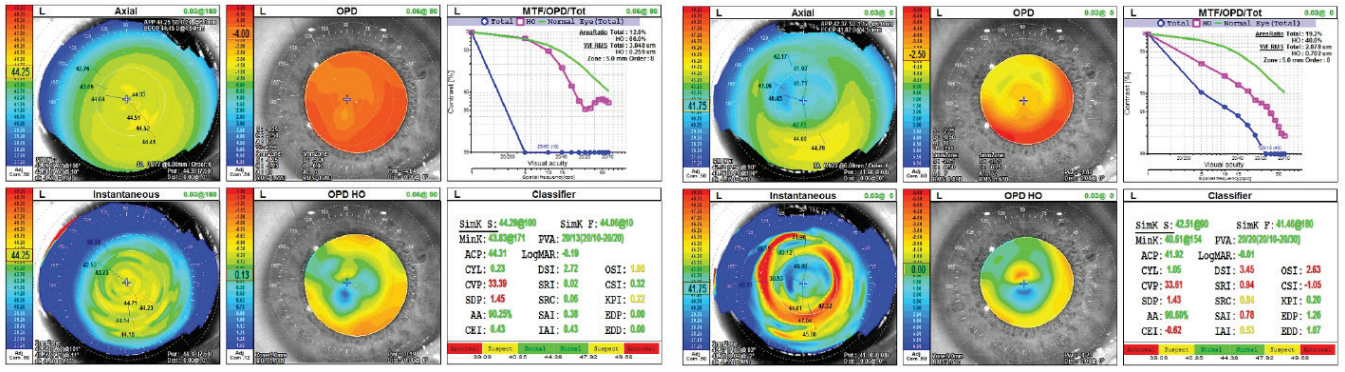

Figure 6. Preoperative (left) and postoperative (right) topography maps from a patient who had undergone SMILE. Note the presence of corneal changes at the 3-month postoperative assessment.

can achieve postoperatively, implanting a phakic IOL does not change the center of the cornea (unlike laser vision correction) which prevents weakening of this structure. The Collamer material of the ICL, which is exclusive to STAAR Surgical, also minimizes inflammation, flare, and cellular reaction.[3,4]

In all refractive surgery there is a risk. In my opinion, the worst-case scenario with the ICL is cataract formation, which as I mentioned previously in this article, has occurred very rarely during my 20-year experience with the ICL and has not happened since I started using the models with the KS-Aquaport. Where a cataract does occur, the ICL is easily removed, allowing for cataract surgery. Sizing issues could also occur, but that requires a simple ICL exchange procedure. In my experience with 500 implants, the incidence of lens exchange with the Visian ICLwith the KS-Aquaport is 0.6%.

On the other hand, the worst-case scenarios with LASIK as well as with SMILE, are postoperative ectasia (Figure 5) requiring CXL, intrastromal corneal ring segments, or possibly deep anterior lamellar keratoplasty; additionally with SMILE is a retained lenticule fragment, inducing corneal changes (Figure 6) and requiring a customized ablation or possibly a DALK procedure.[5] Lastly, implantation of EVO Visian ICL will not induce or worsen dry eye disease[6], which is an important benefit for any refractive patient.

CONCLUSION

With a proven history for longer than 20 years and more than 900,000 ICLs implanted worldwide, the EVO Visian ICL is my go-to choice for most of my refractive surgery patients—including patients with low and moderate myopia. ■

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Corneal Biomechanics in Refractive Surgery

An overview of what we know today.

BY CYNTHIA J. ROBERTS, PHD

Why should refractive surgeons care about corneal biomechanics? The answer is simple: Because corneal biomechanics play a significant role in visual outcomes after any procedure that relies on removing or incising the cornea lamellar tissue to produce a change in the shape of the cornea. Outcomes from today's conventional, wavefront-guided, and topography-guided laser vision correction procedures, and even new refractive surgery procedures such as small-incision lenticule extraction, are affected by corneal biomechanics. Further, even if the cornea is incised without removing tissue, such as in an arcuate incision, the structure of the cornea still changes, which drives the response observed.

But what does that mean exactly?

