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 32,000 full-time equivalent doctors of optometry currently in practice in the United States. Optometrists practice in more than 7,000 communities across the United States, serving as the sole primary eye care provider in more than 4,300 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.

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American Optometric Association

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 for a significant portion of the American public and are often the first health care practitioners to diagnose ocular surface disorders. Although some classifications use "ocular surface disease" in the context of clinical damage to the intrapalpebral surface, that term is not universally understood. In this Guideline, the term "ocular surface disorders" encompasses a number of disease entities, some specific and some nebulous. These disorders range from poorly defined contributors to ocular surface discomfort (either direct or indirect disruptors of the tear film) to nonspecific entities described elsewhere as ocular surface disease, to dry eye, including aqueous deficiency secondary to systemic causes (e.g., any of the family of collagen vascular diseases) that may result in dry eye. As knowledge expands and classification systems change, this terminology continues to evolve.

This Optometric Clinical Practice Guideline for the Care of the Patient with Ocular Surface Disorders describes appropriate examination and treatment procedures to reduce the risk of visual discomfort and disability from these entities. The most common ocular surface disorders stem from tear film abnormalities and blepharitis, each of which may lead to ocular surface disease. The Guideline contains recommendations for timely diagnosis, treatment, and, when necessary, referral for consultation with or treatment by another health care provider. This Guideline will assist optometrists in achieving the following goals:

- Identify patients at risk of developing ocular surface disorders
- Accurately diagnose patients with ocular surface disorders
- Differentially diagnose age, drug, environmental, and systemic disease-related causes of ocular surface disorders
- Improve the quality of care rendered to patients with ocular surface disorders

- Reduce the prevalence and degree of disability from ocular surface disorders
- Inform and educate patients and other health care providers about the visual complications, risk factors, and treatment options associated with ocular surface disorders.

I. STATEMENT OF THE PROBLEM

Any condition that reduces the production, alters the composition, or impedes the distribution of the preocular tear film (POTF) may cause a noticeable irritation to the structures of the front surface of the eye and a degradation of vision. These conditions are often related to problems with the structure or function of the eyelids, cornea, or conjunctiva. Depending upon the severity of symptoms, individuals may be limited in their ability to see clearly and comfortably and may be at increased risk of developing secondary infection or chronic inflammation that may not respond to treatment. The two most commonly encountered ocular surface disorders are tear film disorders and blepharitis. Additional subclassifications include those "dry-eye" states associated with systemic connective tissue disorders, specifically Sjögren syndrome (SS). With regard to lipid deficient tear-film disorders, the involvement of meibomian gland dysfunction (MGD) has been proposed.[1-3]

An estimated one-fifth of visits to eye care practitioners are related to ocular complaints secondary to dry eye (DE).[4] As many as 25 percent of American adults may be affected by dry eye or have dry eye symptoms.[5] In addition, self-treatment with tear supplements represents a major share of the over-the-counter pharmaceuticals market. These statistics indicate a significant need for access to professional care.

Although the ultimate etiology of ocular surface disorders is often enigmatic, there is general agreement that proper management is based on clinical observation and diagnosis of the underlying causative mechanism(s). Multiple etiologies may exist. Some clinicians focus on objective signs;[6] others emphasize symptomatology.[7] Specific tests are available to assist in differentiating the various forms of tear film abnormalities and identifying potential treatment strategies. Careful clinical observation, accurate diagnosis, and appropriate intervention can eliminate or minimize the deleterious effects of ocular surface disorders on the quality of life.

Lid disorders, such as blepharitis, are major contributors to dry eye symptoms. Blepharitis can generally be identified and categorized by its clinical presentation. Alteration of the POTF may be the initial cause of

symptoms. Identifying patients with risk factors for these conditions, offering preventive recommendations, and providing timely treatment may help ensure high-quality and cost-effective care.[8]

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 borders. The anterior border has two or three rows of stiff, sensitive cilia. The sharp posterior border, positioned against the globe, contains the orifices for the tarsal glands. The meibomian gland—25 in the upper lid and 20 in the lower—are located within the tarsal plates and secrete lipids for the oily layer.[9] They are not connected to the lash follicles. Although the maximum capacity of tears of the open eye and the fornices is 25 microliters, the normal volume is only about 7 microliters. Each blink removes the existing tear film and distributes fresh tears across the exposed cornea and conjunctiva.

