Ocular Surface Disease and Keratorefractive Surgery

One surgeon's education and experience.

BY LEE T. NORDAN, MD

In the middle of February, I attended and lectured at the ACES-SEE Meeting at the Atlantis Resort in Nassau, Bahamas. Several of the meeting's presentations related to ocular surface disease and its preoperative treatment with respect to keratorefractive surgery. As the speakers discussed their different strategies for addressing pathology of the eyelid and corneal epithelium, I started to think about my experience with ocular surface disease and keratorefractive surgery.

MY EARLY EDUCATION

My fellowship at the Jules Stein Eye Institute in Los Angeles was dedicated to cornea and external disease. My mentor was Thomas Pettit, MD, an intellectual of the highest caliber who was an astute clinician and talented teacher. The routine was for me initially to examine a patient alone, to discuss the findings with Dr. Pettit, and then jointly to examine the patient so that he could point out the many interesting aspects of the case that I had probably missed.

While we were discussing the first patient I had examined during my fellowship, Dr. Pettit mentioned, "I'm sure you noticed the ovality of the meibomian glands as a result of the subconjuctival fibrosis—or something to that effect." It was clear that Dr. Pettit knew many things that I did not, but I paid attention and learned a fair amount about external disease. During the course of the year, I won perhaps three chocolate ice cream sodas when my diagnosis ended up correct compared with Dr. Pettit's. I easily lost 100 vanilla ice cream sodas to him. It was truly an educational and enjoyable year.

The first thing I learned was that, as a group, external disease is whimsical and difficult to treat. I decided that the Schirmer test was not only useless but often provided misinformation. Many patients presented with a totally normal anterior segment and a Schirmer test of less than 1 mm, and many had severe corneal disease with copious amounts of tears. Some clinicians said there was something about the quality of the tears. The mucin-tear-lipid composition of tears was a great theory, but it did not seem to pan out when I was trying to devise a specific clinical treatment for a given patient. Why did artificial tears give relief for more than 10 minutes, given that they are diluted and blinked away within seconds? Why did many patients prefer one brand of artificial tears over another when both products had the same ingredients? The entire area was, and still is, confusing.

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External disease, however, is an important factor in determining a patient's candidacy for keratorefractive surgery.

MY EXPERIENCE

I performed tens of thousands of keratorefractive surgeries and personally examined every patient pre- and postoperatively. I never had a case in which the corneal epithelium was completely clear preoperatively but became chronically diseased postoperatively or developed a problem with epithelial healing. In other words, if the condition of the lids and the level of tear production pre-

operatively had been compatible with a completely normal cornea, then a steady state for that particular eye had existed for many years. Keratorefractive surgery would not typically be expected to alter this balance.

I did not treat mild degrees of chronic inflammation of the eyelid, which the vast majority of patients experience from time to time. If an asymptomatic lid deformity in an older patient revealed no corresponding corneal defect, then I felt that the lid needed no extra preoperative treatment.

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My intent is not to be a nihilistic clinician with respect to ocular surface disease but rather to discuss what degree and type of lid disease ophthalmologists must and should treat preoperatively and how aggressively. If the surgeon does not provide the patient with a long-term solution to a problem of chronic external disease, then he is essentially providing no treatment.

Of course, I had several cases of punctate keratopathy caused by the postoperative drops, a problem that was sorted out in due course. The complication, however, could not be prevented by preoperative treatment. I never performed Schirmer testing before surgery, and all of my many patients with supposedly dry eyes, including those with rheumatoid arthritis (perhaps *low tear production* is a better term), saw just fine postoperatively as long as their corneal epithelia were pristine preoperatively.

CONCLUSION

Readers will consider the message of this article either the ramblings of an old, forgetful ophthalmologist or the insights of a skilled keratorefractive surgeon. I am in favor of treating important ailments of the eyelid and cornea; they certainly exist. I am not, however, a proponent of the pseudotreatment of minor imperfections of the eyelid and nonexistent "tear pathology" that makes no difference. Admittedly, the two categories can be difficult to differentiate in many cases. I am just reporting my experience. Comments are welcome. ■

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