When corneal tissue is removed through excimer laser ablation or by femtosecond-based procedures, the structure of the cornea changes. Changing the structure of the cornea changes the shape—due to the biomechanical response—and changing the shape changes vision and, therefore, it changes outcomes. Long story short, changing the structure of the cornea changes the shape of the cornea; the two are intimately connected.

The bottom line is that corneal biomechanics is important because any time the structure and shape of the cornea change, the result will be changes in vision.

A BRIEF HISTORY

In 2000, I published an editorial in the *Journal of Refractive Surgery*, "The Cornea is Not a Piece of Plastic," in which I discussed the potential of refractive surgery and the elusive goal of 20/10 aberration-free vision.[1] At the time, the promise of wavefront-guided procedures was that, if the optical aberrations of the cornea could be measured, we could then correct those aberrations and actually provide patients with so-called perfect vision.

We all know that did not happen, and today many surgeons don't even perform wavefront-guided procedures. The surgical mindset changed very quickly from aberration-free vision to reduced surgically induced aberrations.

Even today, we still cannot achieve the perfect corneal shape or the perfect wavefront structure with tissue removal in laser refractive surgery. But can we achieve an optimal shape and structure? To answer this question, one must look at the biomechanical response to refractive surgery in more detail (Figure 1).[2] There are two possible biomechanical responses after refractive surgery: stable and unstable. A stable response is what typically occurs with procedures that require corneal tissue to be removed or the cornea to be incised. In an unstable response, a potential biomechanical decompensation occurs that in turn causes progressive ectasia.

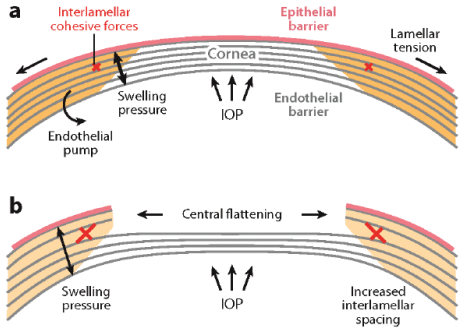

Figure 1. The biomechanical response to refractive surgery.[2] *Reproduced with permission from Annual Reviews.*

A REVIEW OF CORNEAL BIOMECHANICS

We have extensively studied biomechanics in order to better understand the effects of refractive surgery on the cornea. In one study, we compared composite difference maps of 2,380 myopic patients treated with spherical ablation profiles in Hong Kong in the 1990s (Figure 2).[3] Even though the effect that we found was exaggerated because of the spherical ablation profile, it still exists with the aspheric profiles that are in use today. We found that all regions of the cornea showed statistically significant differences from their preoperative states and, there were areas of increased pachymetry in the outer transition zone, where tissue was actually removed.

Figure 2 depicts the results.[3] The blue areas indicate a reduction in curvature; the red areas, which are outside of

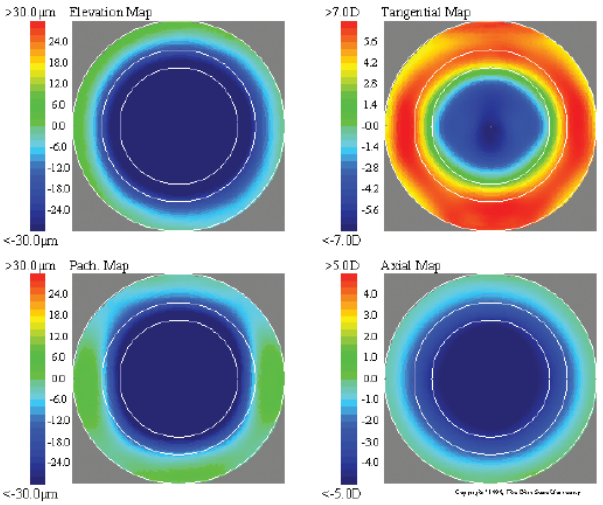

Figure 2. Composite difference maps from 2,380 myopic patients showed statistically significant difference from the preoperative state in all regions of the cornea.[3] *Reproduced with permission from Kugler Publications.*

the optical zone but within the transition zone, indicate an increase in curvature; and the green circle indicates zero change in curvature. In essence, the whole cornea is affected by the ablation, meaning the cornea, as an organ, responds to the change in its structure due to the internal loading with IOP.

