Dry Eye Diseases and Ocular Surgery: Practical Guidelines for Canadian Eye Care Practitioners

for the Canadian Dry Eye Disease Consensus Panel

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ABSTRACT:

In 2014, the Canadian Dry Eye Disease Consensus Panel published Guidelines for screening, diagnosis and management of dry eye diseases (DED). These did not address the implications of DED for individuals who are being considered for or have recently undergone ocular surgery. DED is common in certain surgical cohorts, and the perisurgical setting poses specific challenges, both because surgery can complicate preexisting DED and because symptomatic and non-symptomatic DED place the patient at risk of poor surgical outcomes. The Consensus Panel has developed this Addendum to the 2014 Guidelines to offer guidance on DED care before and after ocular surgery.

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Perisurgical Dry Eye Disease

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INTRODUCTION

Dry Eye Disease and the 2014 Canadian Guidelines

Dry eye disease (DED), also known as keratoconjunctivitis sicca, comprises a group of inflammatory ocular-surface diseases that, collectively, are ubiquitous[1, 2] in the general population. DED is highly prevalent in specific subpopulations, including adults over age 50,[1] women, people with long-standing diabetes,[3] and people who wear contact lenses.[4, 5] Rarer conditions associated with severe DED include Sjögren syndrome (SS),[6] Steven-Johnson syndrome, nocturnal lagophthalmos, exposure keratopathy, and graft-versus-host disease.[7, 8]

DED encompasses multiple conditions in which the precorneal tear film is scarce, unstable, inadequately distributed, or of abnormal composition. For instance, tears may be deficient in lipids (Meibomian gland dysfunction, a common cause of evaporative DED), water (e.g., in SS), or mucins (e.g., due to genetic deficiency or loss of conjunctival goblet cell function).[8] Tears in DED become hyperosmolar and may accumulate cytokines that trigger and perpetuate local inflammation. Hyperosmolar tears can damage the corneal and conjunctival epithelia, in part because they carry high levels of inflammatory mediators, such as the degradative enzyme matrix metalloproteinase-9.[9]

DED may present with any combination of ocular dryness, fatigue, redness, burning, itching or stinging pain, foreign-body sensation, and light sensitivity, as well as mucus filaments, eyelid irritation and crusting. Patients may report blurred or fluctuating vision and may experience reduced visual acuity and impaired functioning in visual tasks. Use of dyes for ocular-surface staining during routine exams often detects conjunctival and corneal abnormalities such as superficial punctate keratitis.[10] In extreme cases, DED can lead to corneal ulceration, neovascularization, permanent scarring, and irreversible visual loss.[11]

OCULAR NEUROPATHIC PAIN

Patients who suffer from neuropathic eye pain may describe their subjective experience in terms similar to those used for DED. Common pain descriptors used for both conditions include “burning,” “sharp,” and “gritty,” and both can cause light sensitivity. This similarity of presentation frequently leads to confusion and treatment dissatisfaction, particularly for individuals who develop persistent or intractable postsurgical pain.[24, 89]

The origins of neuropathic pain are obscure, but nerve damage from various sources may be a proximate cause.[23, 34, 90] Aberrant nerve regeneration and several other central and peripheral neurologic events[22] have been proposed to reinforce the neuropathy, causing the pain to become chronic.[24, 89]

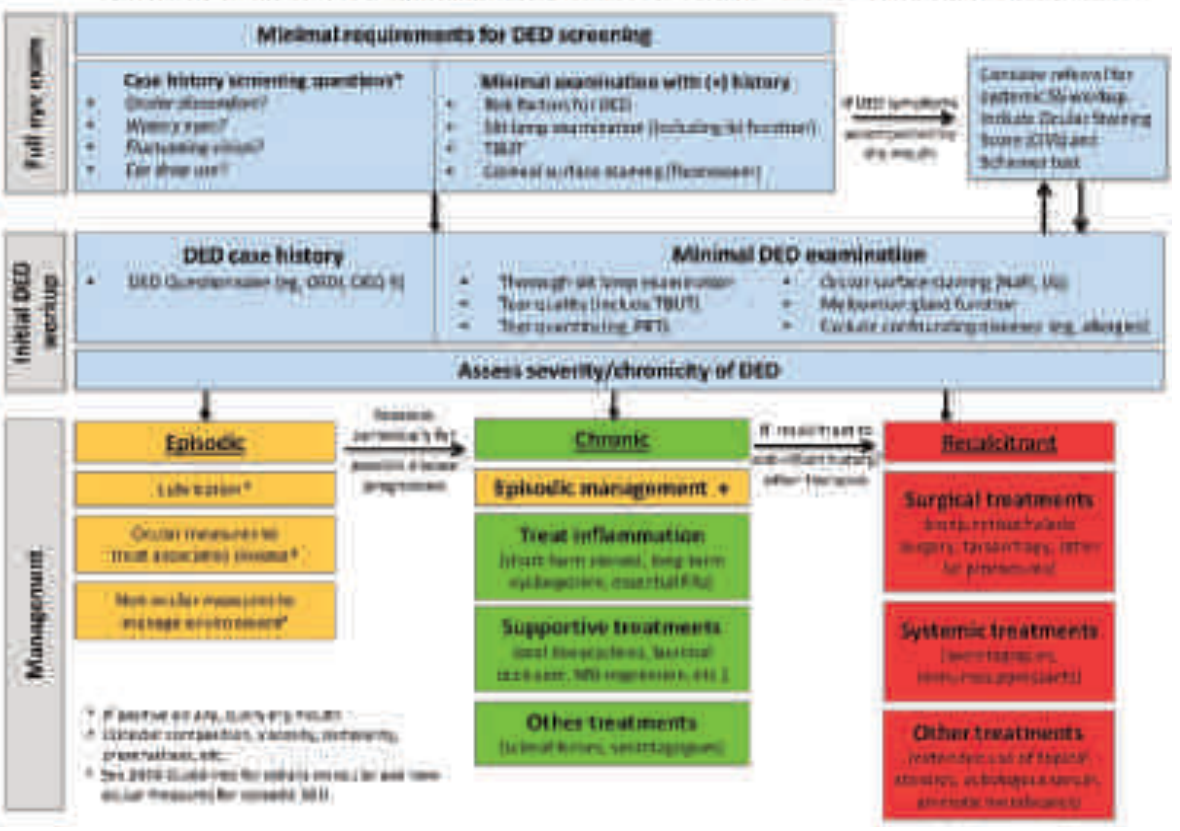
Eye pain may be identified for the first time as neuropathic following surgery. However, neuropathic ocular pain is also widely found in patients with no history of ocular surgery. Pain reported after surgery sometimes follows a pre-existing pattern that might have been misattributed to DED or other causes. A neuropathic origin should be suspected when ocular hypersensitivity, hyperalgesia (exaggerated pain response to suprathreshold noxious stimuli), or allodynia (pain in response to normally non-noxious stimuli) are not commensurate with objective signs of DED, such as corneal and conjunctival staining.

