Expulsive Suprachoroidal
Hemorrhage With Scleral
Buckling Surgery

Intraocular hemorrhage is one of the most serious complications that can occur during a retinal reattachment procedure. We describe a patient who developed a massive suprachoroidal hemorrhage during a scleral buckling procedure with extrusion of intracocular contents through a penetrating keratoplasty incision.

Report of a Case.—A 62-year-old healthy white woman presented with a rheumatogenous retinal detachment in her left eye on April 26, 1989. She had undergone a combined cataract extraction with lens implant and penetrating keratoplasty in January 1988. Her corneal sutures were removed in November 1988. A neodymium-YAG laser posterior capsulotomy was performed in March 1989. During retinal surgery, cryotherapy under direct visualization was applied to an area of anterior circumferential lattice in the 2 o'clock position and an adjacent retinal tear. The vortex ampulla and veins were not treated.

Intracocular sutures were placed in each quadrant without premature drainage. The buckle and band were put into position. Prior to attempting drainage, the eye became hard. There was a forced 270° rupture of the corneal wound, with extrusion of the posterior chamber lens and intraocular contents. Large hemorrhagic choroidal detachments were noted. Despite sclerotomies in three quadrants draining hemorrhagic fluid, the pressure remained elevated. With further drainage and eventual excision of extruding tissue, the eye softened, allowing the cornea to be reattached. As the hemorrhagic choroidal detachments resolved, the retina was noted to be totally detached, with proliferative vitreoretinopathy. The eye had become hypotonic with light perception visual acuity.

Comment.—The development of a subretinal or choroidal hemorrhage during scleral buckling surgery becomes significant when the hemorrhage gravitates under the macular region. The hemorrhages may occur after perforation or rupture of a choroidal vessel or from precipitous and excessive hypotony that can occur with the release of subretinal fluid or global rupture. Thermal treatments of the retina, including diathermy and cryopexy, can also cause choroidal hemorrhages. The one case report in the literature that we are aware of describing the development of an expulsive choroidal hemorrhage during scleral buckling surgery occurred after the drainage portion of the procedure.

In our patient, although there may have been some transient elevation of intraocular pressure during the cryopexy, there was no subsequent reduction in pressure since drainage was not performed. The vortex system was not traumatized nor inadvertently treated with cryotherapy. Other risk factors associated with expulsive hemorrhage, including hypertension, glaucoma, myopia, and local anesthesia, were not present in our patient. Although hemorrhage was not noted during the cryopexy, it is possible that it began shortly afterward and progressed. Another possible cause of the hemorrhage included the mechanical disruption of a choroidal vessel that can occur as the globe is deformed, either with a scleral depressor or cryoprobe or from the placement of a scleral buckle. Further complicating the development of the choroidal hemorrhage in our patient was the presence of a corneal graft with no corneal sutures. The tensile strength of the cornea had been noted to be reduced after penetrating keratoplasty. The manipulation of the globe during a scleral buckling procedure may stress the corneal wound, facilitating a rupture. In our patient, although a choroidal hemorrhage developed prior to the wound rupture, the further reduction in pressure may have resulted in further and more massive hemorrhage. It is prudent for the retinal surgeon to consider placement of corneal sutures in an effort to reduce the problem of all dehiscences, especially if the patient has had suture removal in the relatively recent past.

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Herpes Reactivation and Ophthalmoplegia From Pituitary Adenoma Invading the Cavernous Sinus

Ocular motor nerve palsies are common in patients with herpesvirus reactivation. The palsies are caused by an ischemic vasculitis affecting the ocular motor nerves along with the trigeminal nerve. On rare occasions, a tumor in the cavernous sinus can produce ophthalmoplegia and skin manifestations of herpesvirus infection.

Report of a Case.—An 82-year-old man was found by a family member to be mildly confused and complaining of right orbital pain. On examination he had a fever of 39.8°C and herpetic vesicles on the right side of the nose and upper lip. Skin testing disclosed mild hypoaesthesia involving the right ophthalmic (V1) and maxillary (V2) trigeminal divisions. Visual acuity was 20/20 OU. The patient was unable to open his right eye (Fig 1). It was completely ophthalmoplegic with a fixed 6.5-mm pupil.

The patient's ophthalmoplegia was initially attributed to herpetic virus. However, a computed tomographic scan performed 2 days later revealed a sellar mass invading the right cavernous sinus (Fig 2). The lesion appeared nonhomogeneous and slightly denser than surrounding brain tissue. The patient was treated with steroids and acyclovir sodium before undergoing transphenoidal resection of a hemorrhagic pituitary adenoma.

Comment.—Outbreak of herpes simplex or herpetic zoster can be triggered by trigeminal rhizotomy or manipu-
Ciliary and cornea peripheral Fig 1.—Right eye. A semilunar opacification in the inferotemporal peripheral cornea continuous with a partial immune ring is shown (arrow). A translucent zone is noted between the opacity and the limbus. Ciliary and conjunctival injection is also noted.

Fig 2.—Left eye. Similar corneal opacity continuous with a complete corneal immune ring (arrows) is shown. Other findings are similar to the right eye.

Fig 2.—Axial computed tomographic scan showing a lesion in the right cavernous sinus. The irregular bright signal within the mass was blood.

ulation. The skin lesions usually erupt within a few days of the surgical procedure. In our patient, we suspect that pituitary apoplexy caused simultaneous herpesvirus reactivation and ophthalmoplegia by compression of cranial nerves III, IV, V, VI, and V1 in the cavernous sinus.

Herpesvirus reactivation usually occurs spontaneously. Paralysis of extraocular muscles is due to extension of inflammation from the trigeminal nerve to ocular motor nerves within the cavernous sinus. This case underscores that before ophthalmoplegia is ascribed to herpesvirus, the possibility of a mass within the cavernous sinus producing both ophthalmoplegia and herpes reactivation must be considered.

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**Bilateral Corneal Immune Ring Opacity in Behcet's Syndrome**

Ring-shaped infiltration of the corneal stroma, known as Wessely ring, is believed to result from intrastromal interaction of antigens, from either an infecting organism or altered corneal tissue, with antibodies diffusing into the cornea from the limbus. These complexes activate, complement, and subsequently attract inflammatory cells. We report a rare case of Behcet's disease in which both antibodies and antigens probably originated from the limbal vasculature. They diffused either separately or as an immune complex and precipitated in the stromal midperiphery, where an optimal concentration was achieved.

**Report of a Case.—**A 16-year-old girl known to have Behcet's syndrome for 7 years was referred to our department. Her ophthalmic history consisted of recurrent nonpurulent conjunctivitis. Her visual acuity was 20/20 and the intraocular pressure was normal bilaterally. In the inferotemporal corneal periphery a semilunar stromal opacity was noted 1.5 mm from the limbus continuous with a stromal immune ring (Figs 1 and 2). The rest of the anterior and posterior segments were normal. Systemic and local steroid therapy did not cause resorption of the immune ring.

**Comment.—**Behcet's syndrome is a chronic, recurrent, inflammatory disease with widespread clinical manifestations, characterized by recurrent orogenital aphthous lesions, uveitis, and hypopyon. The etiology is unknown but it may be viral or autoimmune. The most common corneal pathologic lesion is band keratopathy, which is a complication of chronic iridocyclitis. Rarely, conjunctivitis varying from catarrhal inflammation to phlyctenular like erosions may be found. Superficial punctate keratitis, recurrent corneal ulcerations, and circumscribed stromal opacities that may become vascularized have also been reported.

In systemic vasculitides the peripheral cornea may be affected either by ischemia or deposition of immune...