We also studied the composite difference maps by levels of correction (fewer than 2.00 D, 2.00 to 4.00 D, 4.00 to 6.00 D, 6.00 to 8.00 D, 8.00 to 10.00 D, and more than 10.00 D; Figures 3A through 3F, respectively).[3] We found that the lower the correction, the more modest the reduction in central pachymetry and curvature. Therefore, as the level of correction increased, the central flattening, the peripheral steepening, and the peripheral corneal thickening increased. Further, the cornea cannot distinguish between where the optical zone ends and the transition zone starts—it only knows where tissue has been removed.

In a myopic procedure, the corneal response enhances the effect of the procedure and induces greater central biomechanical flattening. With a hyperopic procedure, however, the corneal response reduces the effect of the procedure. Both procedures induce unintended paracentral and peripheral shape changes. The result is aberrations, specifically spherical aberration in myopic procedures (This is what hampered wavefront-guided approaches because they could not conquer spherical aberration since it is induced biomechanically.).

SPHERICAL ABERRATION

For quite some time, it was thought that the loss of ablation efficiency in the periphery—because of the slope of the cornea relative to the laser beam—was the cause of spherical

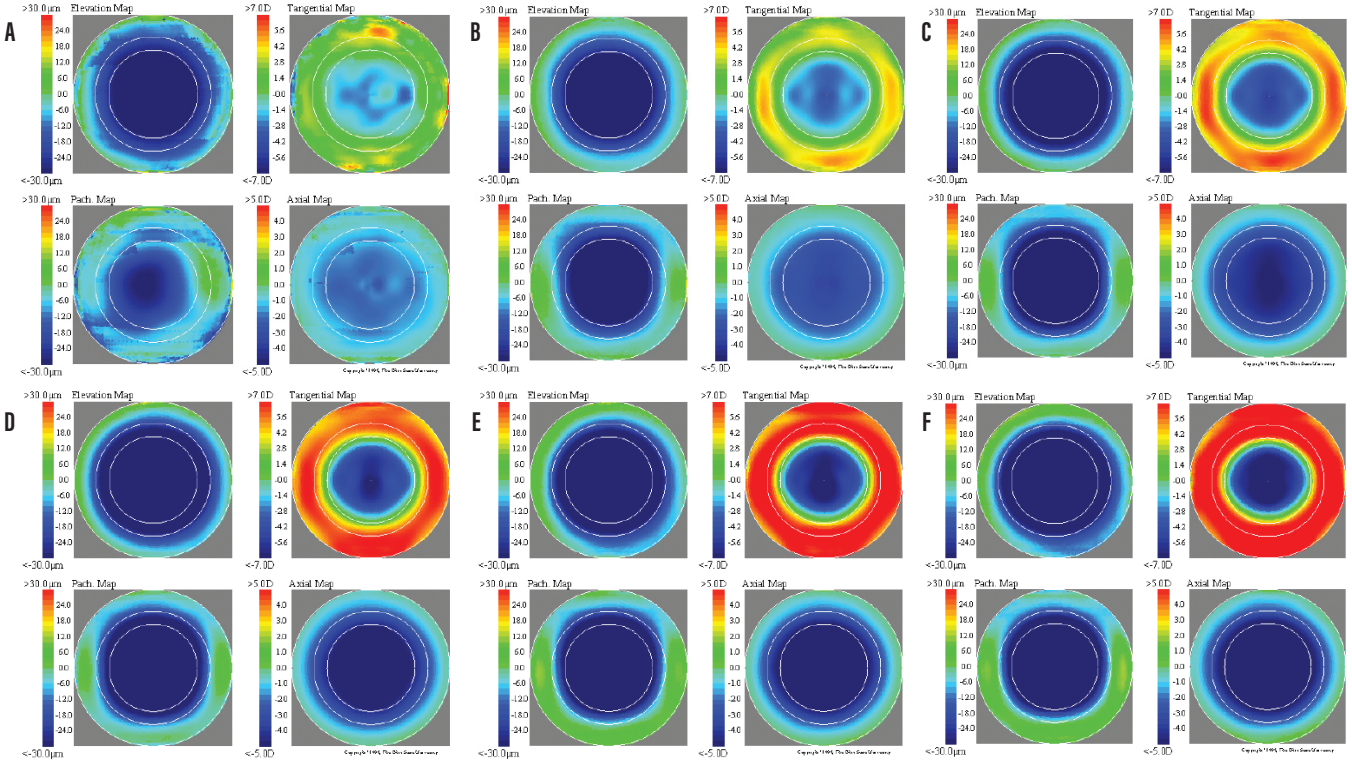

Figure 3. Composite anterior difference maps in patients with a myopic correction fewer than 2.00 D (A); from 2.00 to 4.00 D (B); from 4.00 to 6.00 D (C); from 6.00 to 8.00 D (D); from 8.00 to 10.00 D (E); and more than 10.00 D (F).[3] *Reproduced with permission from Kugler Publications.*

aberration induction. However, this could be compensated by simply adding more laser energy pulses in the periphery during treatment.

An additional concept is that biomechanical changes—specifically the peripheral thickening and steepening of the cornea—induce spherical aberration. Studying the contralateral eyes of patients who had undergone LASIK with two laser platforms (one in each eye), my colleagues and I were able to show that less spherical aberration was induced with the ablation profile that had a larger transition zone. In other words, the transition zone is not neutral: Wherever the ablation occurs, the corneal structure is altered and the shape is modified.[4]

