Among the wide assortment of techniques available for the correction of ametropia, LASIK has obtained considerable international reception over the past two decades and is currently the procedure of choice within the field of refractive surgery. LASIK can result in residual refractive errors causing undercorrection, overcorrection, re-gression, and surgically induced astigmatism. If visually significant, correction requires that an enhancement procedure be performed. Notwithstanding customary efficacy associated with postoperative refractive outcomes, postoperative enhancement rates vary between 5% and 28%. Details concerning postoperative enhancement requirements and related predisposing factors, clinical considerations, and treatment options will be discussed herein.

**METHODS**

An extensive literature search was performed using the PubMed database and Google search engine. Search terms included “LASIK,” “PRK,” “laser-assisted in situ keratomileusis,” “refractive surgery,” “overcorrection,” “undercorrection,” “re-lift,” “mini-flap,” and related terms.

**RESULTS:** The presence of residual refractive error following LASIK is a challenging situation. After excluding anatomical causes (e.g., ocular surface disease, cataract, and macular pathology) and intraoperative flap complications, evaluation of the residual stromal bed must be performed. Depending on the length of time since primary LASIK, procedures such as flap re-lift, flap re-cut, and surface ablation may be performed.

**CONCLUSIONS:** Residual refractive errors can be seen after LASIK surgery and may benefit from an enhancement procedure. Different options are available for enhancement, each requiring proper evaluation and an analytical approach to make the procedure safe and effective.

**ABSTRACT**

**PURPOSE:** To review refractive regression and current therapeutic options for patients who have residual refractive error after LASIK.

**METHODS:** An extensive literature search using PubMed was performed for terms such as “LASIK,” “PRK,” “enhancement,” “overcorrection,” “undercorrection,” “re-lift,” “mini-flap,” and related terms.

**RESULTS:** The presence of residual refractive error following LASIK is a challenging situation. After excluding anatomical causes (e.g., ocular surface disease, cataract, and macular pathology) and intraoperative flap complications, evaluation of the residual stromal bed must be performed. Depending on the length of time since primary LASIK, procedures such as flap re-lift, flap re-cut, and surface ablation may be performed.

**CONCLUSIONS:** Residual refractive errors can be seen after LASIK surgery and may benefit from an enhancement procedure. Different options are available for enhancement, each requiring proper evaluation and an analytical approach to make the procedure safe and effective.

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ETIOLOGY OF RESIDUAL REFRACTIVE ERROR

Undercorrection is a common complication of LASIK. Diagnosis occurs rapidly, and vision remains suboptimal thereafter. Decreased incidences of undercorrection and central island formation have been attributed to improvements in nomogram parameters, a switch from broad beam to scanning excimer laser technology, and improved awareness regarding intraoperative procedural considerations.3,4 Earlier studies noted a positive correlation between the rate of undercorrection and the degree of preoperative refractive error. However, this correlation was not present in a recent prospective study of more than 30,000 eyes. Overcorrection has a lower reported estimated occurrence. It has been positively correlated with the escalating quantity of preoperative spherical equivalent and spherical aberration treated.5

Residual or surgically induced astigmatism may eventually require an ensuing enhancement procedure. It has been demonstrated that patients receiving spherical ablations with low preoperative astigmatism (< 1.00 diopter [D]) had statistically significant increases after ablation. In addition, greater degrees of surgically induced astigmatism were correlated with higher preoperative astigmatic values.6 Other risk factors associated with the development of surgically induced astigmatism include a preoperative keratometric power of greater than 44.00 D and an ablation zone diameter of less than 6 mm.