This possibility can be tested by instilling anesthetic drops, thus blunting nociceptive signals from corneal neurons.[22, 24] By definition, neuropathic pain originates in the brainstem or elsewhere in the central nervous system. Therefore, although this test is not diagnostic, the observation of pain that persists despite topical anesthesia is suggestive of neuropathy. Neuropathic pain is also reported to be poorly responsive to artificial tears, relative to physiologic (nociceptive) pain.[91]

Common comorbidities include general (non-ocular) neuropathic pain, as well as depression, anxiety, and sleep disturbance;[22, 92, 93] patients reporting otherwise-unexplained eye pain should be queried about these other conditions as well.

In 2014, the Canadian Dry Eye Disease Consensus Panel presented Guidelines on DED screening, diagnosis and management.[8] This guidance (Fig. 1) offered general principles for diagnosing and managing episodic, chronic, and recalcitrant DED and for monitoring the effectiveness of treatment. Based on these Guidelines, it is essential for all eye care professionals to screen for ocular-surface diseases and manage them appropriately. As discussed in the Guidelines, episodic DED can sometimes be managed with lubricating eye drops, eyelid hygiene, and/or modifications to the living and working environment. Conversely, chronic DED is an inflammatory disease that requires anti-inflammatory treatment.[8] Currently available options to manage ocular-surface inflammation include topical corticosteroids, which are generally reserved for short-term use, as well as essential fatty acids and cyclosporine 0.05% emulsion in castor oil (Restasis®, Allergan Inc.; throughout this Addendum, cyclosporine refers to this formulation).[8] Additional topical anti-inflammatory products, such as lifitegrast 5% and cyclosporine 0.1% in a cationic formulation, may soon reach the Canadian market for use in cases of DED.[12-17] To date, there have been no reports on the perisurgical use of these additional products.

Figure 1: *Diagnosis and management of DED, according to the 2014 Canadian Guidelines*[8]



Scope and Aims of this Addendum

The 2014 Guidelines did not address DED management in individuals undergoing ocular surgery. This topic is significant because of the variety of ways that uncontrolled DED can become problematic for patients undergoing procedures such as cataract surgery or refractive surgery. In addition, ocular surgeries of many types (Table 1) can induce ocular-surface inflammation, which results from direct trauma to the cornea, light toxicity from the surgical microscope, tear-film evaporation during surgery, and irritation due to topical anesthetics, surface antiseptic solutions, and preservative-containing eye drops.[18-21] As a result, surgery can precipitate *de novo* DED or exacerbate the condition in patients with pre-existing symptomatic or asymptomatic DED. Postsurgical ocular dryness and pain that present during the recovery phase are usually transient, but can persist in certain individuals. Chronic pain without significant ocular-surface disease ("pain without stain")[22] can also occur, and may be present before or after surgery; such neuropathic eye pain, resulting from lesions within the somatosensory nervous system,[23] is not readily distinguished from DED. This confusion poses a variety of clinical problems that can be frustrating for both patients and caregivers (see sidebar on *Ocular Neuropathic Pain*).[22, 24]

As discussed below, pre-existing ocular-surface disease complicates presurgical biometry, keratometry, topography and refraction, thus increasing a patient's risk of unsatisfactory visual correction and of refractive regression. DED can degrade the quality of vision (QoV) after surgery, leading to discomfort and dissatisfaction, even among patients whose visual acuity has been corrected effectively. Conversely, measures that restore the tear film, reduce tear osmolarity, or suppress corneal and conjunctival inflammation may improve postsurgical DED symptoms and visual outcomes. Thus, there are several compelling reasons to manage DED, both before and after surgery. Nevertheless, in surgical candidates and others, ocular-surface disease frequently goes unrecognized and untreated.[8, 25] It is crucial for eye care professionals to recognize DED signs and symptoms in surgical candidates, to begin treatment promptly, and wherever possible, to reverse ocular-surface inflammation before proceeding to surgery.

