Trigeminal nerve lesions at differing levels can result in complete or partial corneal anesthesia and ensuing epithelial breakdown. Disease progression can lead to corneal ulceration, melt, and perforation. Herein, we report a series of 5 cases of patients without diabetes who developed neuropathic corneal ulceration presumed secondary to long ciliary nerve compromise. This occurred within 5 to 10 weeks following vitrectomy surgery with endolaser and silicone oil tamponade for retinal detachment.

**Case 1**
A man in his 60s with a recurrent rhegmatogenous retinal detachment and proliferative vitreoretinopathy (PVR) underwent a pars plana vitrectomy with 180° retinectomy; confluent endolaser, including both horizontal meridia; and silicone oil to reattach the retina. Six weeks later, he developed a central epithelial defect with a secondary infective crystalline keratopathy. He was treated with an intensive topical regimen using a fluoroquinolone. Corneal anesthesia was documented 4 weeks later and a differential diagnosis of herpetic keratitis considered, with additional topical and systemic antiviral therapy instituted. A botulinum toxin–induced ptosis aided resolution at 6 months but with 20% central corneal thinning and marked stromal scarring.

**Case 2**
A woman in her 70s required augmented barrier endolaser at oil removal 4 months post–rhegmatogenous retinal detachment repair with 180° retinectomy. Ten weeks later, she developed a neurotrophic corneal ulcer. Resolution was achieved after 3 weeks of treatment with prophylactic topical antibiotics and intensive topical ocular lubricants, with resultant mild stromal thinning and residual scarring in the visual axis.

**Case 3**
A woman in her 50s with recurrent PVR rhegmatogenous retinal detachment required a 220° retinectomy, endolaser, and oil to reattach the retina. A painless paracentral corneal epithelial defect (Figure 1) and fixed dilated pupil were noted 5 weeks postoperatively. Complete resolution was achieved after 3 weeks with combined topical antibiotics and intensive topical ocular lubricants.
Case 4
A woman in her 50s required a vitrectomy, PVR membrane peel, and 270° retinectomy with oil following 3 previous failed retinal detachment repairs over a period of 1 month. She presented with a painless reduction in vision 10 weeks later and was diagnosed as having a neurotrophic ulcer. Complete resolution was achieved after 4 weeks of treatment with prophylactic topical antibiotics, intensive lubricants, and a switch to an unpreserved topical steroid.

Case 5
A man in his 50s with dense strabismic amblyopia required a vitrectomy and 210° retinectomy with oil for a PVR retinal detachment in his better eye. He underwent pars plana vitrectomy, oil removal, epiretinal membrane peel, and augmented barrier laser with sulphahexaflouride 10 weeks later. One month postoperatively, he developed a contralateral cutaneous herpes zoster infection without ocular involvement, which resolved with systemic antivirals. Four weeks later (8 weeks after oil removal and augmented barrier laser), he presented to his local ophthalmic unit with a painless reduction in vision in his operated-on eye and was diagnosed as having a neurotrophic ulcer (Figure 2). Systemic antivirals were recommenced, in addition to topical antibiotics and infrequent lubricants. One week later, on review at our institution, systemic therapy was discontinued and resolution was achieved at 3 weeks with intensive lubricants, with residual central stromal scarring and 30% thinning.

Case Findings and Treatment
In these cases, there was no history of diabetes mellitus and no cases had undergone previous corneal surgery or intraoperative epithelial debridement. The preceding episode of contralateral cutaneous herpes zoster ophthalmicus in case 5 was deemed to be coincidental; although bilateral corneal nerve alteration has recently been described in unilateral herpes zoster ophthalmicus, contralateral corneal ulceration has not. Furthermore, resolution was achieved following discontinuation of antiviral therapy. There was no history of herpetic eye disease in the other 4 cases.

Discussion
Corneal sensation is derived from the ophthalmic branch of the trigeminal nerve, predominantly via the long ciliary nerves. Neurotrophic keratopathy following transcleral cyclodiode laser is well known and treatment in the regions of the long ciliary

Figure 1. Case 3
The arrow highlights the paracentral corneal epithelial defect at presentation.

Figure 2. Case 5
The arrow highlights the paracentral corneal epithelial defect 1 week after presentation.

Figure 3. Corresponding Fundus Image of Case 3
The arrows indicate extensive confluent chorioretinal scarring at the 3- and 9-o’clock positions.

All 5 eyes were treated with Argon (532-nm) endolaser, with standard power and duration and frequency settings (200-250 mW, 0.2 second, and 0.2 second, respectively). Fundus examination findings showed marked confluent chorioretinal scarring at the 3- and 9-o’clock positions in each case (Figures 3 and 4). We concluded that confluent intraoperative endolaser at these sites compromised long ciliary nerve function, with resultant corneal anesthesia and ulceration. Concurrent short ciliary nerve damage may have occurred in case 3, who also had mydriasis. No other clinical signs suggested a lesion elsewhere in the trigeminal nerve nor a polyneuropathy.
nerves is avoided. Retinal surgeons are not afforded the same luxury as treatment-immune sites because confluent retinopexy may be required for sustained retinal reattachment. Retinal laser–induced internal ophthalmoplegia has been reported in diabetic patients following diode photocoagulation and corneal sensitivity reduction after argon retinal laser; this is thought to be due to short and long ciliary nerve damage, respectively. Reduction in corneal sensitivity following retinal detachment surgery with sectoral scleral buckles and encircling bands has been reported. Internal ophthalmoplegia and neurotrophic ulceration has recently been reported in an premature child following diode laser and an encircling buckle; this was again presumed secondary to ciliary nerve damage.

The presence of silicone oil in all 5 eyes may have contributed to the observed corneal changes. However, the temporal relationship between the onset of the keratopathy and the operative intervention favors a peroperative event (ie, endolaser) over oil toxicity as the cause. In our series, corneal ulceration occurred between 5 and 10 weeks postintervention. A more favorable outcome was achieved where the diagnosis was made early and appropriate treatment commenced (cases 2, 3, and 4). Where alternative underlying causes were initially entertained (cases 1 and 5), the outcome appears to have been less favorable.

To our knowledge, this is the first reported series of cases where neurotrophic corneal ulceration has occurred secondary to endolaser in adults. Clinicians should be mindful of the long ciliary nerves intraoperatively and, where possible, avoid heavy confluent treatment at these sites, without compromising adequate retinopexy. Where corneal anesthesia occurs, it is important to recognize this early and treat promptly to minimize the risk for ulceration and visual loss.

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REFERENCES

The arrows indicate extensive confluent chorioretinal scarring at the 3- and 9-o’clock positions.

Figure 4. Corresponding Fundus Image of Case 5

The arrows indicate extensive confluent chorioretinal scarring at the 3- and 9-o’clock positions.