The underlying etiology of postoperative regression is not fully understood and is likely multifactorial (Table 1). It refers to a gradual tendency to return to the original preoperative ametropic state, whether myopic or hyperopic. Regression generally occurs 3 to 6 months following LASIK, although it can continually progress up to 3 years postoperatively.7,8 Proposed factors contributing to its occurrence include stromal remodeling, epithelial hyperplasia, flap thickness, central corneal thickness,9 lenticular nuclear sclerosis, forward shifting of the cornea, increased intraocular pressure,10,11 and vitreous chamber elongation. Regression is typically found after spherical corrections and is present to a greater degree after hyperopic compared to myopic LASIK.7 In both groups, patients seem to experience an equally distributed increased propensity for postoperative regression with a higher magnitude of preoperative refractive error.3,7 Despite this predominant observation, a recently performed large-scale study of purely myopic patients did not find a positive association between regression and high refractive errors. The authors documented an enhancement rate of only 0.5%.4 They speculated that the sparsity of enhancement procedures within the highly myopic patient population may have been attributable to forfeiture of patient candidacy as a result of inadequate postoperative residual stromal bed thickness.

Epithelial hyperplasia has been well substantiated as a cause of regression in both myopic and hyperopic eyes.12,13 The incorporation of additional epithelial cells within the center or periphery of the cornea results in myopic and hyperopic shifts, respectively, according to the Law of Thicknesses established by Barraquer. The quantity of postoperative epithelial hyperplasia has been demonstrated to increase linearly with progressively higher ablation depths. However, regressive effects resulting from epithelial proliferation appear to affect lower myopes more severely than higher myopes due to a progressively decreasing rate of thickening per each additional diopter of myopia.12

Decreased flap thickness has been hypothesized to contribute to regression due to closer apposition of stromal and epithelial surfaces. When exposed to growth factors intended for the epithelium, stromal keratocytes may differentiate into fibroblasts. These newly formed cells have been postulated to play a role in regression after LASIK. However, a direct comparison of eyes with corneal flap thickness of 100 µm or less and 110 µm or more showed no increased rate of regression within the group containing thinner flaps over a period of 1 year.

Postoperative stromal remodeling has been associated with myopic and hyperopic regression, due to stromal thickening and thinning, respectively. Interesting-
ly, Iversen et al. did not find a significant association between refractive regression and stromal thickness specifically, but rather central corneal thickness over a period of 3 years postoperatively. Development of so-called ‘index myopia’ resulting from nuclear sclerotic cataract formation can induce a myopic shift, representing another potential precipitant of postoperative regression. Risk factors for lenticular nuclear sclerosis include advanced age, smoking, ultraviolet radiation exposure, and diabetes mellitus type 2. Accelerated progression of presbyopia after LASIK has also been documented, with relatively younger patients experiencing a more pronounced alteration in refractive power required for near vision.

Vitreous chamber enlargement in adulthood is another possible etiology of regression. In a longitudinal study of individuals who had not undergone refractive surgery, vitreous chamber enlargement and a myopic shift were found in both myopes (48%) and emmetropes (39%) over a 2-year period. Of significance, the corneal curvatures and lens thicknesses did not significantly change. The cause of enlargement was unknown, but occupation-related accommodation was thought to be a possible etiology.

Multiple factors likely play a role in the incidence and progression of regression. The initial phase within the 3-month to 3-year period is likely due to a combination of epithelial hyperplasia and stromal remodeling. These anatomical changes occur in direct response to surgical manipulation. Regression past this time period often occurs to a lesser degree and is more than likely due to natural phenomena that would have transpired if no surgery had taken place. Likely etiologies include vitreous chamber enlargement and lens nucleus changes. Additional predisposing factors for the development of postoperative regression include older age, pre-existing astigmatism, dry eye, and smaller ablative zone. The annual rate of hyperopic regression has been reported as +0.03 to +0.07 D in low, +0.15 D in medium, and +0.37 D in high hyperopic subsets. The annual rate of myopic regression has been reported as -0.10 ± 0.18 D in moderate and -0.18 ± 0.22 D in high preoperative myopic error.

