OPTOMETRIC CLINICAL PRACTICE GUIDELINE

Care of the Patient with

Ocular Surface Disorders

American Optometric Association®

OPTOMETRY: THE PRIMARY EYE CARE PROFESSION

Doctors of optometry are independent primary health care providers who examine, diagnose, treat, and manage diseases and disorders of the visual system, the eye, and associated structures as well as diagnose related systemic conditions.

Optometrists provide more than two-thirds of the primary eye care services in the United States. They are more widely distributed geographically than other eye care providers and are readily accessible for the delivery of eye and vision care services. There are approximately 36,000 full-time-equivalent doctors of optometry currently in practice in the United States. Optometrists practice in more than 6,500 communities across the United States, serving as the sole primary eye care providers in more than 3,500 communities.

The mission of the profession of optometry is to fulfill the vision and eye care needs of the public through clinical care, research, and education, all of which enhance the quality of life.

Healthy People 2020

OPTOMETRIC CLINICAL PRACTICE GUIDELINE
CARE OF THE PATIENT

WITH OCULAR SURFACE DISORDERS

Reference Guide for Clinicians

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INTRODUCTION

Optometrists, through their clinical education, training, experience, and broad geographic distribution, have the means to provide effective primary eye and vision care to a significant portion of the American population and are often the first health care practitioners to diagnose an ocular surface disorder.

The ocular surface is a delicate structure. Its proper functioning is dependent on a number of systems that contribute to its physiological integrity. In addition, the ocular surface is vulnerable to potential environmental insults by the nature of its function and anatomic location. The integrated functions of various components of the ocular surface must perform optimally. Disruption of the system may or may not produce symptoms. Therefore, investigation of the component parts of the ocular surface must necessarily be done without disruption (or at least minimal invasion) of physiological function. Symptomatic patients may try to solve their perceived problems with self treatment. Such approaches may delay accurate diagnosis of ocular surface disease.

This Optometric Clinical Practice Guideline for the Care of the Patient with Ocular Surface Disorders describes contemporary and appropriate examination, treatment and management protocols to reduce the risk for visual discomfort and disability. The most common ocular surface disorders stem from tear-film abnormalities and lid-gland dysfunction ("blepharitis"), either of which may lead to ocular surface disorders.

The use of terms such as dry eye (DE), ocular surface disease (OSD), or deficient tear syndrome (DTS), represents attempts to describe signs of clinical damage to the intrapalpebral ocular surface or symptoms of such disruption from a variety of causes. Each of these terms has its drawbacks, due to either lack of inclusiveness or universal acceptance signifying the evolution of our understanding of the topic. In this Guideline, "ocular surface disorders" will be used to encompass these disease entities as well as the related symptoms that result from a variety of abnormalities. These include abnormal lid anatomy or function, abnormal or altered tear production or composition, and related

subclinical signs. As knowledge expands, terminology will evolve as classification schemes are refined based on anatomy or pathophysiology.

This Guideline contains recommendation for timely diagnosis, treatment and, when necessary, referral to, consultation with or treatment by another health care provider. This Guideline is intended to assist optometrists in achieving the following goals:

- Identify patients at risk for developing ocular surface disorders
- Accurately diagnose patients with ocular surface disorders
- Differentially diagnose age, drug, environmental and systemic disease-related causes of ocular surface disorder
- Improve the quality of care rendered to patients with ocular surface disorders
- Reduce the prevalence and degree of disability and morbidity, including the financial burden, secondary to ocular surface disorders
- Inform and educate patients and other health care providers about the visual complications, risk factors and treatment and management options associated with ocular surface disorders.

I. STATEMENT OF THE PROBLEM

Conditions that alter the production, composition, or distribution of the preocular tear film (POTF) may result in symptoms or signs of damage to the structures of the ocular surface. These situations may lead to noticeable irritation, reduction of visual function, and even chronic tissue changes. Such conditions are often related to abnormalities of the structure or function of the eyelids, glands of the lid and their secretions, conjunctiva, or cornea. Additional consequences of chronic compromise to the ocular surface include risk of infection and chronic inflammation that may not respond to treatment. A classification scheme uses the term deficient tear syndrome (DTS) to encompass these "dry eye" etiologies.[1]

An International Dry Eye WorkShop (DEWS) report has separated dry eye systematically into aqueous-deficient and evaporative. Subclassifications of the former include those associated with the Sjögren's syndrome (SS) and non- Sjögren's dry eye, which includes lacrimal-gland dysfunction of both primary and secondary etiologies. The evaporative disorders are subdivided into intrinsic and extrinsic caegories.[2] The role of inflammation in dry eye and ocular surface disorders has been emphasized as a consequence of hyperosmolarity.[3,4]

The reported epidemiology of dry-eye conditions varies. Depending on the definition, population studied, criteria of inclusion, and other factors, the incidence and prevalence are often difficult to estimate. For example, the prevalence of dry eye among the Asian population may be greater than that of Caucasian populations.[5] It has been estimated that 5 million Americans over the age of 50 years have dry eye,[2] and 25% of the US population reports or suffers from dry eyes or some abnormality of the exposed ocular surface.[6,7] Because there are many undiagnosed cases, due to situational or environmental contributors, the actual number is probably much greater.

A complete group of tests for ocular surface disorders has been reported by the Dye Eye WorkShop (DEWS).[8] These include but are not limited to patient history, questionnaire, tear film break up time using fluorescein vital dye staining, Schirmer testing, and evaluation of lid and meibomian

gland morphology and excretion. This practical sequence was based on earlier protocols.[9,10] Careful clinical observation and accurate diagnosis along with timely and appropriate intervention can eliminate or minimize the deleterious effects of ocular surface disorders and improve the patient's quality of life.[11]

This Guideline will offer a brief historical outline of earlier classifications and a summary of the current understanding regarding diagnosis and management of ocular surface disorders. Identifying patients at risk for these conditions and offering appropriate treatment options will help to ensure cost-effective care and improvement in the quality of life.

A. Description and Classification of Ocular Surface Disorders

1. Normal Lid Margin Anatomy

The lid margin is about 2 mm thick and has a thin gray line separating its anterior and posterior portions. The anterior portion has two or three rows of eyelashes. The posterior border, in close apposition to the globe, contains the orifices for the tarsal glands. The meibomian glands—approximately 30 to 40 in the upper and 20 to 25 in the lower lid—are embedded in the tarsal plates and secrete lipids that comprise the oily layer of the tear film.[12,13] Although the maximum tear capacity of the ocular surface and fornices is about 25 µL, the normal volume is only about 7 µL. Each blink renews the tear film and distributes a fresh layer across the exposed cornea and conjunctiva.

2. Normal Tear Film Composition

The preocular tear film (POTF) has three identified but dynamically interacting layers – lipid, aqueous, and mucous.[10] The pilosebaceous meibomian glands in the lids produce most of the outermost (lipid) layer. The Zeis and Moll glands of the eyelid margins, which are associated with the lashes, also contribute to this layer. Oily secretions in this layer function to contain the aqueous phase of the POTF by reducing surface tension. In addition, the lipid layer stabilizes and retards evaporation of the underlying aqueous layer.[12,14,15]

In the normal healthy eye, the lipid layer thickness is less than 0.1 µm. Meibomian lipids (meibum) are mainly waxy and cholesterol esters.[13,16] High molecular weight and low polarity are important properties for the formation, stabilization, and protection of the POTF; alteration of polarity in disease states such as blepharitis may have an adverse effect on its stability and lead to ocular surface disorders and symptoms of dry eye. Interference fringe patterns become distorted in the presence of a contaminated or thickened lipid layer.[13,17] In addition, meibomian secretions may be distinctly altered in patients with meibomian gland dysfunction.[18]

The aqueous layer makes up about 90 percent of the POTF. The major contribution to this layer comes from the accessory exocrine lacrimal glands of Krause and Wolfring.[19,20] The aqueous layer contains lysozyme and proteins, including lactoferrin, that exhibit antibacterial activities. Laboratory analysis may prove useful for diagnostic evaluation of the aqueous layer.

The innermost layer of the POTF is the mucous layer. Produced primarily by the goblet cells of the conjunctiva, mucus lubricates the lids and serves as an adsorbing interface between the aqueous layer and the hydrophobic corneal epithelium. In addition, it collects cellular debris from the ocular surface.[20,21] The glycocalyx on the epithelial microvillae anchors the mucous layer.[10] The model for tear film breakup is based partially on thinning of the aqueous layer and subsequent contact between the lipid and mucin layers.[9,22] Other mechanisms, such as neural receptors, may play a role in tear film breakup.[23]

Therefore, ocular surface disorders can result from compromise to the structure or function of the conjunctiva, eyelids and their glands, conjunctiva and its accessory glands, or cornea. This Guideline describes the most common clinical etiologies of ocular surface disorders: blepharitis and dry eye. (See Appendix Figure 1 for ICD-10-CM Classification.)

3. Dry Eye-Related Ocular Surface Disorders

The term “dry eye” refers to ocular surface disorders in which the common etiology is aqueous deficiency or disruption. The DEWS definition of dry eye disease encompasses both symptoms and objective evidence of ocular surface disruption and characterizes the breadth of the problem: "Dry eye is a multifactorial disease of the tears and ocular surface that results in symptoms of discomfort, visual disturbance, and tear film instability, with potential damage to the ocular surface. It is accompanied by increased osmolarity of the tear film and inflammation of the ocular surface."[24]

Because dry eye involves more than aqueous deficiency, the term "dry eye" alone is inadequate to describe ocular surface disorders. At least three classification schemes have been proposed to help clarify the complexity of ocular surface disorders and dry eyes (Appendix Figures 2-4).[1,24,25] Each has its unique aspects and is worthy of consideration by the practicing clinician.

The 1995 Classification Scheme based on the National Eye Institute (NEI)/Industry Workshop separated dry eye into deficient aqueous tear production and increased evaporative loss.[25] Increased evaporative tear loss is associated with eyelid disorders and meibomian gland dysfunction (MGD), as well as exposure, contact-lens wear, and environmental situations (Appendix Figure 2). Anterior blepharitis, as classified by Thygesson, involves eczema and lash manifestations but has been superseded by an emphasis on posterior blepharitis.[26]

In 2006, a Delphi panel proposed a classification scheme for “dysfunctional tear syndrome” (DTS), which comprises a range of disorders. DTS is subdivided into groups with and without lid margin disease as well as tear distribution abnormalities. In addition, this group developed a severity scale based on symptoms and signs (Appendix Figure 3). This report also proposed treatment guidelines that represent perhaps the most useful management algorithm for practicing optometrists.[1]

The 2007 DEWS report expanded on the 1995 classification scheme of the NEI and Industry groups to expand the causes of ocular surface disease to include allergic conjunctivitis, chronic keratoconjunctivitis, conjunctivitis, and post-refractive surgery (Appendix Figure 4).[24] Each of these classifications suggests that ocular surface disorders are complex manifestations that have numerous etiologies which may interact with each other. These interactions are the result of the multiple components of the ocular surface that protect its physiological integrity.