Table 1: *Ocular surgeries that may require DED diagnosis and management*

Type of surgery	Surgery induces or complicates DED?	DED complicates surgery?	DED highly prevalent in surgical cohort?	References
Refractive				
LASIK	Yes	Yes	Yes	This article
PRK	Yes	Yes	Yes	110
Cataract				
Phacoemulsification	Yes	Yes	Yes	This article
Corneal/conjunctival				
Penetrating keratoplasty	Yes	Yes	Yes	111-113
Endothelial keratoplasty	Yes	Yes	Yes	111-114
Pterygium excision	Yes	No	Yes	115
Conjunctivochalasis removal	No	Yes	Yes	116-119
Vitreo-retinal				
Vitrectomy	Yes	?	Yes	120, 121
Eyelid				
Blepharoplasty	Yes	No	Yes	122-124
Ptosis repair	Yes	?	No	122
Glaucoma				
Trabeculectomy	Yes	Yes	Yes	125, 126
Others				
Strabismus repair	Yes	No	No	127

Therefore, we developed this Addendum to the 2014 DED Guidelines for Optometrists specifically to address the perisurgical management of DED. The focus here is on cataract surgery, mainly phacoemulsification with intraocular lens (IOL) implantation and refractive procedures such as laser-assisted in situ keratomileusis (LASIK). Comorbid DED in glaucoma is discussed in the sidebar on *Dry Eye Disease in Glaucoma*.

DRY EYE DISEASE IN GLAUCOMA

DED is a common comorbidity with glaucoma, occurring in 15% to 59% of glaucoma patients.[94-99] This association can be explained at least in part by medicated eye drop polypharmacy, and specifically by the presence and duration of exposure to irritants found in medicated and non-medicated products, including active pharmaceutical ingredients and preservatives such as benzalkonium chloride (BAK).[99, 100]

Inflammation in response to BAK may be heightened by the presence of hyperosmolar tears,[101] as is common in DED. Moreover, chronic use of BAK-containing products can lead to increased tear-film osmolarity, and DED can complicate ophthalmic surgery, including procedures for glaucoma.[94] In patients using BAK-containing products, a preoperative course of topical steroids may decrease conjunctival inflammation and increase the rate of successful trabeculectomy; of course, it is essential to monitor intraocular pressure if steroids are used.[102, 103]

Preservative-free artificial tears (ATs) are associated with reduced DED symptoms[97] and should be used in preference to preserved ATs.[8] However, it is rarely feasible for patients with glaucoma to avoid the use of irritating topical medications, especially as the disease progresses and topical monotherapy must be abandoned.[100, 104, 105] For this reason, anti-inflammatory treatment may be considered, to ameliorate DED in patients requiring long-term exposure to topical glaucoma medications.[106] Whereas it has been generally assumed that controlling glaucoma is the primary goal when these two conditions co-exist, recent findings suggest that surface optimization in patients with comorbid glaucoma is compatible with reducing intraocular pressure.[107, 108]

Trabeculectomy and other surgeries for glaucoma temporarily exacerbate DED, but offer the prospect of long-term IOP control with greatly reduced need for topical glaucoma medications. Indeed, 40% of patients no longer require any topical glaucoma medications for up to 3 years post-trabeculectomy.[109] With the advent of less-invasive surgical procedures, it may be possible to allow for better control of IOP at an earlier point in the progression of glaucoma, thus reducing the chronic exposure to irritating topical medications. So-called micro-invasive glaucoma surgeries (MIGS) are *ab interno* procedures, meaning that they are carried out from inside the eye and cause minimal or no trauma to the conjunctiva. MIGS procedures are considered sufficiently low risk that their use can be justified even in individuals with mild to moderate glaucoma.[96] Although direct evidence is still lacking, MIGS procedures are therefore expected to carry less risk of inducing DED, compared to traditional surgery.

The general DED management approaches described in the Canadian Guidelines are considered to be applicable to patients requiring surgery. For this reason, this Addendum focuses on questions that are specific to perisurgical DED care, such as:

What evidence implicates uncontrolled DED in adverse post-surgical outcomes?
How should the presence of DED affect decisions on the timing or appropriateness of a procedure?
How should DED be managed before and after a procedure?
How should optometrists and ophthalmologists co-manage DED in individuals being considered for ocular surgery?