2. Normal Tear Film Composition

The tear film has three layers. The pilosebaceous meibomian glands in the lids produce most of the outermost (lipid) layer. The Zeis and Moll glands of the eyelid margin and lashes also contribute to this layer. Oily secretions in this layer function to contain the aqueous phase of the POTF (by reducing surface tension), and stabilize and retard evaporation of the aqueous layer below it.[9-12] In the normal healthy eye, the lipid layer's thickness is less than 0.1 micron. Meibomian lipids (meibum) are mainly waxy and cholesterol esters. The high molecular weight and low polarity of meibum are important properties for the formation of the tear film; alteration of polarity in disease states such as blepharitic

conditions may affect its stability. Thickened or contaminated lipid layers that distort observable interference fringe patterns may be used diagnostically.[13]

The aqueous (intermediate) layer makes up about 90 percent of the tear film, most of which is produced by the accessory exocrine lacrimal

glands of Krause and Wolfring.[14] This layer contains proteins that exhibit antibacterial activity, including lysozyme and lactoferrin, which are useful for diagnostic evaluation of the aqueous layer.[15-16]

The innermost layer of the tear film is the mucous layer. Produced primarily by goblet cells in the conjunctival fornices, mucous 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.[17] The mucous layer is anchored to the epithelium by microvillae. The traditional concept of tear film breakup is based on contact between the lipid and mucous layers or local breakdown of the mucous layer.[18-21]

Ocular surface disorders can result from a compromise of the structure or function of the cornea, eyelids, conjunctiva, or sclera. This Guideline describes the most common clinical etiologies of ocular surface disorders: dry eye and blepharitis. (See Appendix Figure 4 for ICD-9-CM classifications of dry eye and blepharitis.)

3. Dry Eye-Related Ocular Surface Disorders

The term "dry eye" refers to ocular surface disorders in which the common etiology is aqueous deficiency. This may lead to a cascade of secondary disorders. Adequate POTF function is necessary for clear and comfortable vision. In addition, the POTF has other physiologic functions:

- It is the initial refracting surface of the eye.
- It serves as the primary source of oxygen to the anterior cornea.
- It supplements the eye's antibacterial defenses.
- It provides lubrication for the eyelids and ocular surface.
- It flushes away metabolic waste products and debris.
- It performs a needed anti-inflammatory function following ocular surface insult.

Although decreased production of basal tears (aqueous) is associated with aging, concomitant stenosis (collapse) of the lacrimal punctae usually results in maintaining the equilibrium between tear secretion and

elimination in older individuals. Even though they may be free from other dry eye symptoms, these patients may have secondary reflex tearing.[22] Others may have dry eye symptoms related to POTF inadequacies.

Dry eye symptoms can result from an alteration or misdistribution of any tear-film component. Ocular surface disorders may exist in isolation but frequently overlap, depending upon the underlying pathophysiology and its duration.[23] Clinically, multiple forms may co-exist.

a. Aqueous Deficient Dry Eye

The symptoms of aqueous deficient dry eye are usually bilateral and may produce a foreign-body sensation and lacrimation. As the most common tear film abnormality, aqueous deficient dry eye usually results from reduced aqueous production and may be secondary to lacrimal gland deficiency as seen in Sjögren syndrome. Appearing clinically as a decreased tear meniscus, with 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 reduced wetting on Schirmer's testing, as well as ocular surface staining, although these latter signs are not specific to aqueous deficient dry eye.

Aqueous deficiency secondary to SS results from lacrimal gland inflammation, infiltration, and atrophy.[24] Thought to be autoimmune in origin, primary SS is associated with collagen-vascular or connective tissue disease, most frequently rheumatoid arthritis. 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 contemporary literature reviews.[25] 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).[26] It also may occur in women who are pregnant or

taking birth control pills, in whom estrogen and prolactin levels are elevated.[27, 28]

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

A reduction in the number of conjunctival goblet cells, resulting in a decrease in mucin production, can be caused by any condition that damages the conjunctiva. Mucin deficient dry eye conditions include allergic conjunctivitis,[29] ocular cicatricial pemphigoid (OCP), erythema multiformae (Stevens Johnson syndrome), 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.