What has also emerged is the importance of underlying inflammatory processes in ocular surface disorders. This has been emphasized in various publications and reviews as a basis for etiopathology and treatment.[27-29]

Included among dry eye-related ocular surface disorders are the following:

- Aqueous-deficient dry eye associated with the Sjögren's syndrome
- Non-Sjögren's aqueous deficiency (e.g., age-related)
- Blepharitis
- Anterior (lash- and lid-associated)
- Posterior (lid margin- and meibomian gland-associated)
- Contact lens-related evaporative tear disruption
- Blink and lid anatomy abnormalities
- Situational and environmental evaporative tear loss
- Conjunctivochalasis (redundant bulbar conjunctival tissue)
- Allergic, chronic infective, and non-infective conjunctivitis and keratoconjunctivitis
- Post refractive-surgery disruptions of the ocular surface or POTF.

These disorders may overlap as well as co-exist. Management requires precise diagnostic criteria and specific interventional strategies.

One paradox is that patients who have increased reflex tearing may suffer from ocular surface disorders for which the irritation serves as the stimulus. Moreover, patients whose puncta have collapsed (stenosis) may have reduced tear clearance, which may compensate for reduced aqueous production.[30]

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a. ***Aqueous-Deficient Dry Eye***

The symptoms of aqueous deficient dry eye are usually bilateral and may produce foreign-body sensation and lacrimation. Aqueous-deficient dry eye results from reduced aqueous production and may be secondary to lacrimal-gland output deficiency as seen in Sjögren's syndrome. Appearing clinically with reduced tear meniscus, and debris and strands of mucous in the tear film, it can lead to the formation of corneal filaments (filamentary keratitis) in advanced cases. Additional clinical signs include reduced tear breakup time and decreased wetting on Schirmer testing, as well as ocular surface staining, although these latter signs are not specific to aqueous deficient dry eye.

Sjögren's syndrome (SS) is a chronic systemic inflammatory disorder characterized by lymphocytic infiltrates in exocrine organs. Most sufferers present with symptoms such as xerophthalmia (dry eyes), xerostomia (dry mouth), and parotid gland enlargement. Extraglandular features may develop, including arthropathies such as arthralgia, arthritis, and myalgia. In addition, Raynaud's phenomenon, pulmonary disease, gastrointestinal disease, leukopenia, anemia, lymphadenopathy, neuropathy, vasculitis, renal tubular acidosis, and lymphoma may be accompanying manifestations.

Primary SS occurs in the absence of other underlying rheumatic disorders. In contrast, *secondary* SS is associated with at least one other underlying rheumatic disease, such as systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), or scleroderma. Given the overlap of SS with other rheumatic disorders, it may be challenging to determine whether a particular clinical sign is exclusively a consequence of Sjögren's syndrome or accompanies an disorder.*

Aqueous deficiency secondary to SS results from lacrimal gland inflammation, infiltration, and atrophy.[31] Thought to be autoimmune in origin, primary SS is associated with collagen-vascular or connective tissue disease, most frequently rheumatoid arthritis. Briefly, primary SS involves the glands of the lids and mouth. Secondary SS involves a

* http://emedicine.medscape.com/article/332125-overview accessed 5/13/2010.

[redacted]

systemic autoimmune disease such as rheumatoid arthritis, which then results in the symptoms of dry eye or dry mouth.[31,32] A detailed review of the distinction between primary and secondary forms of SS is beyond the scope of this Guideline but can be found in literature reviews. [31,32] Dry eye symptoms may be the first manifestation of SS.

Lacrimal insufficiency occurs most often in menopausal women; its onset is typically during the fifth decade of life. Clinical signs and severe symptoms have been associated with estrogen, taken alone or in combination with progesterone or progestin as hormone replacement therapy (HRT).[33] It also may occur in women who are pregnant or taking birth control pills, in whom estrogen and prolactin levels are elevated.[34,35]

Other local, systemic, and exogenous conditions that can adversely affect tear production include:

- Dacryoadenitis
- Facial nerve paralysis
- Chemical burns
- Congenital alacrima
- Gamma radiation
- To varying degrees, systemic medications: antihypertensives (diuretics, adrenergic antagonists, and beta-blockers); antihistamines (especially first-generation H-1 inhibitors); medications that have anticholinergic effects (tricyclic antidepressants, phenothiazines, etc.); and hormone replacement therapy (estrogen, progesterone)
- Congenital dysautonomia (Riley-Day syndrome)

b. Mucin-Deficient Dry Eye

Reduction in the number of conjunctival goblet cells, resulting in a decrease in mucin production, can be caused by conditions that damage the conjunctiva. Mucin-deficient dry eye conditions include allergic conjunctivitis,[36] ocular cicatricial pemphigoid (OCP), erythema multiformae (Stevens-Johnson syndrome; SJS), severe trachoma, or chemical (especially alkali) burns. Impaired goblet cell function can also result from marked vitamin A deficiency, although it is rare in developed countries. Most recently mucin-deficient dry eye has been reported as a consequence of facial nerve paralysis.[37]

In OCP and SJS, goblet cell loss is due to an autoimmune response that deposits immunoglobulins at the basement membrane zone of the conjunctiva.[38] This then leads to the clinical picture of bullae at the subepithelial level. Progressive infiltration results in contraction of the conjunctiva with symblepharon formation.[39]

Paradoxically, Goblet cell density may increase secondary to thermal or chemical injury.[40] The resulting ocular surface disorders differ from OCP or SJS at the cellular level though appearing clinically similar. A grading system is available for the ophthalmic manifestations in patients with chronic SJS.[41] This includes conjunctival damage such as the development of symblepharon and anklyoblepharon as well as corneal vascularization and conjunctivalization. The scale is quantitative and continuous (range, 0–39).

c. Surface Abnormalities

Any structural defect of the lid can interfere with tear film distribution. Impairment of normal blink action usually results in an irregular mucin layer. A term that may represent these situations inclusively is "lid-wiper epitheliopathy."[42-45] Incomplete or infrequent blinking, which results in excessive tear evaporation and exposure keratopathy, can be caused by Bell's palsy, lagophthalmos, thyroid-related eye disease, foreign body, or lid trauma. Other lid abnormalities that prevent efficient resurfacing of the tear layer include ptosis, trichiasis, and madarosis.

d. Epitheliopathies

Corneal epitheliopathies are characterized by an irregular epithelial surface where microvilli are prevented from allowing mucin to adhere to the cornea. The causes include corneal scars, chemical burns, recurrent corneal erosions, contact lens complications, trauma from entropion or refractive surgery, incomplete blinking, or lash abnormalities such as trichiasis and distichiasis. Lid-wiper epitheliopathy is an all-inclusive term for such disorders that are related to contact lens wear or occur following refractive surgery.[56-59]

e. Contact lens wear

Contact lens wear can induce dry eye symptoms in patients who have a pre-existing, asymptomatic, marginally dry eye condition.[60] Not only do contact lens materials require greater surface wetting than the corneal epithelium, but wearing contact lenses thins the POTF and interferes with the spreading of mucin onto the cornea. Refitting a dry eye patient with silicon-hydrogel lenses has been found to provide symptomatic relief of dryness for up to three years following refitting.[61]

4. Blepharitis

This Guideline will review the historic Thygesson classification of what is now recognized specifically as anterior blepharitis, as well as the Delphi panel's algorithm for classification of dysfunctional tear film states. It will describe blepharitic conditions and their management. It will also address the contributions of tear distribution abnormalities in the context of surfacing abnormalities, epitheliopathies, and their management.

a. Ocular Surface Disorders Arising from Lid-Margin Disorders (Anterior Blepharitis)

Major contributing factors to the alteration of lipid secretion are lid and lash disorders, which may be potentiated by inflammatory elements. Any of the forms of blepharitis may represent the initial sign of altered

lipid secretions that result in premature evaporation of aqueous tear components.

Anterior blepharitis. Dermatologic manifestations of anterior blepharitis involve the keratinized lid skin and may include eczema, which is typically secondary to allergic contact dermatitis.[46-48] Other etiologies of anterior blepharitis include infection, seborrhea, and the combination of both.[26]

Staphylococcal blepharitis. Usually caused by one of two *Staphylococcus* species, *S. aureus* or *S. epidermidis*, staphylococcal blepharitis is an inflammation of relatively short duration. It is more prevalent in warmer climates and often occurs in middle-aged women who have no other skin abnormalities. In addition to the hallmark signs of lid swelling—erythema of the lid margins, scaly collarettes at the base of the lashes, and possible skin ulceration—a frequent result is evaporative dry eye due to the inefficient lipid-layer function. An aqueous-deficient component accompanies this situation.[49,50] Hordeola and chalazia are potential coexisting conditions.

Seborrheic blepharitis. Also called squamous blepharitis, seborrheic blepharitis is part of a dermatologic condition that includes the scalp, face, and eyebrows (seborrheic dermatitis), all of which have cultured with populations of normal surface organisms. It is present in 1 to 3 percent of immunocompetent adults, and is more prevalent in men than in women. Although skin inflammation is not necessarily evident, greasy, foamy scales called scurf surround the bases of the cilia. Seborrheic dermatitis may be seen in conjunction with other skin diseases, such as rosacea, and acne vulgaris. *Malassezia* yeasts have been associated with seborrheic dermatitis. Abnormal or inflammatory immune system reactions to these yeasts may be related to development of seborrheic dermatitis.[51]

Seborrheic/staphylococcal blepharitis. Another common form of anterior blepharitis is combined seborrheic/staphylococcal, or mixed, blepharitis.[52] Associated with seborrheic dermatitis, it is characterized by secondary keratoconjunctivitis, papillary and follicular hypertrophy, conjunctival injection, and mixed crusting. Its severity waxes and wanes

over its chronic course. Bacterial cultures are positive in approximately 98 percent of cases. The organisms found most frequently have changed from *S. aureus* to *S. epidermidis*, *Streptococcus* (A and B), *Bacillus* sp., *Corynebacterium* sp., *Propionibacterium*, *Escherichia coli, Pseudomonas* sp., *Citrobacter* sp., *and Candida* sp.[53] Histological examination reveals chronic, moderate, nongranulomatous inflammation.

Meibomian seborrheic blepharitis. Meibomian seborrheic blepharitis can be identified by the presence of increased meibomian and seborrheic secretions without inflammation. Tears are foamy and sudsy, resulting in burning symptoms, especially in the morning. Itching and tearing are common concurrent symptoms. The meibomian glands are dilated, leading to copious secretions and bulbar conjunctival injection. The clinical signs are consistent with disturbed meibomian gland function. This form of blepharitis may be more appropriately grouped with the posterior variety.

Seborrheic blepharitis with secondary meibomianitis. Seborrheic blepharitis with secondary meibomianitis (meibomitis) is similar in clinical presentation and symptoms to seborrheic blepharitis. However, it has episodic inflammation and meibomianitis that result in a spotty presentation of clogged meibomian glands and anterior seborrhea. Lipid secretions are of toothpaste consistency, contributing to an unstable POTF. Cultures reveal the presence of normal flora. This form of blepharitis may also be grouped with the posterior variety. The clinical signs are consistent with disturbed meibomian gland function.

Meibomian keratoconjunctivitis. Meibomian keratoconjunctivitis (primary meibomianitis) is the most severe lid margin inflammation. Typically occurring during the fourth decade of life, it has no predilection for gender but is more common in colder climates. It is frequently associated with rosacea and is part of a generalized sebaceous gland dysfunction pattern that clogs the meibomian gland opening with desquamated epithelial cells. This is most likely due to altered polarity of the lipid secretion.[12] Because lipid secretions have a higher melting point than the ocular surface temperature, stagnation of free fatty acids within the gland's inspissated opening results in a lipid-deficient tear film. It is very likely that this form of blepharitis should also be grouped

with the posterior variety. The clinical signs are consistent with disturbed meibomian gland function.