DED, VISUAL FUNCTIONING, AND OCULAR BIOMETRY

The precorneal tear film, the first refractive surface of the eye, functions best when mirror-smooth. The tear film is maintained by neuroendocrine mechanisms that regulate secretory function and the blink rate in response to shifting environmental stresses.[10, 26, 27] A healthy tear film is sufficiently thick, uniform, and balanced with appropriate components to protect the ocular surface from insult and to avoid optical aberrations between blinks.[28-31]

Abnormalities in the tear film, affecting either tear quantity or composition, can lead to aqueous-deficient or evaporative ocular-surface diseases. Surgical trauma compromises tear-film regulation, at least temporarily. For example, loss of tactile sensation at surgically denervated sites in the cornea impairs basal and reflex tearing and reduces the blink rate, leading to a compromised tear film and ocular surface while the damaged nerves regrow.[27, 32-34]

DED affects QoV without necessarily degrading visual acuity.[1, 29, 35] For instance, DED is associated with glare, impaired contrast sensitivity and symptoms of higher order aberrations (HOAs); patients experience fluctuating vision during the interval between successive blinks, due to a non-uniform and unstable tear film.[27] QoV is an important predictor of daily function, especially for tasks that place high demands on the visual system such as reading or driving.[36, 37]

In patients undergoing ocular surgery, visual disturbances related to DED may increase the risk of postsurgical patient dissatisfaction. For both refractive procedures and cataract surgery with IOL implantation, surgical planning requires accurate keratometry and/or topography. Uncontrolled DED can alter the shape of the cornea,[38] reducing the accuracy and precision of biometric findings. Tear hyperosmolarity is associated with greater statistical scatter in keratometric readings. Ocular-surface irregularity and instability reduce the precision of preoperative IOL and refractive calculations, increasing the risk of suboptimal refraction following surgery, especially when toric or multifocal implants are used.[39] In addition, even for individuals who achieve good postsurgical acuity, DED can reduce QoV over a period of weeks to months, and sometimes longer.[40]

Table 2 summarizes various goals for DED treatment in the general population and in candidates for ocular surgery.

Table 2: *Goals of DED management*

For all individuals with DED	For individuals with DED undergoing surgery
• To ameliorate eye discomfort and fatigue • To prevent corneal erosion and surface anomalies associated with ocular-surface disease and tear hyperosmolarity[3, 28, 32, 39] • To prevent optical aberrations that reduce visual quality (e.g., blurring, glare, loss of contrast sensitivity)[30, 35] • To improve performance and facility in demanding visual tasks (e.g., reading[36] and driving[37])	• To achieve more accurate and precise biometric/keratometric refractive measurements, allowing IOL power or LASIK surface parameters to be calculated more confidently • To improve postsurgical visual acuity and quality of vision • To prevent or minimize postsurgical DED[40]

The Growing Need for Collaborative Care for DED

Several demographic and societal trends have increased both the urgency and the burden of managing DED. First, both DED and ocular surgeries are increasingly common as the population ages. Second, the increasing reliance on and use of electronic devices can cause or exacerbate DED by decreasing the user's blink rate.[41, 42] People who use these devices require a high level of visual functioning and may seek surgical intervention specifically to improve their QoV. Third, ophthalmic technology itself has changed with the introduction of measurement approaches that offer unprecedented precision in ocular biometry but that rely on a healthy tear film if they are to be used optimally. Likewise, multifocal and toric IOLs offer the prospect of corrected near and distance vision, but they appear to be more sensitive than earlier-generation IOLs to both visual aberrations and errors in biometry.[43] This difference is intrinsic to the IOL technology, but dissatisfaction also results in part from higher expectations, including the desire for optimal visual functioning with minimal dependence on distance or reading glasses.

For all of these reasons, the growing need for diligent pre- and postsurgical DED care is placing increasing demands on caregivers' time. Efficient approaches, including collaborative DED management by optometrists and ophthalmologists, will be needed to meet these demands.

CATARACT SURGERY

If we extrapolate from 2014 data from a single province, approximately 450,000 phacoemulsification procedures may be carried out annually across Canada, making cataracts one of the most common reasons for ocular surgery.[44, 45] The demand for this surgery is projected to more than double by 2036.[46]

The current standard of care for cataract extraction is phacoemulsification followed by implantation of an IOL, which may be either monofocal or premium. The latter type of IOL includes a variety of designs that allow for correction of distance, reading, and intermediate vision, generally resulting in less dependence on glasses or contact

lenses. Depending on their design, premium IOLs may be prone to glare and halos, so it is important to minimize other visual disturbances in these patients, including aberrations related to DED.[47, 48] Interestingly, cataracts themselves can induce HOAs,[49, 50] and this effect on QoV is complicated by poorly controlled DED.[29, 30]

Pre-existing DED is common among individuals with cataracts, since some risk factors (notably advancing age, diabetes[3], and female sex[51]) predispose patients to both conditions.[52] However, DED commonly goes unrecognized and untreated in patients undergoing cataract surgery. Trattler et al. reported that, out of 136 American patients undergoing cataract surgery, while only 22% had a prior diagnosis of DED, a larger proportion showed objective signs of surface disease (tear break-up time (TBUT) ≤5 seconds, 63%; corneal staining, 77%) suggesting widespread underdiagnosis.[25] Interestingly, subjective symptoms of DED were less common, with only 31% reporting stinging and 41% reporting foreign-body sensation. These findings are consistent with previous reports suggesting that patient self-reporting is an unreliable screening tool for DED.[41, 53, 54]