In OCP and the Stevens-Johnson syndrome (SJS), goblet cell loss is due to an autoimmune response that deposits immunoglobulins at the basement membrane zone of the conjunctiva,[30] which leads to the clinical picture of bullae at the subepithelial level. Progressive infiltration results in contraction of the conjunctiva with symblepharon formation.[31]

Goblet cell density may increase secondary to thermal and chemical injuries.[32] The resulting ocular surface disorders differ from OCP or SJS at the cellular level though appearing clinically similar.

c. Lipid Abnormality Dry Eye

Lipid abnormality dry eye is typically associated with lid disorders caused by inflammation, trauma, or scarring. Lipid abnormality secondary to chronic blepharitis results from alteration of the polarity of lipid secretions from lid and lash glands, leading to a tear evaporation rate up to twice the normal rate.[33] Common microbes secrete lipases, which hydrolyze lipids, with the subsequent release of free fatty acids that are highly surface active and capable of triggering dry spot formation in the POTF.[11] When blepharitis is included as the ultimate cause of lipid abnormality DE, this etiological classification represents the majority of cases of DE.

d. Surfacing 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. Incomplete or infrequent blinking, which results in excessive tear evaporation and possible 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.

e. Epitheliopathies

Corneal epitheliopathies are characterized by an irregular surface of microvilli that prevents mucin from adhering to the cornea. The causes include corneal scars, chemical burns, recurrent corneal erosions, contact lens complications, trauma from entropion, or lash abnormalities such as trichiasis or distichiasis.

Contact lens wear can induce dry eye symptoms in a patient who has a pre-existing, asymptomatic, marginally dry eye condition. 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. Irritation to the tarsal conjunctiva stimulates excessive production of mucous, and blinking is often inhibited.

2. Blepharitis-Related Ocular Surface Disorders

Major contributing factors to the alteration of lipid secretion appear to be lid and lash disorders, which may be precipitated by inflammation (blepharitis). Blepharitis may be the initial clinical sign of the altered lipid secretion that results in premature evaporation of the aqueous tears.

"Blepharitis" is a non-specific term for inflammatory conditions of the eyelids. Dermatologic manifestations of blepharitis may include eczema[34] involving the anterior or posterior lid margins. Blepharitis can have secondary manifestations that adversely affect visual comfort or acuity by disrupting the ocular surface of the cornea, and contact lens wear can become intolerable.[35] Certain systemic conditions, such as acne rosacea, chlamydial, and viral infections, may predispose an individual to inflammation of the eyelids. Most forms of blepharitis are chronic, but acute forms do exist. The most common types of blepharitis are:[36,37]

a. Staphylococcal Blepharitis

Usually caused by Staphylococcus aureus or Staphylococcus epidermidis organisms, 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 margins, scaly collarettes at the base of the lashes, and possibly skin ulceration, a frequent result is aqueous deficient dry eye.[37,38] Hordeolum and chalazion may also occur.

b. 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 culture normal populations of surface organisms. Although skin inflammation is not necessarily evident, greasy, foamy scales called scurf surround the bases of the cilia.

c. Seborrheic/Staphylococcal Blepharitis

Another common form of blepharitis is seborrheic/staphylococcal blepharitis, also called ulcerative or mixed blepharitis.[36] 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 the course of the disease. Bacterial cultures are usually positive. Histologic examination reveals chronic, moderate, nongranulomatous inflammation.

d. Meibomian Seborrheic Blepharitis

Meibomian seborrheic blepharitis can be identified by the presence of increased meibomian and seborrheic secretions without acute inflammation. Tears are sudsy and foamy, producing a burning sensation, especially in the morning. Itching and tearing are common symptoms. The lid glands are dilated, leading to copious meibomian secretions and bulbar injection.

e. Seborrheic Blepharitis with Secondary Meibomianitis

Seborrheic blepharitis with secondary meibomianitis (meibomitis) is similar to seborrheic blepharitis but has sporadic episodes of inflammation and meibomianitis that result in a spotty presentation of clogged meibomian glands and anterior seborrhea. Lipid secretions are of toothpaste consistency, producing an unstable POTF. Cultures reveal the presence of normal flora.

f. Meibomian Keratoconjunctivitis

Meibomian keratoconjunctivitis (primary meibomianitis) is the most severe lid margin inflammation. Typically occurring in persons in their fifties, it has no predilection for gender but is more common in colder climates. It is frequently associated with acne rosacea and is part of a

generalized sebaceous gland dysfunction that clogs the meibomian openings with desquamated epithelial cells. Because lipid secretions have a melting point higher than the ocular surface temperature, stagnation of free fatty acids within the gland and inspissated openings results in a lipid deficient tear film.

g. Angular Blepharitis

Angular blepharitis is localized on 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.

h. Demodicosis

Demodicosis is the inflammatory reaction to a common mite that inhabits the eyelash follicles in most persons over age 50. There are two species of mite:[39]

- Demodex folliculorum, which is present in hair and eyelash follicles, consumes epithelial cells, produces follicular distension and hyperplasia, and increases keratinization, leading to cuffing of the bases of the cilia.