Angular blepharitis. Angular blepharitis is localized to the lid at the outer canthus. The staphylococcal form is typically dry and scaly while the form caused by *Moraxella (Morax-Axenfeld*) diplobacillus is wet and macerated, and has a whitish frothy discharge. There is the possibility of secondary bacterial conjunctivitis or keratitis resulting from the *Moraxella* organism.[54]

Demodicosis. Demodicosis is the inflammatory reaction to a common mite that inhabits the eyelash follicles in persons over the age of 50 years. There are two species of mite, *Demodex folliculorum* and *Demodex brevis*. *D. folliculorum*, which is present in hair and eyelash follicles, consumes epithelial cells, produces follicular distension and hyperplasia, and increases keratinization, leading to cuffing at the base of the cilia. *D. brevis*, which is present in sebaceous and meibomian glands, may destroy the glandular cells, produce granulomas in the eyelid, and plug the ducts of the meibomian and other sebaceous glands that affect formation of the lipid layer. *Demodex* has been associated with rosacea, but a causal relationship has yet to be established.[55,56]

b. Ocular Surface Disorders Arising from Lid-Margin Disorders (Posterior Blepharitis)

Posterior blepharitis is recognized as a significant cause of disruption of the tear film.[57] Meibomian gland secretions represent a complex functioning unit that interacts with the lids as well as the aqueous layer of the tear film. Some models suggest that the appropriately functioning lipid layer comprises both non-polar and polar components.[12] Abnormal functioning of the meibomian glands results in the clinical signs and symptoms of meibomian gland dysfunction (MGD), including distinct changes in viscosity and clarity of expressed contents, increased tear film osmolarity, which may be reflected by complaints of burning and stinging, and premature evaporation, leading to decreased tear-film stability.[58,59] Clinical signs of ocular surface damage include, for example, epithelial staining. Clinicians should observe the lids for

apposition to the globe, teleangiectasis at the lid margin, and obstructed meibomian gland orifices.

B. Epidemiology of Ocular Surface Disorders

1. Dry Eye

a. Prevalence

In terms of prevalence and characterization, dry eye may be the most ill defined of all ocular disorders. Contributing factors include the lack of a defined diagnostic test or protocol and the lack of congruity between patient symptoms and clinical tests.

Severe forms of aqueous-deficient dry eye can be associated with systemic diseases, especially collagen-vascular diseases. Up to 20 percent of persons with rheumatoid arthritis have keratoconjunctivitis sicca (KCS).[46,62] Patients with Sjögren's syndrome have the classic triad of dry eye, dry mouth, and arthritis. Other systemic conditions that may result in aqueous-deficient dry eye include lupus erythematous and ocular rosacea. In addition to systemic conditions, other causes may include drugs such as antidepressants, beta blockers, diuretics, oral contraceptives, and topical beta-blockers used to treat glaucoma. Individuals likely to be affected include: postmenopausal women, patients with *Helicobacter pylori*, older people, computer users, and long-term contact lens wearers.[63-65]

True mucin deficiency is rare; one report estimates the prevalence of OCP to be 1 in 20,000 persons.[66] Cicatricial pemphigoid is the most common of the immunobullous disorders causing conjunctival cicatrization secondary to destruction of goblet cells. The disease is usually bilateral and more common in females, with most cases occurring between 30 and 90 years of age, but most frequently in the seventh decade of life.[67] Loss of goblet cells occurs as a complication of inflammatory injuries to the conjunctiva or OCP. It is also a possible side effect of prolonged topical cholinergic and anticholinesterase administration used in the treatment of glaucoma.[68-72] This medically

induced complication is rarely seen since the introduction of contemporary glaucoma treatment options.

Most problems involving lipid layer instability are related to glandular dysfunctions that produce thickened meibum, leading to accelerated surface evaporation. This complication leads to an unstable or dysfunctional tear film. Therefore, there is a close association of various forms of meibomianitis especially with posterior blepharitis. Lipid layer abnormalities resulting from complete absence of meibomian gland secretion are rare.[14] Meibomian gland deficiencies have been evaluated by eyelid transillumination and classified as atrophic or dysfunctional (rosacea) among patients with symptoms consistent with ocular irritation. This form of glandular dysfunction has now been recognized as posterior blepharitis.[42,73]

b. Risk Factors

Among the common risk factors for dry eye are advancing age, the presence of rheumatoid arthritis, Graves' disease, the use of drugs that decrease aqueous or mucous membrane secretions, eyelid or blinking abnormalities, and a history of trauma to the lids.[70-72] Environmental and post-refractive surgery can also be causes of dry eye.[74-78]

2. Blepharitis

a. Prevalence

Epidemiologic characteristics of blepharitis vary, depending on the type. Types of blepharitis range from acute to chronic disorders, with inflammation affecting the anterior or posterior lid margins, along with involvement of both skin and mucous membranes.

The prevalence of dry eye and blepharitis is unknown. The DEWS group has compiled a report devoted to the prevalence of dry eye. [2] The group concluded that between 5 and 35 percent of patients, depending on age, geographic location, definition used in the study, and episodic contributing factors may exhibit dry eye (including blepharitis) signs or symptoms.

Most staphylococcal blepharitis occurs in younger women (mean age, 42 years),[79,80] whereas the seborrheic variations tend to occur in older individuals. Rosacea, a disease of unknown prevalence, is more common in fair-skinned persons between the ages of 30 and 50, especially women.[82] Gross ocular lesions occur in many cases of rosacea, and almost all affected persons eventually develop recurrent or chronic blepharitis and meibomianitis. There is a strong association between KCS and staphylococcal blepharitis.[82]

b. Risk Factors

Underlying dermatologic conditions may represent risk factors for blepharitis. Seborrheic blepharitis is associated with seborrheic dermatitis. Meibomianitis occurs approximately twice as frequently with rosacea as it does with seborrheic dermatitis.[37] Patients with atopic dermatitis and psoriasis may also have a blepharitis as a complication. Patients with SS-related KCS appear more likely to develop meibomian gland disease.[13,73,83]

C. Clinical Background of Ocular Surface Disorders

The ocular surface requires a regular resurfacing of tears to provide comfort and clear vision. The production of sufficient lacrimal fluid of normal composition and its distribution by regular blinking are essential to ocular surface integrity and comfort. Any decrease or alteration in the production of any component of the tear film, especially the lipid layer, or interference with the resurfacing process can impair any of the functions of the POTF.

1. Dry Eye

a. Natural History

In the earliest stages of dry eye, an insufficient or unstable tear film may produce infrequent and insignificant symptoms. Early signs or mild symptoms may be secondary to hyperosmolarity of the tear film and be the cause or result of inflammation.[3,4] Some symptoms may occur only under conditions of stress. These conditions may include, but are not

limited to, low humidity, smoky environment, recirculated air environment, and prolonged computer use.[28,74,77,84-87] As the condition progresses, the eye cannot maintain the volume of moisture required and the symptoms become more common and more bothersome. "Paradoxical epiphora" (hypersecretion) from irritation-induced reflex tearing may be the presenting symptom.

In severe DE conditions, symptoms of burning and visual interference can be debilitating.[88] The cornea appears dull, the conjunctiva and lid margins may be hyperemic and edematous, and superficial punctate staining may be present. Filamentary keratitis, a painful corneal response characterized by strands of partially desquamated epithelial cells, can result from corneal desiccation and accumulation of stagnant mucin and shed epithelial cells. In addition to the lid infections commonly associated with dry eye, the patient with DE has a higher likelihood of having conjunctivitis and keratitis. Therefore, moderate or severe dry eye may adversely affect the quality of life.

b. Signs, Symptoms, and Complications

In mild cases of DE, symptoms of scratchiness, burning, or stinging may be accompanied by mild and/or transient situational blurring of vision when the tear film is disrupted. In moderate cases, ocular discomfort becomes marked and visual acuity may be reduced. As the dry eye becomes more severe, observable signs may include rapid tear film breakup, debris in the tear film, a minimal lower lid tear meniscus, increased mucous threads in the tear film, corneal and conjunctival staining, filamentary keratitis, and loss of corneal luster.

Instability of the tear film can initiate ocular surface complications.[89] Decreased aqueous volume is associated with reduced ocular surface defense and increased susceptibility to irritation, allergy, and infection due to tear stagnation and epithelial compromise.[90-93] A major consequence of reduced aqueous volume is reduced antibacterial function because of decreased lactoferrin and lysozyme levels.[94-96] In addition, staphylococcal organisms can produce toxins that can cause superficial punctate keratopathy.[97]

Seborrheic blepharitis can cause an inferior staining pattern from an alteration of the lid-tear interface, perhaps because of lost tear retention, decreased tear volume, and intrapalpebral desiccation.[98,99] Persistent dry spots, a more significant consequence of an unstable tear film, may be associated with either abnormalities of the tear distribution system or reduced tear flow.

Squamous metaplasia of the conjunctiva occurs secondary to changes in the ocular surface, perhaps as a result of environmental exposure.[100] Impression cytology studies suggest abnormal conjunctival epithelium as well as changes in the goblet cells.[101,102] Two possible etiologies have been proposed: (1) loss or reduction of conjunctival vascularization, which prevents normal epithelial differentiation, and (2) inflammatory changes that induce epithelial alteration. Squamous changes have also been reported in mucin- and aqueous-deficient conditions.[52]

c. Early Detection and Prevention

Factors beyond the patient's control cause some forms of DE. However, appropriate action can help to delay the onset or minimize the degree of symptoms for a large portion of the affected population. The use of tear supplements may make symptoms tolerable in milder situations. Specifically, nonpreserved tear supplements also play a role in the relief of moderate and advanced cases. Lid hygiene, and when appropriate antibiotic intervention for anti-inflammatory effects, minimizes the effects of altered lipid secretion and reduces the possibility of secondary infection. Prompt diagnosis and management of any change in the appearance or comfort of the eye can also limit the occurrence of complications.

2. Blepharitis

a. Natural History

Chronic blepharitis with secondary ocular surface manifestations is not an isolated problem. Rather, it is one of a group of disorders resulting from disruption of the complex and delicate balance among the eyelids, tear film, and ocular surface. The eyelids are vital to the health of the

ocular surface because of their protective function and their contribution to the production and dispersal of the tear film. The milder forms of blepharitis often are annoying because of mild crusting and irritation of the lid margins. Moderate and severe forms are associated with bacterial infections and chronic meibomian gland changes. Not only can they be painful and cosmetically unappealing, but they also cause instability of the POTF and become the source of related problems.

b. Signs, Symptoms, and Complications

The spectrum of visible signs of blepharitis varies with the degree of inflammation. In mild cases of seborrheic blepharitis, biomicroscopic examination may be necessary to view the scales on or at the base of the eyelashes. Additional inflammatory forms of the condition produce more noticeable signs. In severe meibomianitis, the meibomian glands are clogged and the tear film is deficient in normal lipids.