Uncontrolled DED limits the accuracy of preoperative biometry, leading to errors in IOL power or placement.[41, 54] This effect has been clearly demonstrated using repeated readings in patients presenting for cataract surgery. Epitropoulos et al. reported that the mean difference between two successive keratometric readings was 0.28 D among individuals with hyperosmolar tears (n=100 eyes) versus 0.13 D among controls (n=50 eyes). Calculated IOL power differences were up to 5.5 D among 100 eyes with hyperosmolar tears, and the frequency of an IOL power difference ≥0.5 D was significantly higher with hyperosmolar versus normal tears (p=0.02). In addition, 17% of eyes with hyperosmolar tears but only 2% of eyes with normal tears showed a vector astigmatism difference ≥1.0 D (p=0.01) between readings.[39]

Onset of Symptoms of DED Following Cataract Surgery

Cataract surgery perturbs the ocular surface and induces intraocular and ocular-surface inflammation. In addition, the surgical procedure damages sensory and other neurons, and the resulting denervation reduces tactile and other sensation in the cornea.[40] *De novo* DED symptoms are common following phacoemulsification,[10, 27] but they are usually transient. Corneal hypoesthesia, tear-film instability, and other indicators of DED often resolve within 3 months, probably associated with the beginning of axonal regrowth.[18, 19] Corneal sensation gradually returns to near-preoperative levels over the course of 1 year.[40] In a small subset of patients, however, DED symptoms persist indefinitely.[20, 55] For instance, individuals with diabetes are at increased risk of severe and chronic postsurgical DED.[56]

Topical treatments should be applied consistently following surgery to limit the extent or duration of *de novo* DED.[52] Used alongside topical steroids, lubricants have been reported to improve symptoms of DED and visual functioning, relative to standard postsurgical topical care alone.[57, 58] Jee et al. directly compared the effects of preservative-free versus preserved steroid and lubricant eye drops after cataract surgery in 80 patients (80 eyes) with pre-existing DED. In this prospective, open-label study, patients received the preservative-free or preserved products 4 times daily for 1 month and twice daily thereafter. By Month 1 following surgery, subjects who received the preservative-free topical treatment reported significantly less severe symptoms compared to those who received preserved treatment ($p<0.05$). By Month 2, objective DED measures (staining, tear-film stability, inflammatory markers, and conjunctival goblet cells) were significantly improved with preservative-free treatment.[59]

Lubrication alone may be insufficient to manage the inflammation that drives chronic DED.[60-62] This has been shown most clearly in a randomized, multi-centre study of 233 Chinese adults with moderate to severe DED at baseline. Patients were randomized to twice-daily application of cyclosporine 0.05% or the emulsion that serves as its vehicle, with no other treatment permitted except for artificial tears. Whereas both groups experienced significant symptomatic improvement over baseline, significantly greater improvement was seen in corneal staining at 4 and 8 weeks (p=0.025 and 0.05, respectively) and in the Schirmer score at 4 weeks (p=0.035) with cyclosporine versus vehicle.[60] While no such vehicle-controlled study has been reported in a surgical setting, a prospective, contralaterally controlled study compared topical cyclosporine with saline in 32 patients undergoing bilateral phacoemulsification. In these patients, treatment with topical cyclosporine significantly improved tear-film stability and other measures of DED, relative to saline alone. While patient-reported DED intensity improved by the first month of treatment, clinical benefits became statistically significant by 2 months of cyclosporine treatment.[63]

Exacerbation of DED after Cataract Surgery

For patients with pre-existing chronic DED, anti-inflammatory treatment may improve postsurgical acuity and visual functioning.[47, 64] In a small, prospective, contralaterally controlled, randomized, double-masked study, Donnenfeld et al. compared lubricating eye drops (0.4% polyethylene glycol 400; 0.3% propylene glycol) to topical cyclosporine in patients undergoing bilateral phacoemulsification with multifocal IOL implantation. Of the 14 individuals studied, only 3 had been diagnosed as DED. However, because the mean baseline TBUT was low (approximately 6 seconds), others may have had undiagnosed or marginal DED. Treatment was initiated 1 month before surgery and maintained after the procedure. By Month 2 following surgery (i.e., after 3 months of topical treatment), uncorrected visual acuity was significantly better for eyes treated with cyclosporine than with lubricants alone (p=0.005). Contrast sensitivity was also improved with cyclosporine treatment, and there was a numeric trend toward greater tear stability at Month 2. Furthermore, corneal staining, which worsened from baseline in lubricant-treated eyes, improved significantly in cyclosporine-treated eyes (p=0.034 for the between-group difference at Month 2).[47]

Other studies have explored the postsurgical use of topical cyclosporine in cataract surgery.[63-65] It has been suggested that some treatment effects are seen within weeks of phacoemulsification[65] and other surgeries,[66] but these claims are difficult to evaluate, given that the established benefits of cyclosporine occur with longer-term use (≥3 months).[8, 54, 61]

REFRACTIVE SURGERY

LASIK and related photorefractive procedures are widely used to improve uncorrected visual acuity. These procedures generally lead to favorable outcomes and high patient satisfaction.[67, 68] However, postoperative dry eye is a possible complication of these procedures and a cause of discomfort, reduced vision, and overall dissatisfaction. As with cataract surgery (above), DED following LASIK is associated with the effects of surgical trauma on the ocular surface, including loss of sensation in the cornea[40] and suppression of blinking and both reflex and basal tearing.[69] Pre-existing DED is likely common and underdiagnosed, given that contact lens intolerance, which has been linked to DED, is a common motivator for patients requesting LASIK.[67]