**RE-TREATMENT EVALUATION**

The initial approach to any patient experiencing subjective change in visual acuity after refractive surgery includes evaluation of the manifest refraction, cycloplegic refraction, dilated funduscopic examination, topographic/tomographic evaluations, underlying systemic comorbidities (ie, hypertension and diabetes mellitus), current medications, postoperative dry eye, and other adnexal abnormalities. A detailed appraisal for the presence of visually influential lenticular nuclear sclerosis and alternative pathological mechanisms conceivably engendering postoperative regression, such as axial elongation and choroidal thinning, should be performed. Suitable candidates for enhancement should demonstrate refractive stability for an interval that comprises at least two separate clinic visits. However, specific parameters have not been thoroughly defined.

**Residual Stromal Bed Thickness**

Accurate determination of residual stromal bed thickness is of paramount importance prior to performing an enhancement procedure. Miscalculations and/or misinterpretations can result in overestimation or underestimation of the residual stromal bed thickness, which increases the risk of ectasia or inappropriate rejection of candidacy for enhancement, respectively. There are currently several devices that can measure corneal thickness, including ultrasound, confocal microscopy through focusing, optical coherence tomography (OCT), slit-scanning tomography, and rotating Scheimpflug tomography. Direct preoperative measurement of residual stromal bed thickness using OCT, confocal microscopy, or very high-frequency digital ultrasound (VHFDU) appears to be the most optimal method for ensuring measurement accuracy. Intraoperative measurement of residual stromal bed thickness using handheld ultrasound may cause potential measurement errors, including inter-user variability, alteration of stromal bed hydration, applicative force, and variation of perpendicular orientation to the cornea. Single site analyses during intraoperative ultrasonic computation may neglect potential areas of even greater attenuation than that which is measured (ie, irregular flap creation).

Comparative analysis between these instruments demonstrated higher residual stromal bed thickness values reported by handheld ultrasound and OCT in contrast to VHFDU measurements. Each instrument’s tendency for measurement bias should be considered when obtaining evaluative parameters for corneal thickness and residual stromal bed thickness. If there is any doubt about residual stromal bed thickness, surface ablation is a safer alternative for enhancement. Santhiago et al. provided a new metric, called percent cornea tissue altered, for the detection of ectasia risk that can be used for LASIK enhancement. Compared to residual stromal bed, percent cornea tissue altered may provide a more sensitive and individualized measurement for eyes undergoing LASIK enhancement. It has been observed that percent cornea tissue altered exceeding 40% increases the risk of ectasia in enhancement patients.
**EPITHELIAL THICKNESS**

Evaluation of epithelial thickness might be useful for the better evaluation of patients interested in enhancement. Epithelial hyperplasia has been postulated as a cause of regression, as mentioned above. Enhancements should be modified in patients who experience epithelial hyperplasia, especially when receiving surface ablation. Two patients receiving surface ablation enhancements who have the same amount of postoperative regression, one with an epithelial thickness of 50 µm and the other with a thickness of 75 µm, need different degrees of ablation.

The determination of epithelial thickness after LASIK has been performed using different imaging modalities. VHFDU has been used to document increases in postoperative central and peripheral epithelial thickness. Individual measurements for epithelium, stroma, LASIK flap, and residual stromal bed can be obtained with good repeatability. OCT has also been used to follow changes in epithelial thickness. However, direct comparisons have not been performed to determine which modality is superior. After reviewing the literature carefully, we found no guidelines on epithelial changes for patients needing enhancement. Such information can be helpful in creating nomograms for patients undergoing enhancement, thereby making the results of enhancements more predictable.

**KERATECTASIA**

Keratectasia may masquerade as regression and must be ruled out prior to enhancement. Development occurs days to years after the performance of primary LASIK with a mean time of 13 months. Structural changes can be identified in these corneas, including disorganized stromal lamellae and degeneration of collagen fibrils. Although multiple enhancements can also independently increase the potential for keratectasia, the presence of unidentified preoperative risk factors including forme fruste keratoconus, pellucid marginal degeneration, thin cornea, and steep cornea have been strongly correlated with the development of postoperative ectasia. Topography, OCT, and Scheimpflug imaging have been used in an attempt to identify specific parameters that will assist in the improved delineation of these overlapping patient populations. After primary LASIK, there have been observations of both significant and non-significant posterior corneal elevation. It has previously been interpreted that a posterior corneal elevation of greater than 40 µm should represent a basis for preclusion of enhancement procedure performance. However, differences in imaging modality results remain the principal obstacle in confirming a standard threshold.