Severity of the symptoms may also be related to the degree of inflammation. In its milder forms, seborrheic blepharitis may have no associated symptoms. Inflammation of the eyelid margin and skin can produce various levels of irritation and ocular discomfort. Associated tear film disorders, such as lipid deficiency and excessive tear film debris, can disrupt the stability of the POTF and affect vision.

Complications may occur during the acute phase of blepharitis or in response to inadequate management of the chronic form of the disease. Accumulated secretions may produce localized reactions and support the growth of other organisms. The most common complication of blepharitis is alteration of the POTF with consequent signs and symptoms. In severe forms of blepharitis, secondary conjunctival, corneal, and eyelid-margin inflammation may occur.

c. Early Detection and Prevention

Currently, no prophylactic measures exist to control the consequences of blepharitis. Treatments are aimed at reversing the severity of the inflammation. Lid hygiene, consisting of warm compresses and lid scrubs, is the basis for treating all forms of blepharitis. In addition,

associated conditions, such as seborrhea, staphylococcal involvement, and rosacea, should be treated. These conditions may require topical or oral antibiotics. In the event of exacerbation, early recognition, diagnosis, and treatment can help minimize the degree of inflammation and potential for infection. Moreover, clinical recognition of posterior blepharitis as a complication of malsecretion of lipids by the meibomian glands suggests the need for early intervention.

[redacted]

[redacted]

II. CARE PROCESS

This Guideline describes optometric care provided to a patient with ocular surface disorders. The components of patient care described are not intended to be comprehensive, because professional judgment and the individual patient's symptoms and findings may have a significant impact on the nature, extent, and course of the services provided.

A. Diagnosis of Ocular Surface Disorders

Patients with compromised ocular surfaces have greater potential for discomfort or further ocular damage. Early recognition of the signs of infection and prompt diagnosis minimize the potential for severe or chronic complications. Evaluation of a patient exhibiting dry eye symptoms or signs consistent with blepharitis includes many of the elements of a comprehensive eye and vision examination* and a more in-depth evaluation of the ocular surface and adnexa. The evaluation for ocular surface disorders includes a carefully detailed patient history, assessment of associated risk factors, and examination of the anterior ocular structures and their functions.

1. Patient History

Demographic data about the patient should be collected prior to taking the patient history. Included in the patient history are the chief complaint, history of the present illness or condition, ocular history, general health history (which may include a social history and an extended review of systems), and family ocular and medical history. In addition, environmental factors relating to climate, season, vocational setting, and avocational pursuits should be reviewed.

The patient's history and symptoms are effective diagnostic tools in identifying the presence of tear film insufficiency. The history should document associated conditions that make an individual more likely to develop tear film abnormalities. Common ocular complaints include

*Refer to the Optometric Clinical Practice Guideline on Comprehensive Adult Eye and Vision Examination.

burning or stinging, itching, scratchiness, irritation, tearing, increased mucus and reduced contact lens tolerance. A symptom index specific to ocular surface disorders has been proposed and validated (Appendix Figure 5).[103,104]

Patients with Sjögren's syndrome may give paradoxical reports of discomfort with certain instruments.[105] Owing to the visible nature of some forms of anterior blepharitis, the patient can usually describe the onset and course of the condition. Acute-onset inflammation of relatively short duration often responds to treatment better than the chronic long-term forms of the disease. A thorough medical history helps identify any underlying systemic cause. The effects of previous treatments and the patient's compliance in following recommendations may be good indicators of the prognosis of new treatment plans.

2. Ocular Examination for Ocular Surface Disorders

Observations, using external ocular examination techniques, both without magnification and with the biomicroscope, show characteristic early changes of the external eye. Evaluation for suspected ocular surface disorders may include, but is not limited to, the following:

- External view of the eye, noting lid structure, position, symmetry, and blink dynamics
- Biomicroscopic examination of the lid margins, meibomian gland orifices, and their contents
- Biomicroscopic examination of the tear film, noting mucus, debris, interference patterns in the lipid layer, and tear meniscus height
- Biomicroscopic examination of the cornea and conjunctiva, both with and without sodium fluorescein (SF) and lissamine green (LG) staining. Rose bengal (RB) has been replaced by LG, because there is less discomfort or sting associated with its use.

With moderate manifestations of ocular surface disorders, there may be obvious changes in tear film stability (as manifested by inconsistent but reduced breakup time), subtle or transient corneal superficial punctate keratopathy, or more apparent conjunctival staining. In more severe cases, tear film debris may be accompanied by corneal mucus strands,

filaments, furrows, dellen, staining, or erosion, all of which contribute to an overall lack of luster. The cornea may become thickened or show thinning in areas of dellen. The conjunctiva may be hyperemic and have folds in the exposed bulbar portion[106]; this is typically observed in the lower temporal area, where the eyelid meets the globe. The POTF may have increased viscosity, debris and a foamy secretion that spills onto the eyelids, as well as a scanty, inferior tear prism. The lids often have thickened margins, crusting, and madarosis. The more severe the tear film deficiency, the more pronounced the signs will appear.

Tear quantity tests. Tear quantity tests are useful in confirming the diagnosis of aqueous-deficient dry eyes. The most frequently utilized procedures are:

- **Schirmer tear test.** The Schirmer test, either with topical anesthesia (basic secretion test) or without (Schirmer I), can be used to evaluate the quantity of the aqueous layer of the tear film.[10] In this test, the examiner places filter paper in the lower fornix to measure the volume of tears produced during a fixed time period. When performed using a topical anesthetic, it purportedly measures the tear secretion of the accessory lacrimal glands; without anesthetic, it measures the tear production of the lacrimal gland by stimulation of the lacrimal reflex arc. Although it is controversial because the results are often inconsistent, the Schirmer tear test can provide useful clinical information.

- **Fluorescein-enhanced assessment.** After adding fluorescein, a water-soluble, inert dye (*not* fluorescein-anesthetic solution) to the ocular surface, the clinician can observe the rate of dilution of the aqueous component of the POTF, especially with enhancement by cobalt-filtered illumination. In addition, subclinical disruption of the ocular surface will be revealed by staining viewed with the cobalt-filtered illumination. Acceptance of this method has been hampered by lack of a standard.[10,103]

- **Evaluation of the tear prism.** The tear meniscus height can be assessed with biomicroscopic examination both with and without instilling fluorescein dye.[107] A tear meniscus height greater than

0.2 millimeters (mm) should be considered normal.[108] A scanty or absent tear meniscus is an indication of an aqueous tear deficiency.[109] Future directions in tear meniscometry may combine the use of interference patterns.[110]

- **Tear-film debris.** Excessive particulate matter in the tear film, visible by biomicroscopic examination, may indicate inadequate flushing action due to reduced tear flow.

- **Rose bengal/ lissamine green staining.** A useful test for identification of ocular surface disorders has been rose bengal staining. It highlights ocular surface changes associated with insufficient tear flow and conjunctival and corneal desiccation. One scoring system for rose bengal staining assigns values of 0 to 3 for each of the lateral and medial corneal and conjunctival regions of the exposed intrapalpebral ocular surface.[111] A maximum score of 9 indicates severe staining; 0 indicates complete absence of rose bengal staining. A more detailed technique for quantitative assessment of rose bengal staining enables description of the intensity and extent of involvement and may be more useful in documenting subtle changes in response to treatment strategies.[111]

  The introduction of lissamine green stain has offered an alternative to rose bengal that is less irritating to the patient and equally efficacious in demonstrating disrupted ocular surface characteristics.[112,113] Therefore, lissamine green is preferable to rose bengal. The Oxford scale has been proposed to standardize the extent and location of lissamine green as well as fluorescein staining.[114]

Other tests that may be used to evaluate tear quantity are:

– Schirmer II (irritation)
– Cotton thread test[114]
– Lissamine green staining[115,116]
– Phenol red thread test [117]

– Tear volume measurements
– Fluorophotometry; fluorescein dilution[118]
– Lacrimal equilibration time[119]
– Temporary punctal occlusion.

Tear film stability tests. Several procedures are commonly used to evaluate tear film stability:

- **Tear film breakup time (TBUT).** The time required for the tear film to break up following a blink is normally 15–20 seconds[120]; TBUT values of less than 10 seconds may represent a practical index for an abnormal tear film. Some optometrists rely on an empirical test of the integrity of the tear film being maintained within the blink interval. The most recent suggestion is that values between 5 and 10 seconds are thresholds but volume-dependent.[7,121] Because lipid contamination of the mucin layer decreases the surface tension and eliminates the aqueous portion of the tear film in that area, reduced BUT may also indicate mucin deficiency. Some clinicians prefer to measure the noninvasive BUT (NIBUT). Tear-film breakup is observed without the addition of fluorescein to the tear film.

- **Tear-thinning time.** This noninvasive test involves a keratometer to view the mire image and measure the time from a complete blink to distortion of the image.[122]

Other tests that may be used to evaluate the quality of the POTF are:

– Tear osmolarity test[84,123,124]
– Impression cytology[125]
– Conjunctival scraping and biopsy
– Tear protein analysis[126]
– Mucin assay test (tear ferning)[127]
– Lipid layer interference patterns[16,107,128]
– Specular reflection of the tear surface[129,130]
– ELISA tear protein profile.[131]

After nearly a century of research attempting to characterize clinical signs among patients with dry eye, the consensus is that tear film dysfunctions are secondary to lid and lid-gland disruptions. Such disruption leads to, or is a consequence of, osmolarity changes in the aqueous layer of the tear film; it may lead to, or be a consequence of, inflammatory components in the tear film and on the ocular surface. Unfortunately, no single tear quantity or tear quality test is capable of assessing the integrity of the tear film or ocular surface. Diagnosis is more likely to be accurate when it is based on multiple abnormal test results.[1,7,10,102] Ocular surface disorders, whether caused by aqueous, mucus, or lipid deficiencies or abnormalities, must be diagnosed and treated as early as possible to prevent further changes in the exposed ocular surface. Table 1 summarizes normal values that have been established for selected tests.

3. Ocular Examination for Blepharitis

A thorough external examination of the lids and other parts of the adnexa, including comparison of the eyes, helps determine the severity of inflammation. Differentiating among the various presentations of blepharitis requires the use of a biomicroscope to contrast the appearance of the anterior and the posterior lid margins. Evaluation of the patient with blepharitis may include, but is not limited to the following:

- External examination of the eye, including lid structure, skin texture, and eyelash appearance, and evaluation for clinical signs of rosacea (i.e., telangiectasia, pustules, rhinophyma).

- Biomicroscopic examination of the lid margins, the base of the lashes, and the meibomian gland orifices and their contents. Telangiectasia posterior to the meibomian glands may be a key finding in identifying posterior blepharitis secondary to meibomian gland dysfunction.

- Examination of the tear film for lipid layer abnormalities.

- Evaluation of the palpebral and bulbar conjunctiva.