Dry Eye Disease as a Factor in Patient Selection

Because refractive surgeries are elective, only individuals who have or can attain adequate ocular-surface health are considered candidates.[2, 32, 70, 71] Severe DED due to SS or other immune causes is usually considered a contraindication for surgical refractive treatment. However, some reports suggest that even these individuals may be candidates for LASIK if the condition is successfully managed before the procedure.[68, 72] To this end, pre-existing DED should be managed in a stepwise manner (Fig. 1), using therapies described in the 2014 Guidelines.[8] Notably, one retrospective study found that cyclosporine treatment for an average of 3.2 months (range 1–12 months) enabled those with mild DED to proceed with refractive surgery.[73]

Alternative photorefractive surgeries, including flapless procedures such as small-incision lenticule extraction (SMILE), appear to cause less nerve damage and less severe DED than does LASIK.[74, 75] Whether these newer procedures are preferable for patients at high risk of DED has not yet been established.[69, 72]

Transient versus Chronic DED Following LASIK

Following LASIK, tear-film instability and other DED signs and symptoms usually resolve spontaneously, but chronic postsurgical DED, persisting 6 to 12 months after LASIK, has been reported in 0.8% to 20% of patients.[70, 76] The risk of chronic post-LASIK DED increases with age and is greater in women than in men.[71, 73] It has also been suggested that hyperopic LASIK procedures carry a greater risk of chronic DED than does myopic LASIK.[73, 77, 78] Following hyperopic LASIK, patients with pre-existing DED and those who develop chronic DED appear to be at high risk of refractive regression.[70, 77]

In addition, pre-existing mild or subclinical DED appears to be a risk factor for chronic post-LASIK DED.[32] One prospective study followed 139 eyes undergoing LASIK. Subjects were excluded if they had a definitive diagnosis of DED, but could be included with isolated symptoms such as mild or moderate corneal staining or TBUT<5 seconds. In this population, presurgical corneal staining and low tear production were associated with DED persisting 1 year after LASIK.[76] Similarly, patients with reduced tear-film stability at baseline were at significantly greater risk of developing surface abnormalities over at least the first 6 months after LASIK.[79]

Use of topical cyclosporine following LASIK has been explored as a possible supplement to standard post-procedural care for patients with no prior diagnosis of DED. Peyman et al. reported that, in a series of 22 bilaterally

treated subjects, eyes receiving daily cyclosporine were significantly more likely to recover tactile sensitivity within 3 months following LASIK, relative to control eyes ($p \leq 0.011$).[80] The timing of this response is consistent with evidence that the clinical benefits of cyclosporine become significant after ≥3 months of treatment.[8, 61, 66]

Post-LASIK visual acuity is less satisfactory among patients who develop chronic DED relative to patients who have no such adverse effect.[70, 77, 78] In a large (n=565) retrospective analysis, Albietz et al. reported that refractive regression was associated with chronic DED (p=0.008 for DED at Month 12 vs no DED) and with pre-existing ocular-surface staining, reduced TBUT, and reduced corneal tactile sensation.[81] The authors noted that DED in their patients abated with persistent ocular-surface management, involving placement of lacrimal occlusion (punctal plugs), lid hygiene, and consistent use of lubricating eye drops. In eyes with evidence of post-LASIK DED, uncorrected visual acuity and DED signs and symptoms improved in parallel,[81] as has been reported outside the surgical setting.[82]

Taken together, these findings suggest that individuals at risk of post-LASIK DED benefit from topical treatments that restore the tear film and target inflammation.

Exacerbation of DED after LASIK

Salib et al. tested pre-LASIK DED treatment using cyclosporine versus unpreserved artificial tears in patients with a history of DED (n=21 [42 eyes]).[83] Those scheduled to undergo myopic LASIK began twice-daily treatment with the randomized drops 1 month before the procedure. For both of these treatments, corneal-surface staining and subjective DED symptoms improved by the time of the procedure. Topical treatment was maintained over the following 3 months. DED symptoms worsened transiently at the Week 1 observation, with faster recovery seen in the cyclosporine-treated eyes relative to eyes treated with artificial tears. From 3 to 12 months following LASIK, DED symptoms improved relative to the baseline evaluation in both treatment groups. Cyclosporine-treated eyes showed greater predictability in refraction over the 1-year observation period and were more likely to be within 0.5 D of their target spherical equivalent refraction. This difference was statistically significant by Month 3 (p=0.015).[83]

If cyclosporine is to be used in individuals being considered for LASIK, treatment should begin ≥1 month before the procedure and should be maintained for ≥3 months afterwards, to maximize the benefit when DED symptoms are most troublesome. Pretreatment may allow individuals who would not otherwise be considered for refractive surgery to undergo LASIK successfully,[32] and should be considered for those with evidence of mild or asymptomatic DED.[66, 80]

