The past decade has brought a paradigm shift in our understanding of dry eye syndrome. The change was affirmed by a recent statement by the Tear Film and Ocular Surface Society that “it is important to note that meibomian gland dysfunction (MGD) may be the leading cause of dry eye syndrome throughout the world.”1 Dry eye patients have largely been characterized in terms of aqueous deficiency.2 Only recently have eye care specialists begun to recognize and seriously appreciate the importance of MGD in patients with evaporative dry eye symptoms and ocular surface disease (OSD).3 Despite our growing understanding of the role of the meibomian glands, lipid-deficient (and dysfunctional) evaporative dry eye is still a relatively new concept that is only slowly “penetrating the market” of awareness and understanding among patients and eye care providers.4 Interestingly, Bron and Tiffany recently reported that evaporative and aqueous-deficient dry eye should not be considered separate entities. With progression, the two conditions merge and become indistinguishable.5

As busy clinicians, we tend to focus our efforts on areas that we believe will optimize the success of treatment. Historically, therapy for MGD and associated OSD has yielded low returns on our invested time and effort. In these challenging cases, patients present with chronic symptoms and expectations that result in extended chair time. They seldom provide the same level of complimentary feedback offered by surgical patients.

The work of Donald Korb, MD, has dramatically increased my awareness and understanding of the need to diagnose and treat MGD as a crucial component of managing patients with dry eye syndrome. This article is not intended in any way to minimize the devastating impact of keratoconjunctivitis sicca and Sjögren’s syndrome. Rather, I wish to call attention to the profession-wide (both ophthalmic and optometric) underrecognition of the role of MGD in symptomatic evaporative dry eye disease.

THE CONNECTION

The pathophysiologic link between dry eye syndrome and MGD includes hyposecretion of the meibomian glands and the production of abnormal lipids. The dysfunction destabilizes the tear film, making it prone to more rapid evaporation. The compromised ocular surface includes surface epitheliopathy as well as inflammation. Reduced lubrication of the ocular surface leads to mechanical friction between the lid and the cornea, which exacerbates inflammation of the ocular surface and traumatizes the surface microenvironment from lid-wiper epitheliopathy.6 MGD results in ocular surface inflammation in the form of blepharokeratoconjunctivitis.

MISCONCEPTIONS

We lack a complete understanding of the factors that initiate the process that eventually leads to evaporative...
dry eye. Although we can piece together our findings to match the patient’s complaints, we are left with “downstream” treatments. We prescribe anti-inflammatory agents, and we resort to questionable and inconsistently effective cleansing treatments to address any build-up of presumed infectious contributors.

The long-term changes affecting eyelid structure and function are no doubt a consequence of whatever causes glandular physiology to become abnormal. I anticipate that future treatments will attempt to address these earlier fundamental processes. For example, our attention to the role of diet and supplementation is growing, and in some patients, we may have to address certain lifestyle patterns that exacerbate their problems.7,8 Also of importance is that our present approach addresses changes that have taken perhaps decades to develop fully.

**TREATMENT OPTIONS**

Do warm compresses work? Yes and no. Patients with all levels of disease will likely experience some relief, either because of or in spite of this intervention. Patients want to feel better, and following their doctor’s instructions gives them some sense that they are addressing the problem at hand. Unfortunately, even the most diligent patient who follows our instructions to the letter may return with little improvement in symptoms or identifiable progress on examination. Compliance has always been the main problem, however, as only the rare patient will incorporate compresses into his or her daily routine with the same consistency as bathing, shaving, and brushing his or her teeth.

While we emphasize heat and pressure, it is becoming more evident that heat delivered anteriorly is relatively ineffective compared with heat delivered to the posteri-
Figure 3. The Mastrota Meibomian Paddle provides counter-pressure for manual expression performed under topical anesthetic to help express blocked glands in the upper and lower eyelid.

