THE TREATMENT OF BULLOUS KERATITIS BY
GREATER SUPERFICIAL PETROSAL NEURECTOMY*

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While an exposition on bullous keratitis by a neurological surgeon may seem inappropriate, it may better serve our purpose to stress those features that are of greater concern to the neurosurgeon than the ophthalmologist in whose hands the diagnosis primarily rests.

Any discussion of bullous keratitis is necessarily a discussion of corneal edema and the mechanism of its production. As the name implies, bullous keratitis is the term applied to the most severe grade of corneal edema wherein the epithelium is separated from Bowman’s membrane by a collection of fluid. A rather pendulous type of bulla may be formed, usually below the center of the cornea. The bulla is termed pendulous since the subepithelial collection of fluid can be moved about by pressure upon the eyelid.

The onset of the condition may be quite abrupt, occurring after only several hours of local discomfort. However, it may follow after several days of corneal edema. The patient complains of pain, photophobia, lacrimation, and blurring of vision. The eye appears irritated and red. The symptoms become progressively severe until the bullae rupture. Healing is accomplished by such regeneration of the corneal epithelium as might occur after an ordinary corneal abrasion. Bullae are reformed only to break down again. However, the condition may progress to so severe a degree, with such intense pain from the formation and subsequent rupture of the bullae, that only enucleation remains as a means of relieving the patient of his misery.

Before entering into a discussion of corneal edema and the mechanism of its production, it might be well to recall that the transparent cornea consists of five strata from front to back:

1. The corneal epithelium.
2. Anterior elastic lamina (Bowman’s membrane).
3. The corneal stroma.
4. The posterior elastic lamina (Descemet’s membrane).
5. The endothelium of the anterior chamber.

The experimental work of Cogan and Kinsey has resulted in a theory, which is at once reasonable and lucid, concerning the movement of the fluid within the cornea. The fluid is believed to enter the stroma at the limbus and

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to diffuse at all levels toward the center of the cornea. The greatest movement occurs at the corneal periphery, the least at the center. These authors showed that the epithelium and endothelium are impermeable to sodium chloride and function as semipermeable membranes. The corneal stroma, in effect, is sandwiched between two semipermeable membranes—one on its anterior and the other on its posterior surface. The stroma, being permeable to sodium chloride, is unable to function as a semipermeable membrane at all. Since the fluid within the stroma will eventually diffuse to the endothelial or epithelial surfaces, the electrolytes will be retained within the cornea while the water will be lost continuously into the hypertonic fluid over the corneal surface. The evaporation taking place from this surface again raises the local osmotic pressure, enabling the process to continue.

Cogan\(^4,5,6,9\) also showed that the corneal epithelium could not be separated from Bowman's membrane, as in bullous keratitis, by forcing fluid into the cornea from the aqueous. This could be done only by the absorption of a fluid from the outside by a cornea that was hypertonic to the precorneal film. It becomes apparent that if the tears have an inherently lower osmotic tension than the cornea, or if the osmotic tension of the cornea is unduly increased, as by damage to the endothelium, epithelial vesicles or bullae could be formed by the imbition of the excessive fluid. It follows, then, that increasing the tonicity of the tears, or other fluid in contact with the corneal surface, should promptly withdraw the water from the bulla or vesicle. That this occurs is attested to by the use of glycerin drops, contact lens with hypertonic solution, or the air-tight calcium chloride goggles used by Cogan, with good but transient success.

Conversely, as the bullae rupture, a further irritation results in an increased epiphora with a further dilution of the salt content in the precorneal mucous film and the subsequent lowering of tonicity. The lowered osmotic tension results in the formation of more bullae. When the eyes are closed at night and evaporation is prevented, dilution of the precorneal film occurs with the same unfortunate result.

It appeared advisable to direct our attention to the inhibition of lacrimation. Since local extirpation, diathermia, and x-ray treatment of the lacrimal gland had been used unsuccessfully (personal experience-L.V.J.) another approach was considered advisable.

It occurred to one of us (W.A.N.) that denervation of the lacrimal gland could be effectively accomplished by sectioning the greater superficial petrosal nerve. By denervation of the gland one could avoid such failure as might be attributed to accessory lacrimal tissue, after attempted extirpation, or to the incomplete ablation of glandular tissue following other types of therapy.

Originating at the genu of the facial nerve within the facial canal of the petrous bone, the greater superficial petrosal nerve emerges into the middle fossa through the hiatus canalis facialis, i.e. hiatus Fallopii. The nerve lies in a groove as it courses anteromedially and inferiorly across the petrous