Figure 4 shows the average 6-month postoperative tangential maps of 30 eyes enrolled in the study. Regardless of the laser platform used for the ablation, the second-order outcomes (sphere and cylinder) were identical; however, what was different is that one of the lasers induced greater spherical aberration due to a greater increase in the corneal curvature in the periphery.

Remember, the cornea only knows where tissue has been removed, not which laser is used, so the difference in those two profiles was the transition zone. The laser profile that produced greater spherical aberration induction had smaller transition and ablation zones, leaving greater peripheral fibers to generate the biomechanical response.

MANIPULATING THE BIOMECHANICAL RESPONSE

Switching from a 5-mm ablation zone to a 6-mm ablation zone can dramatically decrease the variability of the biomechanical response. This is because, with a larger zone, more of the peripheral fibers that generate the biomechanical response will be removed.

It is widely accepted that the creation of a LASIK flap—the nearly circumferential severing of corneal lamella to a maximum depth of approximately 160 µm—changes the corneal structure and, therefore, produces a biomechanical response and changes the visual outcomes. But what about a reducing the thickness of the flap to 90 µm? Using a corneal tissue culture model, Knox et al[5] measured the increase in central corneal strain (ie, stretch) between 90- and 160-µm thick LASIK flaps, comparing sidecuts with delamination only and found that delamination had very little impact on corneal biomechanics. Almost all of the change in strain was generated by how deep the side cuts were.

In another study using an inverse finite element analysis to compare femtosecond lenticule extraction (FLEx) flaps to small-incision lenticule extraction (SMILE) cap cuts in 10 eyes of five patients,[6] the FLEx flap produced 49% (range, 2–87%) greater mean reduction in stromal collagen fiber stiffness within that flap region than the contralateral cap region in SMILE. Further, SMILE resulted in lower stresses and deformations within the stromal bed.

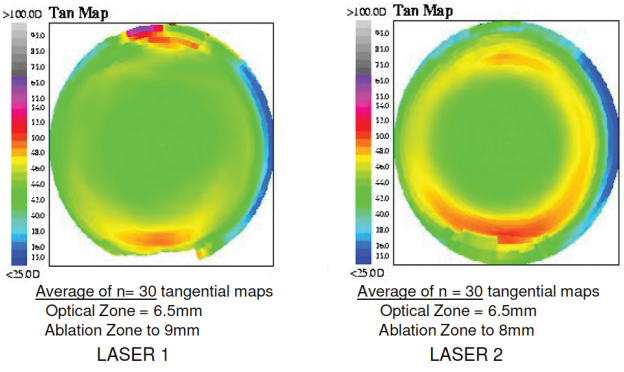

Figure 4. Average tangential maps of 30 eyes treated with two different ablation profiles, one in each eye.[4] *Reproduced with permission from Elsevier.*

