generally swollen. In an in vitro study, before starting in vivo femtosecond laser-assisted Descemet's stripping endothelial keratoplasty (FS-DSEK), scanning electron microscopy (SEM) was performed to identify the depth of the cut in the corneoscleral rim after trephination of the posterior lamellar disk (PLD). In 6 donor eyes, the preoperative mean corneal thickness was 779 μm ± 73 (SD). The relative depth of the lamellar plane identified by SEM was at approximately 47% ± 5% μm from the anterior side of the donor cornea. Based on this calculation and assuming that the electron microscopy fixation process had the same dehydration effects on the different compartments of the cornea, the mean absolute thickness of the PLD was 413 ± 38 μm.

Following FS-DSEK, we measured the thickness of the PLD using optical coherence tomography (OCT) techniques (Visante OCT, Carl-Zeiss Meditec). At 3 months, the mean central corneal thickness was 601 ± 9 μm and the mean thickness of the PLD was 110 ± 9 μm (Figure 1). Clearly, the final deturgescence was the result of organ culture with dextran incubation and the postoperative dehydration by the functioning endothelium.

Many studies have shown a gradual decrease in corneal swelling from the posterior to the anterior side. Müller et al.[1] showed that the tightly interwoven anterior lamellae of the most anterior part of the cornea (100 to 120 μm) are resistant to even extreme hydration states. As far as we know, the exact relationship between deeper stromal layers (middle and posterior stroma) in the cornea and the swelling capacity of these specific depths is unknown. From the data of our preliminary series, it appears that to explain the limited postoperative thickness of the PLDs, the part of the cornea that is resistant to extreme hydration extends deeper than the 100 to 120 μm depth outlined by Müller et al.

Furthermore, we believe that the goal to obtain a predictable thickness for the PLD is probably less important, since a correlation between visual acuity and central corneal thickness after DSEK has not been shown[2] and the insertion of thicker PLDs may be technically easier than of thinner, more fragile PLDs.—*Yanny Y.Y. Cheng, MD, Liesbeth Pels, PhD, Rudy M.M.A. Nuijts, MD, PhD*

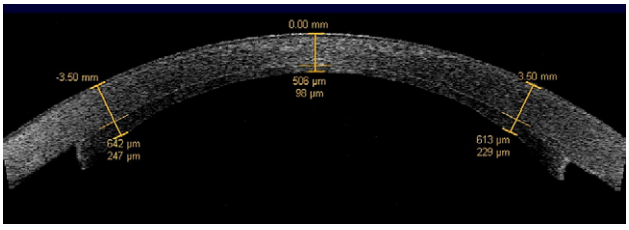

Figure 1. Optical coherence tomography image 3 months after implantation of PLD with FS-DSEK technique.

REFERENCES

1. Müller LJ, Pels E, Vrensen GFJM. The specific architecture of the anterior stroma accounts for maintenance of corneal curvature. Br J Ophthalmol 2001; 85:437–443
2. Price MO, Price FW Jr. Descemet's stripping with endothelial keratoplasty; comparative outcomes with microkeratome-dissected and manually dissected donor tissue. Ophthalmology 2006; 113: 1936–1942

Hoffer barrier ridge concept

In discussing a 360-degree barrier on the posterior surface of an intraocular lens (IOL), Vyas et al.[1] do not mention or refer to the origins of this concept. The Hoffer barrier ridge[2–4] was conceived exactly 30 years ago and first presented publicly in 1979, 28 years ago (K.J. Hoffer, MD, "Posterior Chamber Lens Ridge for Prevention of Capsule Opacification," presented at the 2nd U.S. Intraocular Lens Symposium, Los Angeles, California, USA, April 1979). The concept was intended to decrease the surface area in contact with the capsule and thus increase the pressure at the edge based on the following formula: $P = F/A$, where P = pressure (lb/in^2), F = force, and A = area. To increase the pressure, increase the force and/or decrease the surface area. The purpose was to slow the centripetal progression of pearl migration and create a space between the posterior surface of the IOL and the capsule to prevent traumatic damage to the IOL surface from instrumentation used to perform a posterior capsulotomy (wording from patent.) Two United States patents were issued: US patent 4 244 060, issued January 13, 1981, and US patent RE 31 626, reissued July 10, 1984. The patents have expired, and the concept can be freely used by all.