Studies in which significant changes have been noted have used slit-scanning tomography-based imaging, whereas investigations that have not observed significant changes have used Scheimpflug imaging. Due to the current absence of an absolute reference standard regarding corneal tomographic imaging, it is unclear whether slit-scanning tomography-based imaging may overestimate posterior corneal measurement or whether Scheimpflug imaging may underestimate the true value of this parameter. Attempting to correlate the degree of posterior corneal elevation with the risk of postoperative ectasia in pre-enhancement candidates does not seem to be a beneficial strategy at this point. Also, patients with ectasia can present with abnormal anterior and normal posterior corneal findings. Consequently, we recommend performing both anterior and posterior surface imaging to identify ectasia during the preoperative evaluation preceding enhancement. Stability in both parameters is a sign that ectasia does not exist and regression is more likely present. Patients with changes in posterior curvature should be observed at regular intervals with the assumption that ectasia rather than regression is the most likely etiology.

**ENHANCEMENT MODALITIES**

Numerous procedural techniques exist for the performance of enhancement. These include flap re-lift, surface ablation, re-cut, side cut, mini-flap flap posterior surface ablation, conductive keratoplasty, and arcuate keratotomy. Appropriate selection is contingent on the accurate determination of several factors, including the postoperative time period, previous intraoperative complications, and preoperative screening parameters (Figures A-C, available in the online version of this article). Customarily, it is recommended that enhancement procedures be performed no earlier than 3 months following primary LASIK, due to the preponderant proportion of refractive alterations that occurs within that time period.

**COMMON SURGICAL TECHNIQUES FOR ENHANCEMENT**

**Flap Re-lift.** LASIK flap re-lift is the most commonly performed enhancement method. This technique has demonstrated a high degree of efficacy, predictability, and safety for the correction of residual refractive error. Re-lifting has been conducted using several approaches. In the ‘flaporhexis’ technique, a lens-manipulating hook is used to open the flap edge, which is then grasped by forceps and retracted. An alternative method of initial flap-edge delineation using a needle rather than a hook has been described. The flaporhexis method is rapid, is less likely to cause epithelial inoculation than other procedures because of its minimal use of instruments,
and causes smooth tearing of the epithelium overlying the flap edge\textsuperscript{40}. After lifting the flap, the ablation must be performed in the appropriate manner. Re-treatment ablation diameters should be 5 mm or more and preferably the same size as the initial ablation to reduce the incidence of glare and decreased contrast sensitivity. Additionally, wavefront-guided ablations have been advocated for the correction of higher order aberrations and undesired sequelae following primary LASIK\textsuperscript{41,42}.

Certain circumstances may preclude the performance of flap re-lifting, such as intraoperative complications during primary LASIK (ie, incomplete/irregular flaps, flap edge necrosis, or buttonholes) \textbf{(Figures 1-2)}, previous incisional surgery (astigmatic/radial keratotomy), inadequate flap-diameter (for cases of hyperopic enhancement), stromal scar, and existing interface haze\textsuperscript{7}. Time is also a concern and re-lifting becomes more difficult the longer the flap remains in place. Microkeratome-created flaps have, on average, a longer phase of availability for flap re-lifting than femtosecond laser-assisted flaps. Successful reported effectuation of this procedure has been documented more than 10 years after primary LASIK\textsuperscript{43}. There have been reports in which even microkeratome-created flaps have been resistant to re-lifting following a period of approximately 2 to 3 years after primary LASIK\textsuperscript{18}.