Mechanically Expressing the Gland
While I use the calibrated Meibomian Gland Expresser primarily for the diagnostic monitoring of progress, I have found the Mastrota Meibomian Paddle (Cynacn/Ocusoft, Inc., Richmond, TX) to be extremely effective in expressing a chronically impacted gland.

enced by some patients, especially in low lighting conditions such as driving at night. In order to achieve optimal visual performance with this lens, emmetropia must be targeted. Patients with significant preoperative or expected postoperative astigmatism >1.0D may not achieve optimal visual outcomes. Care should be taken to achieve IOL centration, as lens decentration may result in a patient experiencing visual disturbances under certain lighting conditions. PRECAUTIONS: Do not resterilize. Do not store over 45°C. Use only sterile irrigating solutions such as BSS® or BSS PLUS® Sterile Intracocular Irrigating Solution. Clinical studies with the AcrySof® IQ ReSTOR® IOL indicated that posterior capsule opacification (PCO), when present, developed earlier into clinically significant PCO. Studies have shown that color vision discrimination is not adversely affected in individuals with the AcrySof® Natural IOL and normal color vision. The effect on vision of the AcrySof® Natural IOL in subjects with hereditary color vision defects and acquired color vision defects secondary to ocular disease (eg, glaucoma, diabetic retinopathy, chronic uveitis, and other retinal or optic nerve diseases) has not been studied. The long-term effects of filtering blue light and the clinical efficacy of that filtering on the retina have not been conclusively established. AT- TENTION: Reference the Physician Labeling/Directions for Use for a complete listing of indications, warnings, and precautions.

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Figure 4. The LipiFlow device employs several novel methods to treat the meibomian glands. As I mentioned, the solidified material obstructing the meibomian glands has a higher melting point than normal secretions, and the most efficient method to remove the former is to apply adequate heat to achieve melting and liquefaction. The LipiFlow is designed to address the physical limitations of raising the temperature of the inner surface of the lid where the glands are located. In contrast to externally applied warm compresses, heat is applied directly to the inner surface of both the upper and lower eyelids with a safe and precisely controlled resistive heater. This treating instrument has two primary features. First, a large shell resembling a scleral lens contains both heating and insulating components designed to safely and comfortably vault the cornea and protect the surfaces in proximity to and contact with the device. The instrument is inserted somewhat similarly to a mirrored lens for a retinal examination. The convex surface of the shell contains a proprietary group of heating elements that should safely and consistently deliver heat to elevate the temperature of the palpebral conjunctiva of both the upper and lower lids and consequently the meibomian glands (Figure 5). The advantage of applying the heat from inside the eyelid is that it raises the temperature of the meibomian glands promptly and efficiently, avoiding the 6- to 10-minute delay when the heat is applied by compresses against the external surfaces of the lids. The heater contains multiple thermal sensors to regulate the temperature, precisely delivering 42.5°C to the inner surface of the eyelids for a 12-minute treatment. This duration was shown by Dr. Korb to effectively heat all of the meibomian glands in both the upper and lower lids.12

The instrument’s second major component consists of an inflatable air bladder that covers the external surface of the eyelid once the device has been inserted. During the treatment cycle, the bladder inflates and deflates so as to milk the heated glands. Both the upper and lower lids are squeezed between the inner heated surface of the shell and the outer air bladders. Thus, all of the glands of the upper and lower lids are simultaneously heated and expressed by this new proprietary technique.
Clinical studies thus far have shown a favorable response to this technology, including a series of 139 subjects comparing a single 12-minute LipiFlow treatment to 2 weeks of warm compresses. Majmudar found that the LipiFlow treatment was substantially more effective than the daily application of warm compresses in restoring the functionality of the meibomian glands within the lids. It also doubled the tear breakup time and reduced patients’ symptoms over a follow-up period of 1 month after treatment in this study.

**CONCLUSION**

Although MGD is not a new condition, we are coming to recognize its role in the pathogenesis of the dry eye syndrome. A new diagnostic and treatment paradigm will benefit the vast majority of dry eye patients and allow them to benefit from the latest forms of pharmacologic and mechanical treatments designed to evacuate and rejuvenate dysfunctional meibomian glands.

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