TISSUE REMOVAL IS TISSUE REMOVAL

From these study results, it is clear that SMILE has biomechanical advantages over LASIK. However, the primary biomechanical effect of any laser refractive procedure is tissue removal to generate the desired refractive effect. SMILE does not get away from this; it still requires tissue removal. So if you would not perform LASIK because you think that it puts the cornea at risk, you should not perform SMILE. Whether tissue removal occurs under a flap, under a cap, or even at the surface is a secondary effect, and the major effect is how much tissue is being removed.

Finally, central corneal thickness after laser refractive surgery will also affect IOP measurement error. A thin cornea will require less force to applanate than a thick cornea, independent of the true IOP. Therefore, a high IOP measurement may simply mean that the cornea is thick, leading to overestimation. Conversely, a low IOP measurement may simply mean that the cornea is thin, leading to underestimation.

The modulus of elasticity determines the relationship between central corneal thickness and IOP measurement error. The central corneal thickness has a strong influence on IOP measurement in a stiff cornea, but it has much less impact on IOP measurement in a soft cornea. Although an average drop in IOP of 2 mm Hg after laser refractive surgery has been reported in the literature; in reality, the range is much larger, 22 mm Hg, from -12 to 10 mm Hg of change in measured IOP. As a result, applanation tonometry is worthless after laser refractive surgery, and the reason may be due to the exaggerated pachymetry profile, which invalidates Goldmann's equations.

AN ALTERNATE APPROACH

An alternate approach to refractive correction is phakic IOLs. With the EVO Visian ICL (STAAR Surgical) and other phakic IOLs, there is no tissue removal, no effect on IOP measurements, and only a minimal impact on corneal biomechanics. There is no need to talk about ways to interpret biomechanical measurements with phakic IOLs because the procedure does not require tissue removal.

In conclusion, we know the cornea is not a piece of plastic, and we know that with the EVO Visian ICL, the risk for biomechanical changes in the cornea is reduced compared with procedures that either remove or incise the cornea lamellar tissue to produce a change in the shape of the cornea.

I have heard from refractive surgeons that ICL patients are very happy, and I would propose it is because the quality of vision they have is better than the quality of vision experienced by LASIK, SMILE, and PRK patients. This is because, with phakic IOLs, there is no induction of corneal aberrations since no tissue was removed. With an ICL, the patient gets exactly what correction is in the lens. There is no need to worry about a response to it, and that's why I think patients are so happy. ■

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Dry Eye Disease, Meibomian Gland Dysfunction, and Laser Vision Correction: What We Know and Don't Know

Long-term contact lens wear and laser vision correction procedures can both affect the ocular surface.

BY SHERI ROWEN, MD

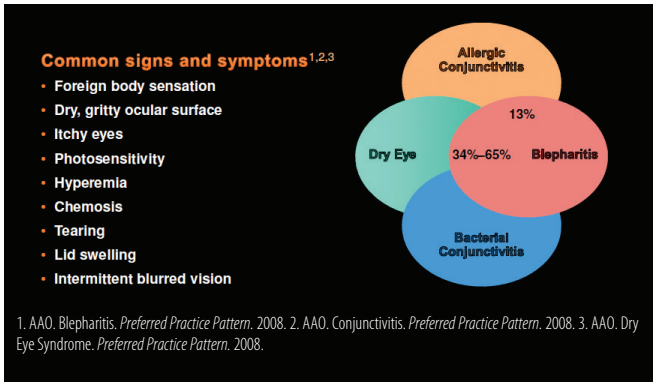
I participated in the early US FDA trials of the Visian ICL (STAAR Surgical), so I have a lot of experience with this technology and have watched the evolution of the ICL in the United States from its infancy to the recent FDA approval of the Visian Toric ICL. I have also been heavily involved with LASIK and have performed a multitude of those procedures. Between these experiences and after treating thousands of patients at my clinic who present with dry eye disease (DED) and other ocular surface issues, I now have a better understanding of what happens to our patients' eyes when we operate on them and also what happens when they have a history of long-term contact lens wear and dry eye disease.