Difficulty in re-lifting the flap increases the probability of the inadvertent intraoperative creation of epithelial defects, which can increase susceptibility for development of epithelial ingrowth, diffuse lamellar keratitis, infection, and flap melts\textsuperscript{18}. It is therefore recommended that enhancement procedures be performed within a period of 6 to 8 months following primary LASIK in patients who possess femtosecond laser-created flaps\textsuperscript{43}. It may also be prudent to attempt flap re-lifting enhancements no later than 3 years after primary LASIK for microkeratome-created flaps to decrease the likelihood of the aforementioned complications.

Despite adequate patient selection, surgical technique, and postoperative timing, epithelial ingrowth remains the most common associated complication of flap re-lifting, with a reported incidence as high as 31\% \textbf{(Figure 3)}. It has been hypothesized that epithelial ingrowth results from stromal inoculation that can occur during the process of flap re-lifting or as a result of peripheral

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{image1.png}
\caption{LASIK flap edge necrosis may occur after primary LASIK or enhancement procedures. It occurs in conjunction with epithelial ingrowth and is a contraindication to a flap re-lift.}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{image2.png}
\caption{LASIK flap buttonholes are areas in the central cornea where the flap is not complete. A central island tissue with epithelium intact is often present when the flap is lifted. They occur more often in patients with high keratometric values and are a contraindication to flap re-lift procedures.}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{image3.png}
\caption{Epithelial ingrowth of the LASIK flap interface. Flap edge necrosis is seen inferiorly. Ingrowth may occur after primary LASIK, but is more common following enhancements.}
\end{figure}
epithelial cell invasion through non-adherent portions of the flap periphery. Inadequate peripheral flap adhesion represents the most probable current hypothesis, in which the implantation of epithelial cells failed to instigate epithelial ingrowth. Documented risk factors for the development of epithelial ingrowth include induction of intraoperative epithelial defects, epithelial basement membrane dystrophy, flap instability, hyperopic LASIK treatment, surgical technique, recurrent corneal erosions, and use of a bandage contact lens.

Microkeratome-created flaps have been associated with a higher incidence of epithelial ingrowth following primary LASIK and successive enhancement procedures. In accordance with the peripheral-invasion hypothesis, the gradual attenuation of flap edges produced by microkeratomes is believed to allow easier access for the migration of epithelial cells into the interface, compared to the more vertical configuration of a femtosecond laser-created flap edge. A recent study by Caster et al. analyzing rates of epithelial ingrowth after re-lifting between femtosecond laser-created flaps and microkeratome-created flaps found that the propensity for the development of epithelial ingrowth increased substantially after 3 years following the primary procedure.

Epithelial ingrowth can result in significant visual impairment as a result of visual axis intrusion or induction of astigmatic error, in addition to flap melt and/or necrosis. Epithelial ingrowth can be effectively managed by flap reflection with debridement of epithelial cells from both the posterior-flap surface and stromal bed, without application of accompanying sutures during flap replacement. Recurrence of epithelial ingrowth after primary debridement merits supplementation of operative measures during secondary treatment, such as YAG laser administration, flap sutures, or use of fibrin glue (Figure 4). Despite the problems associated with re-lifting the flap, good uncorrected distance visual acuities (UDVA) have been obtained.

Surface Ablation. Surface ablation possesses a high degree of applicative value within the expanding assembly of enhancement techniques. Early dissuasion of post-LASIK photorefractive keratectomy (PRK) for the correction of refractive error was initially advanced due to several observed cases of severe postoperative haze. Successive investigations supplementing intraoperative mitomycin C (MMC) during PRK (within previously non-ablated eyes) reported favorable results with a lower incidence of post-procedural haze and improved visual outcomes. PRK has since been used for the enhancement of postoperative astigmatism, myopia, and hyperopia, with generally favorable results. It is particularly useful in patients with thin residual stromal bed thicknesses.