In the early 1980s, an experimental prototype was fabricated first by Peter LeHaye of Iolab and later by Jim Cook of Cilco. The former was called the Iolab 101-H, the second modification of a posterior chamber IOL (following the Kratz 101-K with angulated loops). Modifying the same concept, CooperVision began marketing the concave posterior IOL and then many others developed variations to get around the patent (riders, partial ridges, bumps), all unsuccessful in concept and legal standing. The ridge IOL was at one time a major portion of the IOLs implanted worldwide. It decreased in popularity for 2 reasons. The first was that to be effective, both loops of the IOL had to be implanted in the capsular bag, something rarely accomplished in the 1980s. With 1 loop out (which was too

often the case), pearls could slide under the elevated IOL edge. The second reason was the eagerness of manufacturers to switch sales from royalty-laden plano-convex ridge IOLs to royalty-free biconvex IOLs (without a ridge). This was promoted on the proposition that a poly(methyl methacrylate) (PMMA) biconvex IOL was optically superior (which is not the case) and that a biconvex IOL could not be made with a ridge, especially of silicone, because the ridge would be damaged on injection. This was proved to be untrue by the commercially available Coburn PMMA biconvex ridge IOLs (implanted successfully for several years) and the AMO silicone ridge IOL injection study. However, by then the manufacturers' marketing skills led to the demise of the use of ridge IOLs until the patent expired. Later, the concept resurfaced without crediting the invention.

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REFERENCES

1. Vyas AV, Narendran R, Bacon PJ, Apple DJ. Three-hundred-sixty degree barrier effect of a square-edged and an enhanced-edge intraocular lens on centripetal lens epithelial cell migration; two-year results. J Cataract Refract Surg 2007; 33:81–87
2. Hoffer KJ. Ridged intraocular lens may lower need for discissions after cataract surgery. Ophthalmology Times April 1981; 6(4):4
3. Hoffer KJ. Glare not caused by ridge [letter]. J Cataract Refract Surg 1990; 16:130–131
4. Hoffer KJ. Five years' experience with the ridged laser lens implant. In: Emery JM, Jacobson AC, eds, Current Concepts in Cataract Surgery; Selected Proceedings of the Eighth Biennial Cataract Surgical Congress. Norwalk, CT, Appleton-Century-Crofts, 1984; 296–299

REPLY: We acknowledge Dr. Hoffer's pioneering contribution to the invention and development of the laser ridge to create space for a capsulotomy with PMMA IOLs, but as explained by Dr. Hoffer, the effect of the laser ridge on epithelial migration was unpredictable and the design slowly fell out of favor. It was the introduction of the sharp square edge[1] and the shrink-wrap effect on the capsule that made it possible to significantly and consistently reduce epithelial migration. In contrast to the laser ridge IOL design, the Rayner C-flex™ IOL design aims to minimize the space between the IOL surface and the capsule and create a 360-degree barrier with a sharp square optic edge and a unique enhanced square edge at the optic–haptic junctions. Our high-resolution clinical photographs consistently show excellent encasing effects around the square and enhanced edges, leading to reduced epithelial migration.—*Ashokkumar V. Vyas, FRCS(Ophth), Peter Bacon, FRCOphth, Rajesh Narendran, MRCOphth*

REFERENCE

1. Nishi O, Nishi K, Sakanishi K. Inhibition of migrating lens epithelial cells at the capsular bend created by the rectangular optic edge of a posterior chamber intraocular lens. Ophthalmic Surg Lasers 1998; 29:587–594

Corneal crosslinking with riboflavin and UVA for the treatment of keratoconus

The article by Caporossi et al.[1] about corneal crosslinking with riboflavin and ultraviolet-A (UVA) for the treatment of keratoconus in combination with the findings of other recent studies offers the scientific community an innovative and probably promising solution for this ectatic dystrophy of the cornea. Because the technique is extremely new and currently under study, we believe that future authors of similar articles should be very careful with the experimental design and the interpretation of findings.

A rather significant issue is that although the authors describe noticeable changes in the UVA, best corrected visual acuity, and K-readings of these patients, they do not give an adequate explanation of why this occurs. It is known that the physicochemical properties of collagen change after corneal crosslinking and the stiffening effect stabilizes keratoconus,[2] but in what way? One possible explanation is that the increase in the rigidity of the collagen causes flattening of the corneal apex.

Another important aspect of the corneal crosslinking technique that should be considered is that crosslinking of collagen is a natural effect of aging.[3] We are actually increasing rigidity by aging the cornea, and the long-term results of that might not be beneficial. Apart from this, we cannot exclude with certainty that the induced rigidity of the cornea will not promote premature ocular rigidity in general, creating future severe ocular problems (for example, age-related macular degeneration[4]). Finally, other ocular parameters such as tear function, corneal sensitivity, and alterations in conjunctival epithelium and goblet cells should be included in future studies.

Understanding the true nature of the corneal crosslinking mechanism will guide us to finding a convenient solution for these patients and maybe improve their quality of life.

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REFERENCES

1. Caporossi A, Baiocchi S, Mazzota C, et al. Parasurgical therapy for keratoconus by riboflavin-ultraviolet type A rays