In addition to anti-inflammatory treatment,[66, 80, 83] pre- and post-LASIK therapeutic options include a broad range of options discussed in the 2014 Guidelines,[8] such as lubricating eye drops, scleral lenses, lacrimal occlusion, essential fatty acid supplementation,[84, 85] autologous serum, and oral doxycycline. Perisurgical data on most of these options are highly limited. In one small study of 12 individuals with residual refractive errors after LASIK whose uncorrected vision improved with the application of lubricating artificial tears, punctal plugs appeared to improve visual performance significantly ($p<0.0001$).[86] In addition, Di Pasquale et al. suggested that patients showing continued tear-film instability after ocular-surface inflammation has been addressed may benefit from an eye-warming procedure,[87] to stimulate Meibomian gland secretion and restore tear lipids.[8, 88] In an observational study on patients whose *de novo* DED persisted for 1 year after LASIK, the combination of punctal plugs, topical corticosteroids, and, where indicated, warm compresses, led to subjective symptomatic improvement in DED, as well as significant increases in tear lipid thickness and TBUT.[87]

COLLABORATIVE MANAGEMENT OF DED

Table 3 lists a series of goals for pre- and postoperative treatment of patients who are being considered for ocular surgery. The list is general, in that it could apply equally to cataract and refractive surgery and potentially to other procedures whose outcomes can be compromised by uncontrolled DED. Briefly, all patients who are referred for surgery consultation should be assessed carefully for symptoms and signs of ocular-surface disease. Patients with episodic or chronic DED should be treated prior to surgery with a goal of addressing both the signs and symptoms of DED and stabilizing the tear film and the ocular surface, to ensure that keratometric and topographic data are reliable and precise. Appropriate counseling during this treatment period is also critical to improve the patient's understanding of the condition and to manage expectations. After the procedure, ocular-surface disease may develop or worsen, at least transiently, despite the continued use of DED therapies initiated before surgery. The goal of care during this period should be to manage ocular discomfort and to restore a stable and healthy ocular surface.

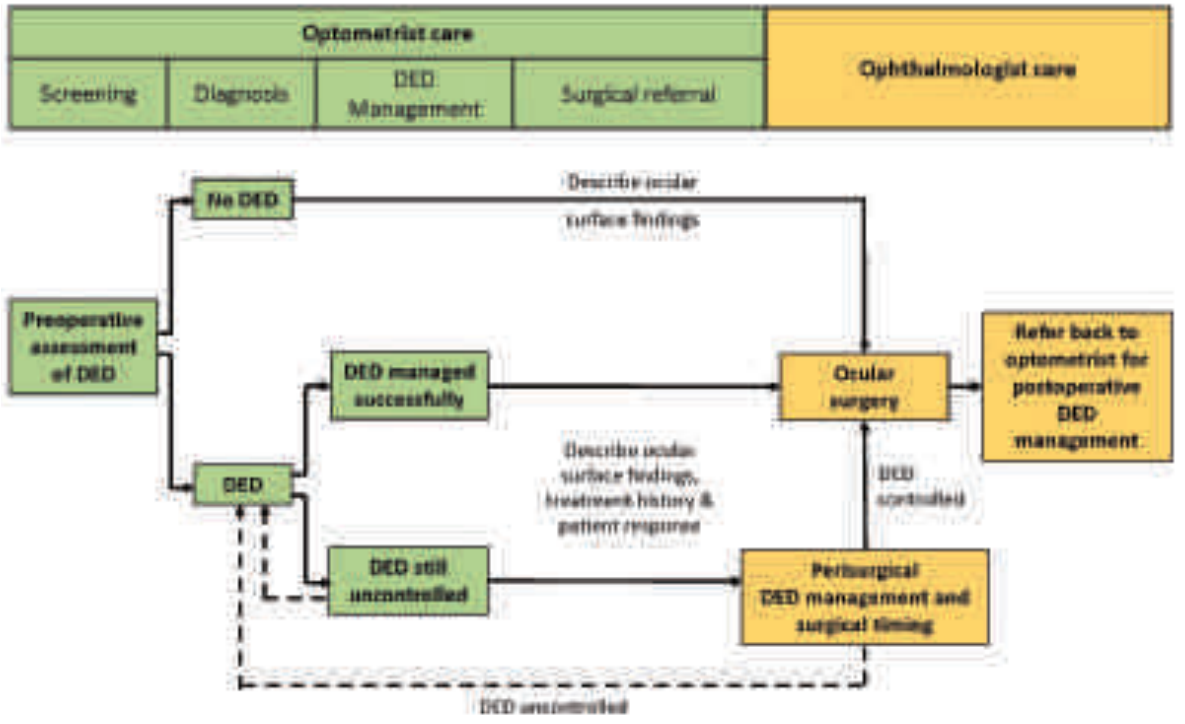
Table 3: *Goals for pre- and post-surgical management of DED*

In the preoperative setting, achieve:	In the postoperative setting:
1. Minimal DED signs on exam	1. Maintain presurgical treatment if ocular surface is stable
2. Minimal and controlled discomfort	2. Otherwise, intensify treatment until ocular surface is stable and returns to an adequate baseline
3. Stable, optimized tear film	
4. Stable keratometric readings (biometric and topographic)	
5. Stable manifest refraction	

While the locus of care before and after surgery may vary somewhat based on the circumstances, in general, optometrists are well-placed to deliver much of the DED care that patients will require before and after surgery. DED is often detected first while the patient is under an optometrist's care. Whether or not the patient is being considered for ocular surgery, the optometrist should assess the ocular surface and initiate appropriate treatment without delay. In all cases, the role of the tear film and ocular surface needs to be addressed with surgical candidates, to reinforce the need for good treatment adherence and to avoid postsurgical disappointment. If premium IOLs are being considered, the patient should be informed that these devices are particularly sensitive to ocular-surface disturbance.