Table 1

Tear Function Tests and Normal Values

Test	Significance	Normal Values
Tear meniscus	Aqueous quantity	Range: 0.1 - 0.6 mm
Schirmer I	No diagnostic value	>15 mm in 5 min
Schirmer basic secretion test	Aqueous deficiency when reduced (accessory lacrimal gland dysfunction)	>5 mm in 5 min
Lactoferrin	Lacrimal gland function	1.42 mg/mL (<1.00 mg/mL is abnormal)
Tear osmolarity	Lacrimal gland function	<312 mOsm/L
Breakup time (BUT)	Tear film stability/mucus deficiency	>10 sec
Noninvasive breakup time (NIBUT)	Microepithelial defects/aqueous adequacy	40 sec
Fluorescein	Microepithelial defects/mucus deficiency	No staining visible
Rose bengal/ lissamine green	Non-mucus-coated epithelium	No staining visible
Impression cytology	Epithelial cell appearance/goblet cell density	Normal microscopic appearance
Interference fringe pattern	Lipid layer integrity	Uniform biomicroscopic appearance
Meibomian gland expression	Meibomian gland function	Clear
Lacrimal gland function	Total lysozyme reactivity (TLR)	<1.0

Each type of anterior blepharitis has specific characteristics that help in making the appropriate diagnosis:

- **Staphylococcal blepharitis.** In the early stages, the symptoms are a foreign body sensation, irritation, itching, and mild sticking together of the lids. If the condition becomes chronic, thickened lid margins, trichiasis, lid-margin notching, madarosis, ectropion, or entropion may result. The lower third of the cornea may have punctate staining, erosions, and infiltrates from exotoxins or a disrupted POTF. An associated bacterial conjunctivitis may develop.

- **Seborrheic blepharitis.** The symptoms may include burning, stinging, itching, and ocular irritation or discomfort. The lids may appear hyperemic at the anterior margin, with the hallmark appearance of scales on the lashes. This condition is usually chronic, but there may be periods of exacerbation and remission. Although there is very little inflammation of the lid margin, KCS may be a secondary presentation and may exacerbate tear film instability.

- **Seborrheic/staphylococcal blepharitis.** There are frequent exacerbations of a mild to moderate inflammatory reaction.

- **Meibomian seborrheic blepharitis.** In this condition associated with seborrheic dermatitis, meibomian openings are dilated. A distinguishing clinical feature is increased meibum, which causes a foamy tear film along the lid margins, especially at the lateral canthus. This observation is characteristic of staphylococcal colonization of the lid margin, as well.[133] The bulbar conjunctiva is injected, and there may be concurrent KCS.

- **Seborrheic blepharitis with secondary meibomianitis.** This chronic condition, with exacerbations, also includes sporadically blocked and inflamed meibomian glands. This situation potentiates an unstable tear film and dry eye symptoms.

- **Meibomian keratoconjunctivitis.** As part of a generalized sebaceous gland dysfunction, meibomian keratoconjunctivitis is frequently associated with rosacea. The gland openings are obstructed by desquamated epithelial cells, resulting in a poor POTF that can be identified by lissamine green or rose bengal staining. The meibomian secretions have a higher melting point than the ocular surface temperature, which results in reduced sebum secretion and plugs of free fatty acids at the gland openings that are often inflamed and pouted. The tear film is very unstable. This constellation of signs probably signals meibomian gland dysfunction and would be consistent with the Delphi panel's description of dysfunctional tear syndrome.[1]

- **Angular blepharitis.** The two appearances of angular blepharitis are the dry, scaly form caused by *Staphylococcus* and the wet, macerated type caused by *Moraxella*.

- **Demodicosis.** *Demodex* are present in the lash follicles of most elderly persons.[133] This condition is usually innocuous. When the mite population reaches critical proportions, symptoms result. There is a crusting of the lid margin, trichiasis, madarosis, loss of lashes, and cuffing at the base of the lashes. The diagnosis can be confirmed by epilating a lash from the affected area and examining the follicle under a clinical microscope for the presence of mites. Patients with rosacea may be more prone to *D. folliculorum* than those without this diagnosis.

B. Management of Ocular Surface Disorders

Treatment and management strategies for ocular surface disorders can vary and may require consultation with or referral to the patient's primary care physician, a dermatologist, a corneal specialist, or other health care provider, as appropriate. Appendix Figure 3 presents a classification for treating and managing patients with ocular surface disorders.

I. General Considerations

A comprehensive approach to treating and managing eyelid, tear film, and conjunctival or corneal abnormalities is important. Periodic re-evaluation is needed because a primary dysfunction of any one of these components often affects the others. The approach to managing a patient with blepharitis is dependent upon identification of the type and severity of the condition so that the appropriate therapy may be instituted. Anterior forms of blepharitis generally have a greater impact on the skin of the eyelid than on the ocular surface. Posterior blepharitis has a greater potential to produce dry eye symptoms and signs.[42,72,124,135] When evaluating patients for ocular surface disorders, the clinician must pay special attention to the lid margins and the preocular tear film.

Contact lens wear may pose a risk to the compromised ocular surface. In addition, success with contact lens wear may be attenuated by complications of tear film deficiency. Conversely, contact lenses may play a role in the management of selected disorders of the tear film and ocular surface. Identifying and treating conditions prior to fitting contact lenses and managing potential problems aggressively are prerequisites for successfully wearing contact lenses. Recommendations for successful contact lens wear include a TBUT greater than 10 seconds. Mild or moderate cases of tear deficiency often can be managed with tear supplementation or by tear conservation. More severe cases of tear deficiency are less likely to be associated with successful contact lens wear; the clinical presence of rosacea, due to lid-gland dysfunction may complicate contact lens wear.

The strategy to help ensure successful contact lens wear by patients with compromised ocular surfaces also requires a comprehensive approach to contact lens fitting.* This strategy includes:

*Refer to the Optometric Clinical Practice Guideline on Care of the Contact Lens Patient for additional information.

- Determining lens diameters, thicknesses, and edge designs that will achieve adequate lens/cornea relationships and minimize blink inhibition

- Recommending appropriate wearing schedules, such as mid-day removal of lenses with rehydration of hydrogel lenses

- Selecting materials with both water content and surface characteristics to match the patient's condition (in the case of hydrogel lenses)

- Considering a more compatible lens material such as silicone-hydrogel polymer

- In all cases, considering tear supplementation for patients whose ocular surface becomes compromised as a result of contact lens wear.

Although tear film deficiencies may complicate or contraindicate contact lens wear, contact lenses may have a role in the management of certain forms of dry eye. Applying a hydrogel or silicone-hydrogel lens to a dry eye can provide a stable, moist environment for desiccated epithelium. Nevertheless, there are associated risks, including surface deposits, increased inflammation, and infection.

2. Treatment and Management of Dry Eye

a. Basis for Treatment

Stepwise determination of the minimum intervention required to achieve results will help ensure a balance of patient compliance, long-term success, and cost effectiveness. The management of dry eye is designed to reduce symptoms and inflammation and to re-establish a normal ocular surface. Efforts should be aimed at maintaining or restoring the POTF and ridding the lids of potential sources of tear film destabilization. Whenever possible, environmental factors contributing to dry eye should be identified and either modified or eliminated. When associated medical conditions are identified, consultation with or referral

to the patient's primary care physician or other health care provider may be indicated.

b. Available Treatment Options

Attempts to relieve dry eye symptoms and re-establish a normal ocular surface have produced a myriad of possible remedies. Traditional approaches include both tear supplementation and tear conservation measures. Several alternatives have been used with varying degrees of clinical success:

Ocular (lid) hygiene. Daily cleaning of accumulated debris from the lid margins removes a potential culture medium for microorganisms. Normal face washing, with attention to the ocular adnexa, is sufficient for most people; however, commercial lid scrubs are available. Regular use of warm compresses is often helpful to individuals whose dry eye condition is exacerbated by the inspissation of meibomian secretion. This strategy is most useful in posterior blepharitis with a positive effect on the meibomian glands.

Topical treatment. A number of pharmaceutical preparations* have gained acceptance as temporary substitutes for the tear layer. These include tear supplements, ointments, and soluble polymeric inserts. The efficacy of commercially available products has been documented.[136,137] In addition, dissolvable and silicone removable plugs have been used to retain or conserve tears by retarding drainage. Two studies have reported the efficacy of apparently safer dissolvable collagen materials over the removable silicone application.[138,139]

A large, placebo-controlled study found that the immunomodulator, cyclosporine, can both ameliorate symptoms and reduce the clinical signs

*Every effort has been made to ensure that drug dosage recommendations are accurate at the time of publication of the Guideline. However, as treatment recommendations change due to continuing research and clinical experience, clinicians should verify drug dosage schedules on product information sheets, especially when drugs are new or infrequently used.

of dry eye.[140] Cyclosporine ophthalmic solution 0.05% has been approved by the U.S. Food and Drug Administration (FDA) for the treatment of KCS. Its inflammation-reducing potential is particularly beneficial in patients with Sjögren's syndrome.[141-143]

Evidence has suggested that topical cyclosporine in combination with punctal occlusion may have a favorable synergistic effect.[144] Since inflammation has been identified as a significant component of ocular surface disorders, it may seem logical that topical anti-inflammatory treatments other than cyclosporine are effective. One study investigated the comparative efficacy of topical steroids and non-steroidal anti-inflammatory drugs (NSAID) compared with tear supplementation.[145] Symptoms were reduced and clinical staining scores improved among the topical steroid treated group but not the NSAID group. This evidence may serve as the basis for using topical steroid drops to limit the inflammatory response for short-term improvement during dry-eye therapy.

In a 3-month prospective study, topical 0.05% cyclosporine performed better for clinical signs and symptoms than did a combination of topical 0.3% tobramycin plus 0.1% dexamethasone for the treatment of posterior blepharitis.[146] The potential side effect of increased intraocular pressure (IOP) with long-term steroid use warrants a baseline IOP reading, with appropriate followup readings.

Tear supplements can be designed to mimic the tonicity, pH, retention time, mucomimetic properties, and lubricating features of the POTF, and to increase the height of the tear meniscus. Available in a variety of formulations, tear supplements have active ingredients representing a wide spectrum of polymeric components (Table 2). FDA requires that all multidose ophthalmic solutions be preserved against contamination from a standard group of pathogens. With chronic use, however, these preservatives may cause adverse effects, including reduction of the desired effect, allergic response, and toxic reaction. Unpreserved unit-dose containers prevent the preservative problem but are more costly. Tear supplements also use preservatives that, when instilled onto the ocular surface, rapidly break down into innocuous compounds. These so-called transiently preserved solutions offer economy of volume and

freedom from the adverse effects of preservatives. Ophthalmic preservatives used in artificial tear solutions and their potential adverse effects include:

– *Thimerosal™.* A hypersensitivity reaction occurs in an estimated 10–25 percent of users.[147] Thimerosal may be used in ophthalmic ointment preparations that are available without prescription (i.e., over the counter).

– *Benzalkonium chloride*. POTF instability, lowered BUT, and disrupted corneal epithelial cell functions occur when benzalkonium chloride is dosed at commercial concentrations. These effects seem to be concentration and chronicity dependent.[148-151] The adverse effect on the ocular surface may be due to alteration of epithelial mucin.[152]

– *Chlorobutanol.* Evaporation and corneal epithelial cell changes occur.[153]

– *Ethylenediaminetetraacetic acid (EDTA).* Adverse effect is contact allergy.[154,155] Storage in the corneal and conjunctival epithelium results in keratitis.[149,156]

Effective management of dry eye may require the instillation of tear supplements as frequently as 1 drop every 30 minutes or as infrequently as 1 drop daily at bedtime. For the patient who requires dosing more than 3 times a day, preservative-free or transiently preserved tears should be recommended. Only evaluation and continual monitoring can establish the frequency and duration of treatment needed. In mild cases, the simple recommendation of repeated blinking may contribute to relief. Table 2 lists the ingredients of contemporary tear supplements.