What I know now, and what most refractive surgeons all agree with, is that laser vision correction should not be performed in patients with untreated DED, as the procedure will exacerbate their condition. But there are also plenty of other considerations that need to go into the decision to perform refractive surgery in our patients. In this article, I review what we know about DED, what we didn't know before about DED, and how the diagnosis of DED should influence our decision-making process in refractive surgery.

WHAT WE KNOW

We know that about 344 million people worldwide have symptoms of DED (Figure 1). In the United States alone, 30 million people suffer from DED, but only 16 million of those are diagnosed, and only 1.5 million are treated with a prescription. So, in short, DED is underdiagnosed and undertreated. This same trend can be seen in other parts of the world as well.

We also know that the symptoms of DED can be variable and disruptive. According to the recent Dry Eye Workshop II (DEWS II) study,[1] DED is now considered a multifactorial disease of the ocular surface, characterized by the loss of homeostasis and accompanied by ocular symptoms in which tear film instability and hyperosmolarity are present, ocular surface inflammation (Figure 2) and damage occur, and neurosensory abnormalities play etiological roles.



1. AAO. Blepharitis. *Preferred Practice Pattern*. 2008. 2. AAO. Conjunctivitis. *Preferred Practice Pattern*. 2008. 3. AAO. Dry Eye Syndrome. *Preferred Practice Pattern*. 2008.

Figure 1. The signs and symptoms of DED.

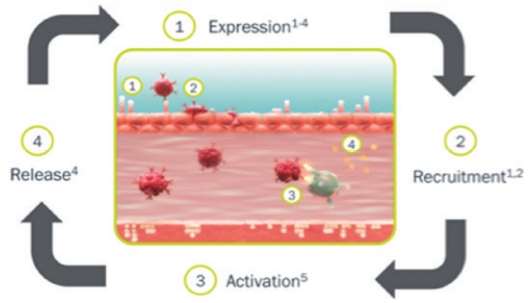


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Figure 3. ICAM-1 binding mediates a self-perpetuating inflammatory cycle in DED.

WHAT WE DIDN'T KNOW

What we didn't know before, but we know now is that there is a higher-than-expected incidence of DED in the younger population, which is likely related to increased screen time and long-term contact lens wear. In these patients, the pathophysiology of DED induces hyperosmolarity and initiates a vicious cycle of ocular inflammation. We also understand females are at a higher risk than males to experience symptoms of DED, but we see the rising incidence of affected males.

There is now also an understanding that the tear film is not a three-layer system, but rather a two-layer system composed of a muco-aqueous layer and a lipid layer. And although we always knew that two types of DED exist—aqueous-deficient and evaporative—today we better understand each of these and have actually identified their unique causes. Studying staining patterns can sometimes help to paint a wonderful picture of the etiological causation of DED. Additionally, we also can now visualize the actual diseased meibomian glands.

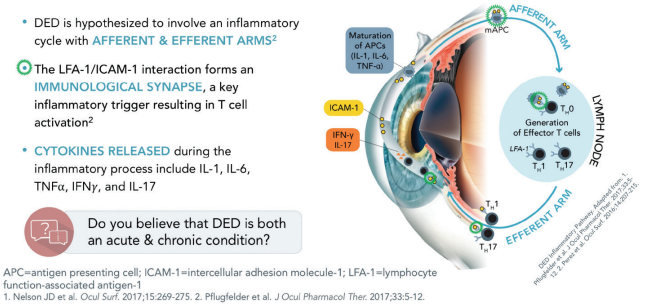
Prior to the release of the DEWS II study, we also did not fully understand the mechanisms of action at play with ocular inflammation. For starters, we didn't know about the roles of intercellular adhesion molecule 1 (ICAM) and lymphocyte function-associated antigen 1 (LFA-1) in DED. Now, however, we understand that DED has afferent and efferent arms of inflammation, causing ICAMs to pop up on the ocular surface. These ICAMs go back through the afferent arm to the lymph nodes, sensitizing and activating naïve T-cells to produce LFA-1 on the ocular surface and triggering a cycle of recruitment, activation, and release of cytokines. This is where the inflammation happens (Figure 3).