Traditionally, epithelial debridement prior to laser application was performed using ethanol. The 7- to 9-mm alcohol well for the removal of epithelium has enabled us to have a faster resolution of epithelial defect. There has been a trend for the substitution of ethanol-based epithelial debridement for phototherapeutic keratectomy (PTK) and the removal of 42 to 50 µm of epithelium in association with intraoperative MMC during enhancement procedures. Despite this, the use of ethanol in conjunction with intraoperative MMC has also demonstrated a high degree of safety and efficacy for post-LASIK enhancement. The intraoperative administration of MMC appears to constitute a safe treatment option for haze prophylaxis, without adversely affecting endothelial cell morphology or density. However, there are accounts of statistically significant reductions in endothelial cell counts after MMC application. Patients with low endothelial cell counts are not good refractive surgery candidates to begin with and should be expressly avoided if MMC use is necessary.

The elapsed time period following the primary LASIK procedure has been proposed as an important factor influencing the probability of postoperative haze and resultant visual decline. Recovery of corneal nerve function after primary LASIK has been suggested as a principal determinant for modulating the manifestation of corneal scarring; in accordance, the authors of this study proposed a minimum recovery period of at least 2 years prior to the performance of PRK for enhancement. Conversely, earlier performance of PRK has been advocated only for the management of acute flap complications (eg, doughnut flap) following the original LASIK surgery for avoiding corneal scarring.

The duration of intraoperative MMC has steadily declined from 2 minutes to a typical length of 30 seconds, which appears to represent an optimal time.
interval for haze prophylaxis. An instance of slight deviation from this time span (15 seconds) described by Liu and Manche\textsuperscript{61} resulted in recalcitrant postoperative haze that was unresponsive to corticosteroid therapy.

PRK in association with PTK has been employed for the successful treatment of flap complications resulting from primary LASIK. Wavefront-guided PRK has also demonstrated excellent results for reduction of higher order aberrations in eyes previously treated with LASIK.\textsuperscript{62} Several critical adaptations must be enacted when performing PRK in synchrony with PTK and MMC; PTK pulses should be evenly distributed to reduce the occurrence of unintended hyperopic outcomes. It has been approximated that 48 discharged pulses will result in 12 µm of stromal ablation in addition to 1.00 D of hyperopic change during PTK. In addition, effects resulting from MMC administration will attenuate the postoperative reparative process, conceivably resulting in further post-procedural hyperopic variation.

The advent of topography-guided photoablation has promising results. Successful outcomes have been reported after topography-guided enhancements after unsuccessful primary LASIK and PRK treatments. In addition, there is now promise for the treatment of irregular astigmatism with topographical guidance and even post-LASIK ectasia when combined with collagen cross-linking.\textsuperscript{63,64}

Laser-assisted sub-epithelial keratectomy (LASEK) is another viable enhancement option for mild degrees of residual myopia (-1.50 to -2.00 D).\textsuperscript{65-67} Hyperopic enhancements and myopic enhancements for ametropia of greater than 2.00 D have been associated with a high incidence of postoperative haze.\textsuperscript{65,66} As a result of this observation, the use of intraoperative MMC was proposed to potentially prevent this complication for attempted corrections greater than -2.00 D.\textsuperscript{66} Variable results have been reported, including good visual outcomes for low power corrections and high degrees of postoperative refractive unpredictability after the application of MMC.\textsuperscript{68}

Additional surface ablation techniques include PTK following manual epithelial debridement for enhancement in patients with post-LASIK epithelial basement membrane degeneration.\textsuperscript{69} Exclusive ablation of the epithelial layer has been evaluated through the use of intraepithelial PRK for the treatment of postoperative epithelial hyperplasia. Statistically significant improvements in visual acuity and refraction were obtained compared to preoperative values. However, 47.6% of treated eyes suffered from residual myopia.

**Other Alternative Techniques**

**Flap Re-cut.** Re-cut designates an additional procedural option for enhancement (Figures 5-6). The new flap is ideally created more posterior to the original, but may end up at a similar depth. However, if re-lift or surface ablation can be done, they should be the procedures of choice.