- **Ointments.** When placed in the lower cul-de-sac, ointments containing emollients dissolve at body temperature and disperse in the tears, providing lubrication and protection. Petrolatum, mineral oil, and lanolin are typically included in the formulation of ointments designed for retention of ocular moisture. Usually used

Table 2
Components in Tear Supplements, Drop or Gel Form

Cellulose ethers	Hydroxypropyl cellulose
	Hydroxypropyl methylcellulose (HPMC) (found in many preparations)
	Methylcellulose hydroxyethyl cellulose
	Carboxymethyl cellulose (CMC) (found in many preparations)
	Hypermellose (found in many preparations)
Polyvinyl polymers	Polyvinyl alcohol (PVA) (found in many preparations)
	Polyvinyl pyrrolidone (PVP) (found in many preparations)
Mucolytic agents	N-acetylcysteine[157]*
Hyposmotic tear supplements	Glycerin, dextran
Vitamin A	Retinol
Others	Mineral oil

*A mucolytic agent that disrupts formation of filaments and may increase TBUT by altering lipid secretions.

at bedtime, ointments may also be used by sedentary patients during the daytime. Because of ointments' viscosity, they can blur vision; thus, a very small amount of ointment may be sufficient for daytime use. Patients allergic to wool may react adversely to lanolin. Preservative-free formulations should be recommended for patients who use these products chronically.

- **Punctal occlusion.** When surface treatments do not relieve symptoms, preocular moisture can be retained by blocking the outflow of tears to the nasolacrimal system.[1] This blockage can be accomplished by dissolvable, removable, or permanent punctual

occlusion. The clinical efficacy of silicone punctual plugs may be limited in both duration (<2 years) and rate of retention (~50%).[158] Complications include, but are not limited to, total extrusion epiphora, partial extrusion, subconjunctival hemorrhage, conjunctival erosion, and fragmentation of the plug.[158] Additional advantages and limitations of punctal occlusion have been chronicled as well.[159-163] Lacrimal plugs made of silicone or a thermodynamic acrylic polymer appear to be safe and effective. Each patient should be followed on a long-term basis to exclude chronic inflammatory reactions, extrusion, or migration, all of which may lead to discomfort.[164] Therefore, recommendation to patients should be made on a case-by-case basis after careful selection. Table 3 lists contemporary means of punctal occlusion.

Table # 3
Means of Punctal Occlusion

Collagen implants—dissolvable plugs that provide preliminary results as to the potential effectiveness of using more permanent means of punctal or canalicular obstruction to reduce tear loss through drainage.
Tapered shaft silicone punctal plugs[165,166]
Cylindrical shaft silicone punctal plugs
Intracanalicular implants
Thermo-sensitive punctal plugs[167]
Thermal cautery and other forms of permanent occlusion—may be indicated when the patient's predisposing condition is permanent
Electrodesiccation using an electrocautery unit—permanently scars the punctum and canaliculus
Laser punctal occlusion[168] (punctoplasty) using the argon laser—less efficacious than thermal or electric cautery
Surgical repositioning of the punctum[169] anteriorly out of the lacrimal tear meniscus—minimizes tear outflow and allows for future surgical adjustments, if necessary

Alternative methods for relieving symptoms specific to ocular disorders include:

- **Hydrophilic bandage lenses and collagen corneal shields.**[170,171] Used with sporadic success, they may have particular application to filamentary keratitis following debridement or when mucus strands are present.[172,173]

- **Moisture chamber goggles.**[174] As a means of reducing evaporation, side and top shields are commercially available to modify a patient's glasses. Swimming goggles accomplish the same goal.

- **Tarsorrhaphy.** Surgical closure of the lids is reserved for cases of severe, unresponsive disease. Initially the lateral third of the palpebral fissure is sutured shut. When this measure is insufficient, complete tarsorrhaphy is performed.[175]

- **Review of medications.** A review of medications should be conducted to identify and eliminate potential drug-related causes of dry eye. Estrogen replacement therapy may be beneficial in patients with KCS[176] In addition, systemic testosterone in combination with esterified estrogen may show similar benefits.[177] Conversely, for postmenopausal women using estrogen alone or in combination with progesterone/progestin, the risk for clinically diagnosed dry eye syndrome or severe symptoms rises by up to 15 percent for each 3 years on hormone replacement therapy (HRT).[47] One study has shown that this specific hormonal imbalance may benefit from omega-3 long-chain polyunsaturated fatty acid supplementation.[178]

- **Salivary gland transplant.**[179] The placement of salivary gland tissue in the conjunctiva has been attempted as a means of producing preocular secretion.[180] Autologous submandibular gland transplantation to the temporal fossa has also been suggested.[181]

[redacted]

- **Limbal grafts**. Proposed for severe cases of ocular surface disease, limbal grafts remain experimental, while guidelines for their implementation evolve.[182,183] This may be a particularly important area for the future treatment of ocular surface disorders related to limbal stem cell deficiency, an emerging diagnostic category.[184]

  Limbal stem cell deficiency is now recognized as a diagnostic category. Treatment involves transplantation of harvested or transplanted limbal cells to the ocular surface by a variety of vehicles.[185-187]

- **Autologous blood serum and other nutrient drops.** Topical application of drops of serum from the patient's own blood, which are unpreserved and non-antigenic in nature, is a means of providing growth factors, fibronectin, immunoglobulins, and vitamins at similar (or higher) concentrations than exist in tears. These are applied in cases of severe dry eye with punctate epithelial defects and corneal damage to promote re-epithelialization.[27] No commercial product is yet available, nor has the FDA approved this treatment. However, the emergence of hyaluronate-based topical drops may offer still another treatment option, because both their nutrient capabilities and their lubricating qualities have favorable treatment attributes.[188-190]

3. Treatment and Management of Anterior Blepharitis

a. Basis for Treatment

Anterior blepharitis usually is the direct result of disruption or infection of the lipid-producing glands that open to the lid margin. Clinical presentation may include internal and external hordeola. The treatment is relatively straightforward. Though essential, lid hygiene alone may not resolve the problem. Depending upon the clinical findings, appropriate anti-infective drugs can be administered topically, systemically, or in combination. Aggressive therapy should initially include a minimum of 6 weeks of lid hygiene and appropriate anti-

[redacted]

infective medications to gain control of the condition, followed by maintenance therapy.

b. Available Treatment Options

Because every category of anterior blepharitis is actually a separate condition, each needs to be addressed individually. However, the Delphi report identified anterior blepharitis as an inclusive category in patients with dysfunctional tear syndrome and recommended lid hygiene and topical antibiotic treatment initially. For patients without lid margin disease, the initial treatment consists of topical tear supplements and immunomodulators.[1] Failure to respond should prompt pursuit of signs of posterior blepharitis.

Staphylococcal blepharitis. Treatment of staphylococcal blepharitis includes an antibiotic ointment to control the infection as well as lid hygiene.[191,192] Lid hygiene can be performed with a commercially available lid scrub formulation or by using dilute baby shampoo (1:10 in water) applied with a facial cloth. Erythromycin, bacitracin, polymyxin B-bacitracin, gentamicin, and tobramycin are all effective antibiotics for treatment of staphylococcal blepharitis. Each of these is available in ointment form. Another ointment that may have application to these situations is tacrolimus, which the FDA has approved for eczema.[193] Antibiotic eye drops can be used, but they do not work as well as ointments, due to reduced contact time. Tear supplements may also be required to alleviate symptoms. If peripheral corneal infiltrates are present without epithelial defects, topical steroids may be used for a limited time.

Seborrheic blepharitis. In the treatment of seborrheic blepharitis, the application of warm, moist compresses to soften and loosen the crusts is followed by washing with a commercial lid scrub or dilute baby shampoo (1:10 in water) on a facial cloth or cotton swab, taking care not to involve the globe. The scalp and eyebrows should be washed with a selenium anti-dandruff shampoo.[194] The emphasis for treatment of seborrheic blepharitis has shifted to include oral antibiotics, especially minocycline.[195-197] The purpose of using minocycline is to alter the polarity of the meibomian secretion composition.[198]

Seborrheic/staphylococcal blepharitis. The use of appropriate ophthalmic antibiotic ointments is required. Later, when the lid is more comfortable, warm compresses and lid scrubs can be added. Warm compresses and lid washing are the same as for seborrheic blepharitis. Though serving as an acceptable means of control, this treatment rarely effects a cure for seborrheic/staphylococcal blepharitis.

Meibomian seborrheic blepharitis. The treatment includes the same warm compress and lid hygiene regimen as for seborrheic blepharitis. In addition, the meibomian glands may be massaged or expressed to remove the plugs at the openings. Antibiotic or antibiotic/steroid ointments may be added when the infection has been identified clinically.[135,199]

Seborrheic blepharitis with secondary meibomianitis. Treatment begins with lid hygiene. Antibiotic or antibiotic/steroid therapy may be added when a clinical infection has been identified. Resistant cases of seborrheic blepharitis with secondary meibomianitis may require systemic tetracycline (up to 1g/day) or doxycycline (100 mg/day) for at least 6 weeks.[200,201] It is not unusual for patients who have this condition to require lower maintenance doses after tapering. Neither tetracycline nor its derivatives should be given to children under the age of 8 years or to pregnant or nursing women. Other antibiotic formulations may be used as well. These include erythromycin ethylsuccinate (EES) and minocycline. Dosing schedules will vary depending upon the patient's presentation.

Meibomian keratoconjunctivitis. This condition responds to warm compresses and massage of the lid to express the meibomian contents. When infection is present, topical antibiotic or antibiotic/steroid ointments should be used. Diabetes should be a consideration when other concurring conditions such as rosacea are absent and the condition is unresponsive to treatment. Oral tetracycline may be beneficial, by inhibiting lipolytic enzymes, especially when rosacea is present. The condition should be stable or improved in 6 weeks[202]; however, some patients may need a lower maintenance dose for a longer period. If keratitis or keratoconjunctivitis is present, the clinician should be aware

of the possibility that methicillin-resistant *Staphylococcus aureus* (MRSA) is the responsible organism.[203]

A prospective study has indicated the efficacy (improved signs and symptoms) of topical cyclosporine (0.05%) in treating posterior blepharitis.[146]

Angular blepharitis. Both forms of angular blepharitis are treated with antibiotic ointment.

Demodicosis. Treatment with a 4% pilocarpine gel (b.i.d. × 2 wk) may, in some cases, be supplemented by the application of antibiotic ointment.[204,205] Nightly lid hygiene, followed by the application of bland ophthalmic ointment tends to inhibit the proliferation of *Demodex*. The ointment is removed the next morning with lid hygiene.[206,207]

4. Managing and Treating the Inflammatory Component of Ocular Surface Disorders

With new information emerging on the inflammatory contributions to ocular surface disorders, a multifaceted approach, including anti-inflammatory therapy may be in order. The use of oral omega-3 fatty acids may be beneficial.[178] In addition, topical application of cyclosporine has been shown to be effective.[144-146,208-210] Adjunctive anti-inflammatory therapies may provide immediate relief and lay the foundation for more targeted therapies. These include the use of topical corticosteroids in addition to the anti-inflammatory strategies cited above.[208-210]

5. Patient Education

Patient education is essential and will assist in compliance. Compliance with management regimens is particularly important in chronic disorders, especially those that may result in considerable morbidity. This concept is applicable to persons with ocular surface disorders, of whom many have underlying systemic conditions. When there is no previously known local or systemic cause for the ocular findings, the patient should

be educated about other conditions possibly associated with the ocular surface disorder and assisted in obtaining further diagnostic evaluations.