Now that we understand the mechanism of action of ocular inflammation, we have come to the brutal realization that symptoms and signs of ocular surface disease in general and DED specifically are underdiagnosed. According to a study by Trattler et al,[2] 50% of people who presented for cataract surgery had DED but were undiagnosed. Although 77% had positive corneal staining, only 22% had a prior diagnosis (Figure 4). In a separate study from Duke University,[3] 64% of participants had abnormal ocular surface disease index (OSDI) scores, and only 16% had been identified with DED (Figure 4).

The patient-reported outcome with LASIK (PROWL) I and II studies[4] found that 51% of patients (average age, 30 years) had an abnormal baseline OSDI score, even before they had LASIK. In these two studies, about two-thirds of contact lens wearers were female, and the average age of dry eye symptom onset in this population was about 27 years old. In total, 44% reported significant symptoms and 59% had abnormal osmolarity. Many of these patients presenting for LASIK obviously had prior DED.

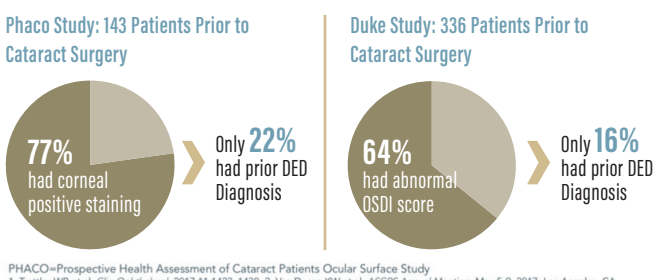
In my own series, I started seeing individuals with a history of dry eye intolerance who had undergone or came in for LASIK. We learned that many of the post-LASIK patients already had DED prior to surgery, and the procedure tipped

Image courtesy of Shire



APC=antigen presenting cell; ICAM-1=intercellular adhesion molecule-1; LFA-1=lymphocyte function-associated antigen-1
1. Nelson JD et al. *Ocul Surf*. 2017;15:269-275. 2. Pflugfelder et al. *J Ocul Pharmacol Ther*. 2017;33:5-12.

Figure 2. Inflammation is a continuous cycle implicated in DED.



PHACO=Prospective Health Assessment of Cataract Patients Ocular Surface Study
1. Trattler WB et al. *Clin Ophthalmol*. 2017;11:1423–1430. 2. Van Dusen KW et al. *ASCRS Annual Meeting*; May 5-9, 2017; Los Angeles, CA.

Figure 4. According to results of several studies, the signs and symptoms of DED before cataract surgery may be underdiagnosed.