Indications for re-cut are essentially identical to many of the contraindications that exist for performing flap re-elevation with stromal bed ablation (ie, thin/incomplete flap, previous radial/astigmatic keratotomy, inadequate flap diameter, buttonhole, or vertical gas bubble). Currently, re-cutting a corneal flap can be accomplished using either a microkeratome or femtosecond laser.\textsuperscript{70} Although it is a viable option, microkeratome flap creation has been associated with a higher than average rate of complications. These include inaccurate incision depth, complete flap transection, incomplete flap creation, and formation of intrastromal tissue fragments as a result of unintentional encroachment of the original interface. The creation of intrastromal tissue fragments characterizes a particularly difficult complication that may be irrevocable and result in severe visual dysfunction. In addition, a comparison between flap re-lifting and flap re-cut yielded superior long-term uncorrected visual acuities and refractive error stability for those receiving re-lifts. The frequency of microkeratome use during enhancement procedures appears to have decreased over time due, in part, to the aforementioned complications and advent of the femtosecond laser in producing more precise and reliable outcomes.

**Vertical Side Cut.** A variation of re-cut involves the creation of a vertical side cut within the established flap using a femtosecond laser (Figure 7). This technique has resulted in a lower incidence of postoperative epithelial ingrowth when compared to flap re-lifting, and is believed to represent a safer alternative for performing flap re-cut.\textsuperscript{71,72} Accessibility of appropriate preoperative imaging devices (ie, OCT or ultrasound) is essential for identifying the peripheral interface boundary.\textsuperscript{73} Complications associated with this technique include a potential for side cut overlap of the previous flap edge and creation of displaced tissue fragments resulting from inadequate distance between the new and established side cuts. It has therefore been recommended that a side cut 1 mm smaller in diameter than the established flap and careful confirmation of side cut centration be performed to ensure that uniformity between each edge margin is achieved.\textsuperscript{74} Ideal patients to benefit from this technique include those with old femtosecond laser flaps (> 1 year) where re-lifting may be problematic and those at risk for re-cut complications. A limitation, analogous to that of flap re-lifting, is the inability for expansion of an ablative zone beyond the original flap diameter.
Mini-Flap. The ‘mini-flap’ (Figure 8) represents an additional femtosecond laser-assisted technique. This supplemental flap is created within the anterior portion of the original flap. It is smaller in thickness and diameter in comparison to the original flap and offers an alternative enhancement option for patients who would otherwise...
be ineligible for enhancement due to inadequate residual stromal bed thickness. In the pilot study describing this technique, no problems were encountered during flap creation or flap lifting. However, this technique is limited by the thickness of the original flap. Microkeratome-created flaps are traditionally thicker and can be as thick as 180 µm. A mini-flap of 100 µm can then safely be performed. Femtosecond laser-created flaps are currently between 90 and 120 µm. This precludes the establishment of a secondary flap within the original.

**Arcuate Keratotomy.** Arcuate keratotomy has been successfully used in the past for the correction of post-LASIK topographical abnormalities and postoperative astigmatic error. Arcuate keratotomy functions through incisional flattening and simultaneous reciprocated steepening, resulting in a “coupling effect.” It can be performed manually or with the assistance of the femtosecond laser. This procedure provides a relative degree of safety, in that procedural failure will not typically adversely affect corrected distance visual acuity. Disadvantages of arcuate keratotomy include unpredictable outcomes with higher levels of astigmatism (often requiring re-treatment) and complications such as epithelial ingrowth into the incision.

**Femtosecond Laser-Assisted Intrastromal Astigmatic Keratotomy.** A relatively new procedure for the enhancement of mixed astigmatism has exploited several beneficial attributes afforded by the femtosecond laser to advance the technique of arcuate keratotomy; this procedural iteration has been termed femtosecond laser-assisted intrastromal astigmatic keratotomy. Femtosecond laser-assisted intrastromal astigmatic keratotomy enables the performance of paired intrastromal incisions, which remain unopened postoperatively. Greater precision of incisional placement, faster procedural time, and lack of epithelial trauma represent advantages conferred by this technique.