The clinician prescribing topical treatment for dry eye should give the patient the rationale for treatment, along with the specific dosages, frequency, and duration. The patient should be made aware of the expected results and given instructions to follow in case of adverse effects. A follow-up examination of the patient should be scheduled to assess the treatment effectiveness.

The treatment of ocular surface disorders requires close, ongoing cooperation between the patient and the practitioner. Thorough discussion of the causes, the rationale for treatment, and the expected results is essential in the management of this condition. Most patients with ocular surface disorders experience significant improvement in their symptoms when the appropriate hygiene, topical, and/or systemic treatments are instituted.

Because there is no cure for the chronic forms of many ocular surface disorders, patients must actively participate in steps to control the inflammatory, infectious, or irritative processes. Thorough explanation of both the chronicity of the disease and the rationale for the therapy helps encourage patient compliance. Specific instructions and realistic expectations for the abatement of symptoms should be reinforced by a scheduled follow-up.

6. Prognosis and Follow-up

For many of the forms of ocular surface disorders, the prognosis is guarded, because the treatment represents only a maintenance strategy. Patient compliance is a major factor in successful management and should be stressed as a component of the care process. When there is an associated systemic cause for the disorder, remission is expected when the underlying condition improves, although intermediary palliative treatment may relieve some symptoms.

Multiple evaluations may be necessary to establish the diagnosis and determine the minimum treatment regimen that produces results. Once a

treatment plan has been shown to be effective, the clinician should provide followup care at appropriate intervals to encourage compliance and continued effectiveness (see Appendix Figure 6, *A Brief Flowchart*).

Follow-up visits for treatment of ocular surface disorders may be as frequent as every few days at the outset, tapering off to once or twice a year after stabilization of the condition (see Appendix Figures 7 and 8). In the absence of other lid or systemic abnormalities, the first acute staphylococcal episode usually can be expected to resolve completely. The chronic forms of ocular surface disorders may be controlled with daily hygiene and topical medication, and, when indicated, courses of systemic medication.

CONCLUSION

Optometrists frequently encounter the clinical challenges of ocular surface disorders. Educating patients about dry eye and blepharitis is a key element in successful control of these ocular problems. With careful diagnosis, treatment, and proper patient education, the long-term comfort of these patients can be maintained. This Guideline serves as a practical aid in the management of patients who present for help with ocular surface disorders.

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IV. Appendix

Figure 1

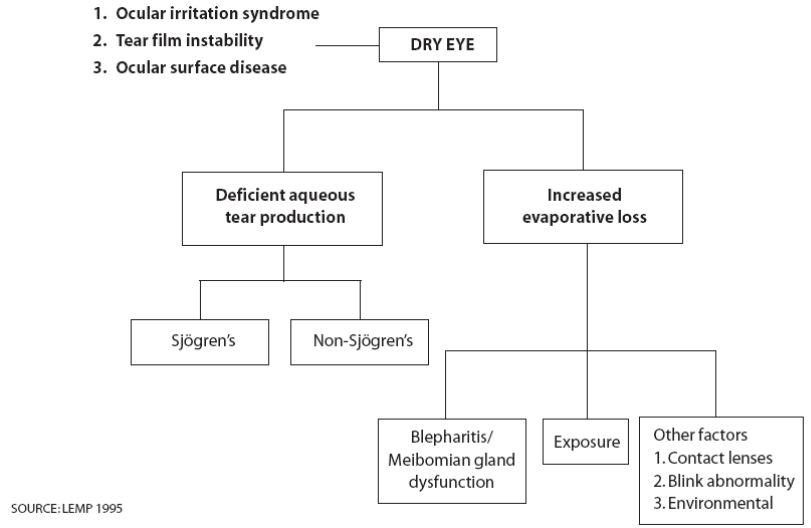
ICD-10-CM Classifications of Blepharitis and Dry Eye Disorders

Inflammation of eyelids	**373**
Blepharitis	**373.0**
Excludes: blepharoconjunctivitis (372.20-372.22)	
Blepharitis, unspecified	**373.00**
Ulcerative blepharitis	**373.01**
Squamous blepharitis	**373.02**
Hordeolum and other deep inflammation of eyelid	**373.1**
Hordeolum externum	**373.11**
Hordeolum NOS	
Stye	
Hordeolum internum	**373.12**
Infection of meibomian gland	
Abscess of eyelid	**373.13**
Furuncle of eyelid	
Chalazion	**373.2**
Meibomian (gland) cyst	
Excludes: infected meibomian gland (373.12)	
Noninfectious dermatoses of eyelid	**373.3**
Eczematous dermatitis of eyelid	**373.31**
Parasitic infestation of eyelid	**373.6**
Code first underlying disease, as:	
pediculosis (132.9)	
Demodex folliculorum (133.8)	
Other inflammations of eyelids	**373.8**
Unspecified inflammation of eyelid	**373.9**
Disorders of lacrimal system	**375**
Other disorders of lacrimal gland	**375.1**
Dacryops	**375.11**
Tear film insufficiency, unspecified	**375.15**
Dry eye syndrome	
Epiphora	**375.2**
Epiphora, unspecified as to cause	**375.20**
Epiphora due to excess lacrimation	**375.21**

Epiphora due to insufficient drainage	**375.22**
Unspecified disorder of lacrimal system	**375.9**
Sicca syndrome	**710.2**
Keratoconjunctivitis sicca	
Sjögren's disease	

Figure 2

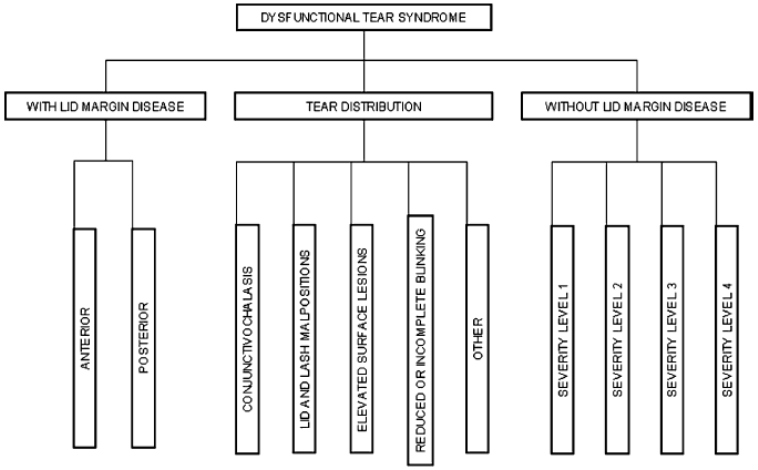
1995 Classification Scheme Based on NEI/Industry Workshop



Reprinted with permission from Lemp MA. Report of the National Eye Institute/Industry Workshop on Clinical Trials in Dry Eyes. CLAO J 1995; 21:221-32.

Figure 3

Delphi Panel Classification Scheme for Dysfunctional Tear Film



Reprinted with permission from Behrens A, Doyle JJ, Stern L, et al. Dysfunctional tear syndrome: a Delphi approach to treatment recommendations. Cornea 2006; 25:900-7.

Figure 4

DEWS Classification (2007)

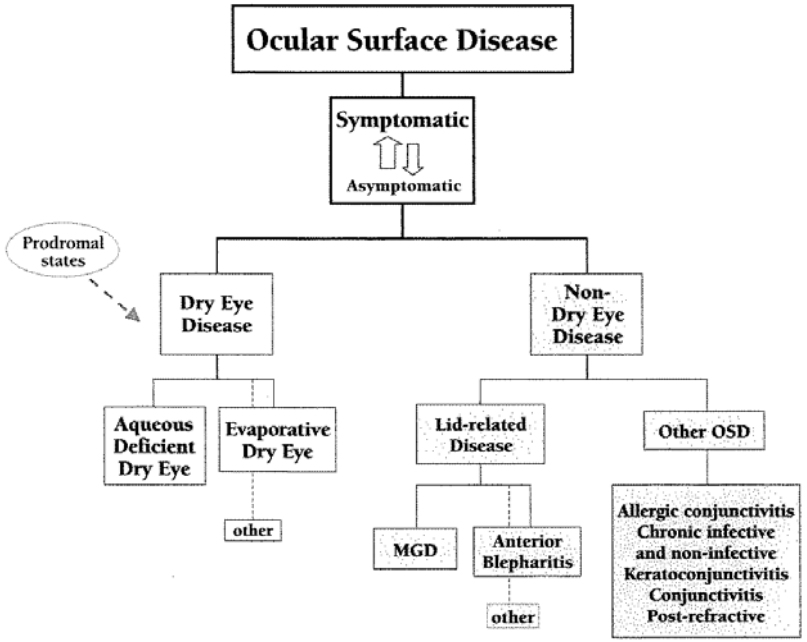


Figure 1. Schematic illustration of the relationship between dry eye and other forms of ocular surface disease. Ocular surface disease is either symptomatic or asymptomatic, but its various subgroups may coexist and interact. Therefore, a patient may suffer from both aqueous deficient and evaporative forms of dry eye, which will consequently be more severe than in the isolated disease. Also, dry eye may coexist with non-dry eye disease. (See text for further details; see also Chapter 1: Definition and Classification.[1]) OSD = Ocular surface disease; MGD = Meibomian gland dysfunction.

Reprinted with permission from Methodologies to diagnose and monitor dry eye disease: report of the Diagnostic Methodology Subcommittee of the International Dry Eye WorkShop (2007). Ocul Surf 2007; 5:108-52.