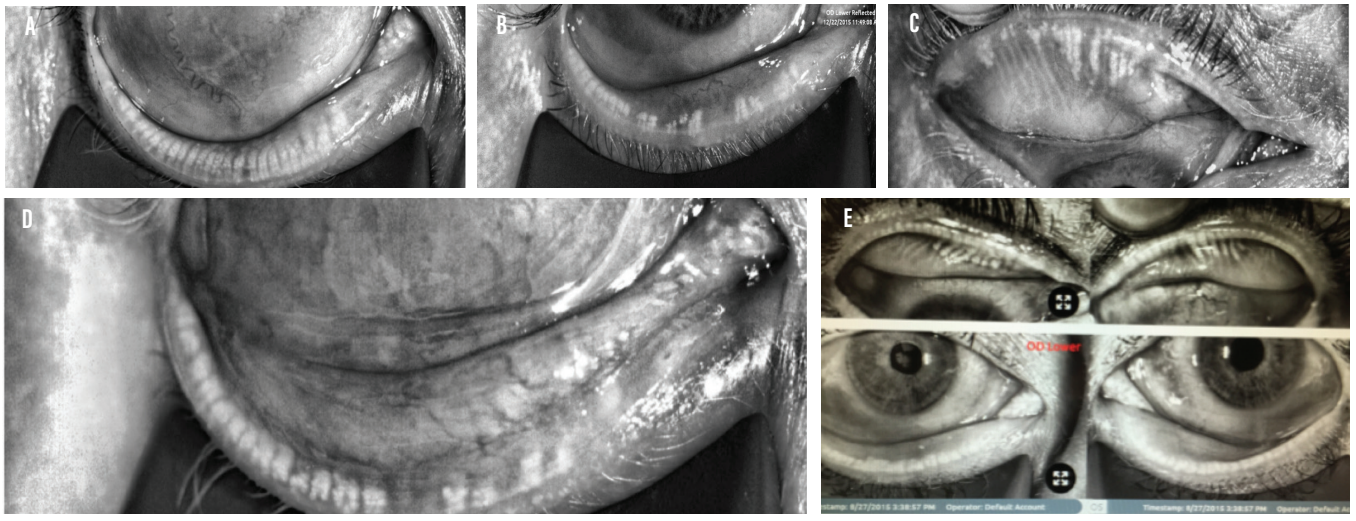


Figure 5. A 50-year-old woman with a 25-year history of DED.

them over into full-on chronically symptomatic DED. It wasn't necessarily the LASIK, but was the prior history of dry eye that increased their risk of having DED. And it starts early—much earlier than we ever imagined. This is not just the disease of the menopausal woman (Figure 5).

WHAT IT ALL MEANS

Results from these and other recent studies paint a vivid picture that many of our patients who present for refractive surgery and even cataract surgery already have DED. We know that long-term contact lens use is a risk factor for DED, and we also know that historically many practitioners have been inclined to perform refractive surgery on these patients because they have become contact lens intolerant. Although in the past we may have considered laser vision correction to be the logical choice, especially for low myopes, we must now consider the presence of DED when selecting a refractive procedure for our patients.

Now armed with better information about the mechanisms of action at play with DED and about the prevalence of DED among the general population, we can be more prepared in our dialogues with patients, possibly steering them away from LASIK, which can exacerbate DED, and toward a refractive surgery procedure that will not induce more DED symptoms.

The bottom line is that we have to look, listen, and then treat our patients. The easiest way to accomplish this is to use the OSDI or Standardized Patient Evaluation of Eye Dryness (SPEED) questionnaires on every patient who walks into your office. In my practice, I also have patients complete an ocular assessment that goes over all of the things that I need to know to better understand the patient's ocular history and to identify preexisting DED: whether he or she has had prior ocular surgery, medication use, osmolarity, MMP-9, fluorescein or lissamine green staining, meibum expression, and LipiView (TearScience). I also believe in educating patients with images; when this is done, they will be very willing to cooperate with your treatment plan.

CONCLUSION

Most patients who present with contact lens intolerance already have DED. In the past, those patients have traditionally received LASIK, and when their symptoms worsened, it was assumed that LASIK was the cause. This is not entirely true: The DED was already there, but the LASIK procedure did exacerbate their symptoms.

So, we have to better educate our patients on the dangers of extended contact lens use, the overuse of digital devices, and the importance of meibomian gland care. We must educate patients about DED early on, so they will be prepared to make the proper choices about refractive correction, and we need to properly diagnose and treat them when we find it.

Following such a protocol will help us to determine who indeed is a candidate for any refractive treatment. In my experience, the Visian ICL family of lenses are are great for patients with DED who were previously diagnosed and even undiagnosed. This technology has much less effect on DED symptoms, as only a small incision is created for lens insertion. ■

1. TFOS International Dry Eye WorkShop (DEWS II). *Ocular Surf.* 2017;15(6):269-650.
2. Trattler WB et al. *Clin Ophthalmol.* 2017;11:1423-1430.
3. Van Dusen KW et al. Paper presented at: the 2017 Annual ASCRS Meeting; May 5-9, 2017; Los Angeles.
4. Eydelman M, Hilmantel G, Tarver ME, et al. Symptoms and satisfaction of patients in the Patient-Reported Outcomes With Laser In Situ Keratomileusis (PROWL) studies. *JAMA Ophthalmol.* 2017;135(1):13-22.

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