

Aqueous outflow and glaucoma surgery

How does non-penetrating glaucoma surgery work?

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Jiscocanalostomy and deep sclerectomy are operations for glaucoma which have been designed to avoid some of the complications of conventional glaucoma surgery. Both procedures involve creating a partial thickness scleral flap and then removing a second layer of sclera deep to the initial flap. This unroofs Schlemm's canal and exposes Descemet's membrane. The resulting trabeculo-Descement's membrane is said to act as a semi-permeable layer of tissue, allowing aqueous to percolate through it. In viscocanalostomy, after removal of the deep scleral layer, Schlemm's canal is then cannulated and expanded with viscoelastic. Viscoelastic is also injected into the region of excised sclera, or "scleral lake", to prevent healing. Variations of these proce-

dures include removing the inner wall of Schlemm's canal and adjacent meshwork, but leaving the inner meshwork intact, or placement of a collagen implant in the filtration bed to prevent episcleral fibrosis.

Do these operations relieve the specific pathological problem of primary open angle glaucoma (POAG)? Or do they function as simply another way to make a hole in the eye?



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Aqueous outflow in normal an glaucomatous eyes

The increased IOP found in glaucoma is caused by an increase in aqueous outflow resistance within the drainage pathways, and not excess secretion of aqueous humor. Outflow resistance in the normal eye probably resides in the trabecular meshwork near Schlemm's canal or within the endothelial lining of the canal wall. In POAG the abnormal increase in outflow resistance is probably also in the trabecular meshwork near Schlemm's canal, termed the juxtacanalicular tissue (JCT) or cribriform layer, although other ideas have been proposed.

Non-penetrating surgery and aqueous outflow

Unroofing Schlemm's canal

Removal of the outer wall can cause damage to the inner wall of the canal. The septae, which bridge the inner and outer walls, can easily damage the inner wall when they are pulled away during the unroofing procedure. Such damage to the inner wall and adjacent JCT region effectively removes these regions and allows aqueous access to the canal. In addition, aqueous enters Schlemm's canal through both the inner and outer walls of the canal, although most aqueous probably enters the canal through the inner wall. Removing the outer wall of the canal and its associated JCT would increase aqueous entry to enter the canal through this route, bypassing the presumed abnormal JCT region of the meshwork in glauco-

Creation of a Descemet window

Descemet's membrane is not permeable enough to relieve the elevated pressure of glaucoma. A window of exposed Descemet's approximately 21 x 21 mm would be required to lower intraocular pressure to the low teens. The relative impermeability of Descemet's

is supported by the 41% goniopuncture rate after deep sclerectomy. If Descemet's membrane is thinned or partially removed during the procedure, however, its permeability would increase. When considered in conjunction with the meshwork, the permeability of the "trabeculo-Descemet's membrane" is probably due to properties of the meshwork remaining after the unroofing of Schlemm's canal, and not the exposing of Descemet's membrane.

Scleral lake

Removal of the inner layer of sclera creates an aqueous space, which in some cases may be the site for placement of a collagen implant. Creation of this scleral lake underneath the outer scleral flap has no theoretic effect on the abnormal outflow resistance found in glaucoma. Ultrasonic measurements of the area of the lake found no relationship to intraocular pressure in a series of human eyes.

Injection of viscoelastic

There is no theoretic basis for relieving elevated intraocular pressure by expanding the lumen of Schlemm's canal. A more likely explanation is that expansion of the canal ruptures both the inner and outer endothelial walls of the canal as demonstrated in the human eye and in the monkey. These ruptures extend into the juxtacanalicular connective tissue, and may also rupture some of the meshwork itself.

Conclusions

Both viscocanalostomy and deep sclerectomy likely function as "gentle" trabeculotomies, allowing aqueous to bypass the site of abnormal outflow resistance, the juxtacanalicular tissue. Aqueous probably enters the canal through inadvertent ruptures in the JCT and inner wall of the canal, and also through the unroofed outer wall. If the ruptured regions of the JCT and canal heal with time, surgery may fail in those eyes that did not develop filtration blebs. This problem is beginning to be seen in the increasing failure rates of deep sclerectomy with time.

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