Figure 5
The Ocular Surface Disease Index

Figure 1. Ocular Surface Disease Index (OSDI) questionnaire for evaluation of dry eye patients

Patient Number	Patient Initials	Physician's Name	Date of Visit (Month / Day / Year)
			/ /

OCULAR SURFACE DISEASE INDEX (OSDI)

Please answer the following questions by checking the box that best represents your answer

Have you experienced any of the following during the last week:	ALL of the time	MOST of the time	HALF of the time	SOME of the time	NONE of the time
1. Eyes that are sensitive to light?	☐	☐	☐	☐	☐
2. Eyes that feel gritty?	☐	☐	☐	☐	☐
3. Painful or sore eyes?	☐	☐	☐	☐	☐
4. Blurred vision?	☐	☐	☐	☐	☐
5. Poor vision?	☐	☐	☐	☐	☐

Have problems with your eyes limited you in performing any of the following during the last week:	ALL of the time	MOST of the time	HALF of the time	SOME of the time	NONE of the time	NOT applicable
6. Reading?	☐	☐	☐	☐	☐	☐
7. Driving at night?	☐	☐	☐	☐	☐	☐
8. Working with a computer or bank machine (ATM)?	☐	☐	☐	☐	☐	☐
9. Watching TV?	☐	☐	☐	☐	☐	☐

Have your eyes felt uncomfortable in any of the following situations during the last week:	ALL of the time	MOST of the time	HALF of the time	SOME of the time	NONE of the time	NOT applicable
10. Windy conditions?	☐	☐	☐	☐	☐	☐
11. Places or areas with low humidity (very dry)?	☐	☐	☐	☐	☐	☐
12. Areas that are air conditioned?	☐	☐	☐	☐	☐	☐

Calculation of OSDI: $\text{OSDI} = \frac{\text{sum of severity for all questions answered}}{\text{4 x (total number of questions answered)}}$

ALL of the time = 4 | SOME of the time = 1
MOST of the time = 3 | NONE of the time = 0
HALF of the time = 2

NOTE: questions answered N/A for the calculations = a NON answered question

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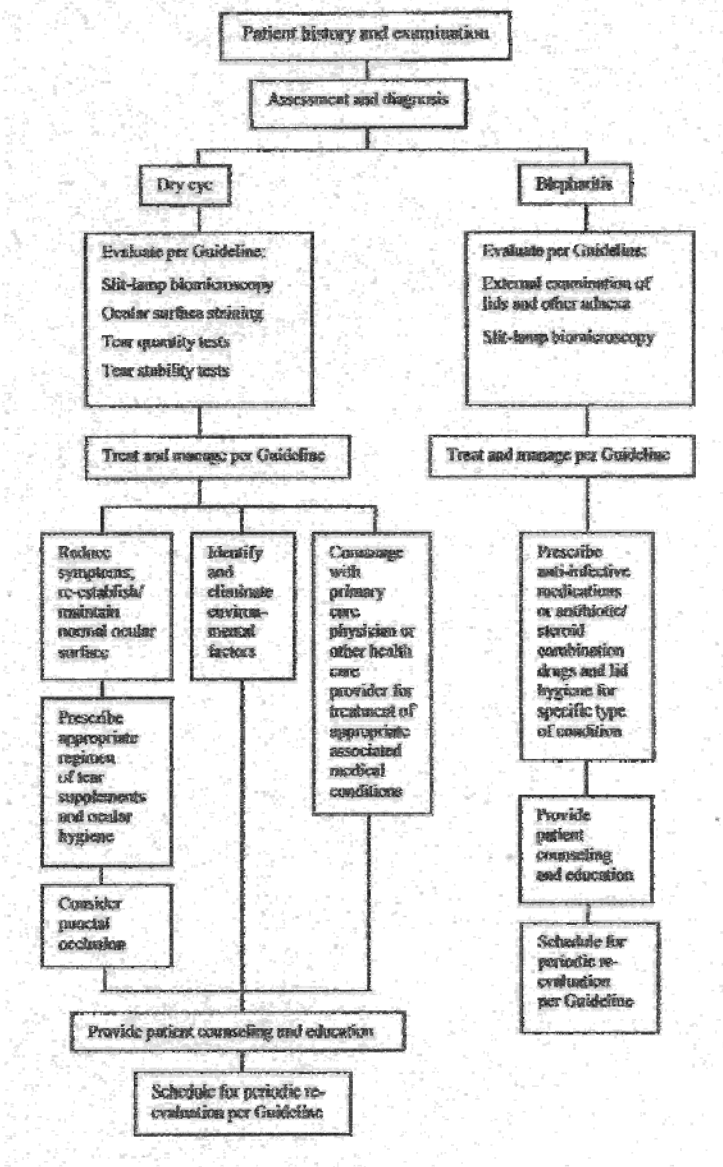
Figure 6
Optometric Treatment and Management of the Patient with Ocular Surface Disorders: A Brief Flowchart



Figure

Frequency and Composition of Evaluation

Degree of Involvement	Frequency of Examination	History
Mild	Annual or p.r.n.	Yes
Moderate	Every 6-12 mo or p.r.n.	Yes
Severe	Every 3-6 mo or p.r.n.	Yes
Associated with systemic disease	Every 1-6 mo or p.r.n.	Yes

*See Guideline for other management strategies.

7

and Management Visits for Dry Eye*

External Evaluation/ Slit-Lamp Biomicroscopy	Supplemental Testing Plan	Management
Yes	Fluorescein, rose bengal/lissamine green staining, BUT	Preserved or unpreserved tear supplements 1.d. up to p.r.n.; patient counseling and education
Yes	Fluorescein, rose bengal/ lissamine green staining, BUT, Schirmer test	Unpreserved tear supplements 4-5 times a day up to p.r.n.; patient counseling and education
Yes	Fluorescein, rose bengal/lissamine green staining, BUT, Schirmer test	Unpreserved tear supplements p.r.n.; ointments h.s.; punctal occlusion; consider tarsorrhaphy; patient counseling and education
Yes	Fluorescein, rose bengal/ lissamine green staining, BUT, Schirmer test	Unpreserved tear supplements p.r.n.; ointments h.s.; punctal occlusion; refer to primary physician; consider tarsorrhaphy; patient counseling and education

NOTE: Anti-inflammatory agents may be introduced at any stage of disease, depending on signs and symptoms manifested by the patient.

Figure

Frequency and Composition of Evaluation

Type of Disorder	Frequency of Examination	History
Seborrheic blepharitis	Weekly until stable, then p.r.n.	Yes
Staphylococcal blepharitis	Twice a week until cleared, then p.r.n.	Yes
Seborrheic/staphylococcal blepharitis	Twice a week until controlled, then q. 6 mo or p.r.n.	Yes
Meibomian seborrheic blepharitis	Twice a week until stable, then as part of preventive care	Yes
Seborrheic blepharitis with secondary meibomianitis	Twice a week until stable (up to 8 wk), then as part of preventive care	Yes
Meibomian keratoconjunctivitis	Twice a week until stable (up to 2 wk), then as part of preventive care	Yes

8

and Management Visits for Blepharitis

External Evaluation/ Slit-Lamp Biomicroscopy	Management Plan
Yes	Lid hygiene t.i.d. until improved, then daily Patient counseling and education
Yes	Antibiotic or antibiotic/steroid ung. h.s. to t.i.d.; tear supplements p.r.n; steroid gtt. or ung. if infiltrates present; lid hygiene t.i.d. until improved, then q.d.
Yes	Antibiotic or antibiotic/steroid ung. h.s. to t.i.d., then lid hygiene q.d. to t.i.d. for control Patient counseling and education
Yes	Lid hygiene up to t.i.d.; scalp shampoo q.d.; meibomian express q.d.; antibiotic or antibiotic/steroid ung. h.s. to t.i.d. Patient counseling and education
Yes	Lid hygiene up to t.i.d.; antibiotic or antibiotic/steroid ung. h.s. to t.i.d.; oral tetracycline or doxycycline (taper) Patient counseling and education
Yes	Lid hygiene; antibiotic or antibiotic/steroid ung. h.s. to t.i.d.; oral tetracycline or doxycycline (taper) Patient counseling and education

Abbreviations of Commonly Used Terms

BUT	Breakup time
BAK	Benzalkonium chloride (also, BAC)
DE	Dry eye
DTS	Dysfunctional tear syndrome
EDTA	Ethylenediaminetetraacetic acid
gtt.	Drops
h.s.	Bedtime
HRT	Hormone replacement therapy
KCS	Keratoconjunctivitis sicca
MGD	Meibomian gland dysfunction
mL	Milliliters
mm	Millimeters
NIBUT	Noninvasive breakup time
OCP	Ocular cicatricial pemphigoid
OSD	Ocular surface disease
POTF	Preocular tear film
p.r.n.	As necessary
q.	Every
q.d.	Every day; daily
SS	Sjögren's syndrome
SJS	Stevens-Johnson syndrome
t.i.d.	Three times per day
TBUT	Tear-film breakup time
TBUT	Tear breakup time
µL	Microliters
ung.	Ointment

Glossary

Adnexa The accessory structures of the eye, including the eyelids, lacrimal apparatus, and the extraocular muscles.

Aqueous layer The clear fluid that makes up the watery component of the preocular tear film.

Biomicroscopy Examination of ocular tissue, using a bright focal source of light with a slit of variable width and height and a binocular microscope with variable magnification.

Blepharitis An inflammatory process affecting the lid margins, the lash follicles, or the openings of the meibomian glands.

Conjunctivochalasis redundant hyperemic conjunctival tissue normally observed at the lower lateral lid margin.

Dellen Shallow, saucer-like, clearly defined excavations at the margin of the cornea, about 1.5 mm × 2 mm, due to localized dehydration; also called Fuchs' dellen.

Dry eye 1. A group of anterior segment eye conditions manifested by a deficiency of the preocular tear film. 2. "A multifactorial disease of the tears and ocular surface that results in symptoms of discomfort, visual disturbance, and tear film instability, with potential damage to the ocular surface. It is accompanied by increased osmolarity of the tear film and inflammation of the ocular surface" (DEWS 2 definition).

Dry eye syndrome (keratoconjunctivitis sicca) Chronic keratitis resulting from insufficient lacrimal secretions.

Dysfunctional tear syndrome one description of the signs of clinical damage to the intrapalpebral ocular surface or symptoms of such disruption from a variety of causes. (Delphi) See also ocular surface disorder, dry eye.

Epiphora An overflow of tears onto the cheek caused by excessive lacrimation, obstruction of the lacrimal ducts, or ectropion.

Fluorescein clearance A water-soluble dye that produces bright green fluorescence in solution.

Fluorophotometry A method of estimating aqueous tear layer flow by measuring fluorescence emitted from the tear film after instillation of fluorescein.

Glycocalyx The interface between the mucin layer of the tear film and the microvillae of the corneal epithelium. Term initially applied to the polysaccharide matrix excreted by epithelial cells forming a coating on the surface of epithelial tissue.

Inspissated Thickened or condensed (secretion), as in the meibomian gland, where it blocks passage of fluid and decreases tear film.

Keratometry Measurement of the anterior curve of the cornea.

Lacrimal punctum The small point-like orifice in the nasal upper and lower lid margins that serves as the opening to the nasolacrimal system.

Laser punctoplasty A form of permanent punctal occlusion using a heat-generating laser to create scar tissue.

Madarosis Loss of eyelashes.

Ocular surface disorder Any condition that reduces the production, alters the composition, or impedes the distribution of the preocular tear film. (*syn*. dysfunctional tear film, DTS).

Ocular surface staining Staining of the cornea or conjunctiva by instilling a dye into the tear film to highlight defects in the surface of the tissue.

Punctal occlusion Temporary or permanent closing or blocking the outflow of tears to the nasolacrimal system.

Schirmer tests Measurements of basal and reflex lacrimal gland function and tear production and volume, using a strip of filter paper.

Tarsorrhaphy Suturing the upper and lower eyelid margins together, either partially or completely.

Tear quantity test Procedure that helps to confirm the diagnosis of aqueous-deficient dry eye.

Tear film breakup The first appearance of discontinuity of fluorescein following a blink. If this is varies from observation to observation, the time in seconds is recorded. If the same area appears to break up time after time, it is a persistent dry spot and signifies a discontinuity at the level of the epithelial-glycocalyx interface.

Tear stability test Procedure that helps to diagnose dry eye. Loss of stability is observed in dry eye of various etiologies, e.g., mucin-deficient, aqueous-deficient, or lipid-deficient dry eye. See: tear film breakup.

Thermal cautery A form of permanent punctal occlusion that uses heat to create scar tissue.

Visual acuity The clearness of vision that depends on the sharpness of the retinal image and the integrity of the retina and visual pathway. It is expressed as the angle subtended at the anterior focal point of the eye by the detail of the letter or symbol recognized.

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