Figure 2 shows a schema for the efficient co-management of patients requiring or requesting ocular surgery, who have been diagnosed with symptomatic or asymptomatic DED. In such cases, the optometrist should describe in a referral letter to the surgeon all ocular-surface findings, including subjective reports and objective evidence leading to this diagnosis. Ideally, the optometrist will manage the condition at this early point, both to improve the patient's immediate comfort and visual function and to streamline management of the surgery. The optometrist's correspondence should describe DED tests and treatments to date, as well as the outcome of these treatments. If appropriate, the optometrist may also recommend that the procedure be delayed to allow time to optimize the ocular surface.

Figure 2: *Proposed schema for the co-management of perisurgical DED*



The ophthalmologist will schedule surgery if the ocular surface is stable and healthy or will refer the patient back to the optometrist for ongoing or enhanced DED treatment. In some cases, the optometrist may request that the ophthalmologist assume care of the patient, including management of the ocular surface/cornea. Following surgery, it is generally appropriate for ongoing DED care to shift back to the optometrist, unless recalcitrant disease or complications manifest that require secondary or tertiary intervention. In a return report, the surgeon should describe the nature and outcomes of the procedure, as well as any changes that may have been made for postsurgical DED management, including changes in prescription and non-prescription topical treatments.

CONCLUSIONS

DED is a chronic inflammatory disorder that optometrists and ophthalmologists encounter on a routine basis. It should be assessed and appropriately treated in all patients. The principles of DED treatment are similar whether or not ocular surgery is being considered and should follow the recommendations of the 2014 *Canadian Dry Eye Disease Guidelines*.[8] However, DED management is particularly important before and after certain surgical procedures, specifically refractive and cataract surgery, because uncontrolled DED may place the patient at risk of less-than-optimal surgical outcomes and, conversely, surgery commonly induces or exacerbates DED. Surgery should therefore be delayed until the ocular surface has been stabilized and an adequate and appropriate tear film is restored, to the maximum extent possible. As outlined in Figure 1, anti-inflammatory treatment for patients with pre-existing chronic DED should be initiated before surgery and maintained for some months afterwards. Consensus recommendations for the management of perisurgical DED are shown in Table 4.

Table 4: *Consensus recommendations for the management of perisurgical DED*

Recommendation 1. For patients with suspected ocular neuropathic pain, it is important to identify associated conditions, such as non-ocular neuropathic pain, depression, anxiety, and sleep disorders.
Recommendation 2. Depending on frequency of use, preservative-free formulations of medicated and non-medicated topical products should be considered for use before and after ocular surgery.
Recommendation 3. Independent of self-reported eye discomfort, patients undergoing cataract surgery should be assessed for signs and symptoms of DED.
Recommendation 4. The ocular surface should be optimized prior to cataract surgery, to increase the accuracy and precision of preoperative biometry and to improve postoperative comfort and visual functioning.
Recommendation 5. Patients with pre-existing symptomatic or asymptomatic DED should be considered for treatment with anti-inflammatory agents prior to surgery, to prevent exacerbation of symptoms.
Recommendation 6. A dedicated DED assessment should be conducted as part of work-up in all patients being considered for refractive surgery.
Recommendation 7. Signs and symptoms of chronic DED, including mild DED, should be evaluated and managed in all candidates for ocular surgery.
Recommendation 8. Patients with ocular-surface staining, tear-film instability, or other signs of DED should be counselled about the risk of exacerbation of DED following ocular surgery.
Recommendation 9. Irrespective of any prior history of DED, patients undergoing ocular surgery should be counselled that DED symptoms can occur following the procedure.

Efficient perisurgical care for patients with DED requires that optometrists and ophthalmologists co-manage the condition and communicate effectively with one another and provide consistent messages to the patient about the ocular-surface findings, risks, and response to treatment. Checklists describing best practices for correspondence between the surgeon and the optometrist are provided in Supplementary Table 1.

Supplementary Table 1: *DED-related information to include in cross-referrals between optometrists and ophthalmologists*

	Optometrist to ophthalmologist	Ophthalmologist back to optometrist
Symptoms	• Sensation • Vision } *Indicate timeline*	• Sensation • Vision • Description of recent surgical procedure • Outcomes (*include postsurgical refraction if done*)
Signs/assessment	Findings • Symptom score (questionnaire) • Tear stability (NIBUT/TBUT) • Corneal (NaFl) and conjunctival staining (LG) • Meibomian gland function • Other testing (e.g., tear osmolarity)	
	Description of DED/ocular surface • Severity • Currently stable? (*if Yes, how long since stability achieved?*)	
Management	• Current and past treatments • Tolerability issues, if any • Adherence history	• Any postsurgical changes made to ocular-surface management, including non-prescription products? (*if Yes, specify products and dosing*)
Recommendations	• Should the patient be scheduled for the earliest possible surgery? • If No, what therapeutic milestones should be achieved before scheduling surgery?	• Instructions for postsurgical care • Next scheduled clinic visit *or* returning to optometrist's care *or* co-management • Patient's goals for vision

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