cated penetrating keratoplasty have an average annual endothelial cell loss rate of 17.3% for the first 3 years postoperatively, 7.8% from 3 to 5 years, and 2.5% from 5 years or longer compared with normal eyes, which have an average annual endothelial cell loss rate estimated at 0.3% to 1% per year. We speculate that this large amount of primary endothelial cell loss is the reason this complication has thus far only been seen in eyes following penetrating keratoplasty. Many of these eyes that have undergone corneal transplantation do not show the typical guttata that would alert the surgeon to the possibility of endothelial cell loss, so specular microscopy is especially valuable in detecting this complication. It seems likely that more of these and similar cases will be observed after LASIK, not only in patients who have had keratoplasty, but possibly also in patients who have corneal endothelial dystrophies or who experience future surgical trauma. Additionally, the length of time during which the lamellar interface is vulnerable to fluid collection is unclear.

Daniel G. Dawson, MD
Madison, Wis
David R. Harden, MD
Minneapolis, Minn
Daniel M. Albert, MD, MS
Madison

Histopathological Analysis of the Cornea After Laser In Situ Keratomileusis

We report a case of an intact laser in situ keratomileusis (LASIK) flap despite traumatic rupture of the eye 13 months postoperatively. An eye that had undergone a successful LASIK procedure was ruptured in an airplane crash 13 months postoperatively. The cornea developed blood staining. A keratoprosthesis was placed so the posterior pole of the eye could be evaluated and then was replaced with a donor corneal graft. The recipient cornea, which included the LASIK flap, was examined by histologic analysis. Despite major ocular trauma 13 months after LASIK, the flap was intact. Keratocytes were diminished in the flap. Blood staining was minimal in the flap but pronounced in the underlying corneal stroma.
A 59-year-old
Report of a Case. A 59-year-old flight instructor, who had undergone bilateral LASIK procedures 12 and 13 months earlier, crashed his single-engine airplane and hit the instrument panel. He suffered deep facial lacerations, including almost complete avulsion of his nose and severe bilateral eye trauma. Examination and limited exploration of his eyes at the time of surgery for his facial wounds showed a visual acuity of no light perception OD and light perception OS, an intraocular pressure of 6 mm Hg OU, a clear cornea and anterior chamber in both eyes, a dislocated lens in the right eye, and blood in the vitreous, retina, and choroid of both eyes.

Two weeks later, exploratory ocular surgery was performed. The right cornea was brown (blood-stained), so a vitrectomy could not be performed. Blood was irrigated from the anterior chamber and was released from the subchoroidal space through a pars plana stab incision. In the left eye, vitreous blood was removed by vitrectomy, and subchoroidal blood was removed by sclerostomy.

Two months later, the patient was referred to the retina service at the University of California, San Francisco, for further evaluation and treatment. Visual acuity was light perception OD and counting fingers OS. Exploration of the right eye showed an occult scleral rupture. An encircling band was placed. A keratoprosthesis was placed so the posterior pole could be examined. The detached and incarcerated retina, associated with proliferative vitreoretinopathy and retinal and choroidal blood, was inoperable. The keratoprosthesis was replaced with a donor cornea.

The cornea that had been removed to place the keratoprosthesis included the LASIK flap. It was placed in formalin and processed for histologic sections, which were stained with hematoxylin-eosin, periodic acid–Schiff, Prussian blue, Masson trichrome, and Bodian stains.

**Histopathological Analysis.** The flap from the LASIK surgery was in place. In the periphery, the superficial and deeper stroma showed no artifactitious separation and no difference in histologic appearance, suggesting a firm attachment between the two. In the center of the cornea, artifactitious separation of the flap from the underlying stroma was apparent, and the histologic appearance of the flap differed from that of the underlying layers. This was consistent with an absence of healing or scarring centrally. Keratocytes were diminished in the flap (Figure 1). Blood staining, very subtle with the hematoxylin-eosin stain, was vividly demonstrated with the Masson trichrome stain (Figure 2). The hemoglobin particles were mostly confined to the stroma below the flap and appeared to pile up at the flap interface. The Prussian blue stain showed a paracentral epithelial iron line but no iron staining in the corneal stroma. The Bodian stain showed a paucity of nerve fibers traversing the Bowman layer.

**Comment.** Evidence from human studies with confocal microscopy and from experimental studies of wound healing after LASIK in rabbits has shown that healing after this procedure is usually limited to the peripheral edge of the flap. The healing sequence in the experimental rabbits is as follows: a localized and limited epithelial plug develops, which interacts with the adjacent corneal stroma and may release cytokines that produce both keratocyte activation and apoptosis. Extracellular matrix proteins such as fibronectin and tenascin are limited to the edge of the flap and lead to local wound healing.

Anderson et al examined the histologic and ultrastructural features of human corneas after successful LASIK in 2 patients postmortem. As in our case 13 months postoperatively, at 20 months postoperatively their case showed occasional areas of artifactitious separation between the flap and the underlying interface centrally with virtually no active wound healing except at the flap edge. Of the 6 previously reported clinicopathological studies of human corneas after LASIK, only the 2 cases in the study by Anderson et al were from uncomplicated procedures. This is the third such case.

Traumatic flap dislocations are rare, but they have been reported after basketball, snowball, dog paw, finger, and airbag injuries as long as 38 months after successful surgery.

The limited peripheral wound healing in most cases, including this case, that received major trauma (eg, ruptured globe, dislocated lens, intraocular hemorrhage, and retinal detachment) is sufficient to secure the flap.

In our case, there were fewer keratocytes in the flap 13 months af-
Figure 2. Central cornea of a patient with traumatic rupture of the eye 13 months after laser in situ keratomileusis. Note the granular staining of hemoglobin particles below the flap and the piling up of these particles at the interface (arrow), with few particles in the overlying flap (Masson trichrome, original magnification ×10).

The vivid staining of the hemoglobin particles at the interface (arrow), with few particles in the overlying flap (Masson trichrome, original magnification ×10).

Choroidal Neovascular Membranes Treated With Photodynamic Therapy

The Treatment of Age-Related Macular Degeneration With Photodynamic Therapy (TAP) Study Group reports 1 and 2 demonstrated a statistically significant reduction in moderate and severe vision loss in patients with predominantly classic subfoveal choroidal neovascular membranes (CNVMs) secondary to age-related macular degeneration (ARMD) treated with intravenous verteporfin (Visudyne, CIBA Vision Corp, Duluth, Ga) and photodynamic therapy (PDT). Patients were followed up every 3 months after PDT treatment with fluorescein angiography. If leakage was identified from the CNVM, patients underwent repeated treatment with verteporfin PDT.

Verteporfin PDT treatment was also associated with fewer eyes experiencing progression of classic CNVM beyond the area of the le-