**Intracorneal Stromal Rings and Collagen Cross-linking.** Although these procedures are not commonly used and are not the primary procedures of choice in enhancement, intracorneal stromal ring implantation has been shown to correct small myopic error and can facilitate refractive stability. The addition of corneal cross-linking to intracorneal stromal ring implantation is a second option for improvement of UDVA and corneal topography in patients with post-LASIK ectasia.

**Miscellaneous Techniques**

Although the literature describes the following surgical techniques as options for enhancement of vision after original LASIK surgery, many clinicians question the safety, predictability, and long-term efficacy of these procedures.

**Flap Undersurface Ablation.** This treatment is controversial, but it is another variation involving flap manipulation and combines re-lifting with concomitant ablation of the posterior stromal surface of the flap. This procedure is particularly appealing to patients who have low residual stromal bed thicknesses and are at increased risk of developing ectasia following retreatment. In addition to preserving the previously ablated region of stromal tissue, flap ablation results in a lesser degree of postoperative posterior corneal shifting when compared to stromal bed ablation.
However, over-ablation of the flap stroma is difficult and unpredictable. If Bowman’s membrane becomes involved, significant flap wrinkling and loss of visual acuity may occur. To prevent this, a minimum flap thickness of greater than 150 μm has been recommended. Appropriate positioning and fixation of the reflected flap and centration difficulty represent procedural challenges. When performing this procedure, the center of the visual axis must first be marked. After reflection of the flap, the ablative zone axis must be rotated 90° to mirror a traditional ablation to the residual stromal bed. The patient must be able to maintain the eye in a stable intraoperative position (if a superior hinge is present) because no tracking capabilities exist with this variation. Replacement of the flap on the stromal bed allows for realignment of the tissue along the proper axis. Observed complications of this procedure include microstriae, interface debris, and epithelial ingrowth. Despite these difficulties, multiple head-to-head comparisons of flap ablation versus stromal bed ablation resulted in similar refractive outcomes.81,82

**Conductive Keratoplasty.** Conductive keratoplasty as an ancillary enhancement technique can be used for myopic overcorrection83 and hyperopic undercorrection.84 However, important issues related to initial overcorrection, delayed onset of refractive stability, regression, and lack of an established nomogram detract from consistent use of this technique.

**CONCLUSION**

The presence of residual refractive error following LASIK is a precarious situation that may occur as a result of overcorrection, undercorrection, or regression. Clinical evaluation of the refractive state and the patient’s subjective status is extremely important. Ocular and systemic diseases with the potential to affect visual acuity require appropriate treatment prior to the establishment of a diagnosis of residual refractive error. If the need for enhancement has been identified, review of the primary LASIK procedure should be performed. Preoperative measurements and the ablation depth are important for establishing accurate residual stromal bed thicknesses. The operative report should be investigated for evidence of complications such as buttonholes or incomplete flaps. Additionally, the postoperative course must be reviewed in case of postoperative haze, epithelial ingrowth, or other significant issues. Supplemental imaging via VHFDU and OCT may also help confirm flap and stromal bed thicknesses. Once the data have been collected, the proper enhancement procedure can be selected. Patients with thick residual stromal beds and recent primary LASIK treatments are good candidates for re-lifts. Patients with thin stromal beds and LASIK treatments several years prior may benefit to a greater degree from PRK surface ablation. Even patients who suffer from post-LASIK ectasia have treatment options to improve visual acuity.

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Figure A. Flowchart describing the flap re-lift modality for enhancement. D = diopters
Figure B. Flowchart describing the surface ablation modality for enhancement using microkeratome-assisted original flaps. FS = femtosecond laser; RSB = residual stromal bed; PRK = photorefractive keratectomy

Figure C. Flowchart describing the surface ablation modality for enhancement using femtosecond laser-assisted original flaps. PRK = photorefractive keratectomy; RSB = residual stromal bed