- Demodex 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 tear layer.

B. Epidemiology of Ocular Surface Disorders

1. Dry Eye

a. Prevalence

Dry eye may be the most ill defined of all ocular disorders. Although keratoconjunctivitis sicca (KCS), the most common form of DE, results from the development of an aqueous deficient tear state in adults, there

are no well-defined studies documenting its prevalence. However, many clinicians believe this form of dry eye occurs most frequently in the fifth decade of life, particularly in women. KCS is a bilateral disease except when there is a specific unilateral cause.

More severe forms of aqueous deficient DE associated with systemic disease, especially collagen-vascular diseases, have been studied thoroughly. Up to 20 percent of persons with rheumatoid arthritis have KCS.[25] Sjögren syndrome is the classic label for the triad of dry eye, dry mouth, and arthritis, but other connective tissue diseases may be involved.

True mucin deficiency is rare; one report estimates the prevalence of OCP at 1 in 20,000 persons.[40] 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.[30,41-44] This medically induced complication is rare with contemporary glaucoma treatment options.

Most problems involving lipid layer instability are related to glandular dysfunctions that produce thickened meibum, leading to accelerated surface evaporation. There is a close association of various forms of meibomianitis with blepharitis. Lipid layer abnormalities resulting from complete absence of meibomian gland secretions are rare.[13] Meibomian gland deficiencies have been evaluated by eyelid transillumination and classified as atrophic or dysfunctional (rosacea) among patients with symptoms consistent with ocular irritation.

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.[41-44]

2. Blepharitis

a. Prevalence

Blepharitis refers to a range of acute and chronic disorders involving the eyelid margin. Because inflammation can occur on either the anterior or posterior lid margins, involvement of the skin and mucous membrane is common. Each form of blepharitis has its own epidemiological characteristics.

Most staphylococcal blepharitis occurs in younger women (mean age, 42 years),[37] whereas the seborrheic variations tend to occur in older individuals. Acne rosacea, a disease of unknown prevalence, is more common in fair-skinned persons between the ages of 30 and 50, especially women.[45] Gross ocular lesions occur in many cases of acne rosacea, and almost all affected persons eventually develop recurrent or chronic blepharitis and meibomianitis. There is a strong association between KCS and staphylococcal blepharitis.[37]

b. Risk Factors

Underlying dermatologic conditions may be associated risk factors for blepharitis. Seborrheic blepharitis is associated with seborrheic dermatitis. Meibomianitis occurs approximately twice as frequently with acne rosacea as it does with seborrheic dermatitis.[36] Atopic dermatitis and psoriasis may also have a blepharitis component. Patients with SS-related KCS appear more likely to develop meibomian gland disease.[46]

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. A decrease or alteration in the production of any component of the tear film 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. Some symptoms may occur only under conditions of stress, such as very dry or smoky environments.[47] 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, burning symptoms and visual interference can be debilitating. 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. Not only are lid infections commonly associated with dry eye, but there is a higher likelihood of secondary conjunctivitis and keratitis. Therefore, dry eye can 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 blurring of vision when the tear film is disrupted. In moderate cases, the ocular discomfort becomes marked and visual acuity may be reduced. Depending on the severity and the cause of the dry eye, observable signs may include rapid tear film breakup, debris in the tear film, a scanty 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.[48] Decreased aqueous volume is associated with reduced ocular surface defense and increased susceptibility to irritation, allergy, and infection due to tear stagnation.[3,49-50] A major consequence of reduced aqueous volume is reduced antibacterial function due to decreased lactoferrin and

lysozyme levels.[15-16,51] Staphylococcal organisms can produce toxins that can cause superficial punctate keratopathy.[52]

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 interpalpebral desiccation.[37] A more significant consequence of an unstable tear film, persistent dry spots may be associated with either abnormalities of the tear distribution system or reduced tear flow. Repeated blinking or treatment with tear supplements can help to relieve this drying.

Squamous metaplasia of the conjunctiva occurs secondary to changes in the ocular surface, perhaps as a result of environmental exposure.[53] Impression cytology studies suggest abnormal conjunctival epithelium as well as changes in the goblet cells.[54,55] Two possible etiologies have been proposed: (1) loss of vascularization, which prevents normal epithelial differentiation, and (2) inflammatory changes that induce epithelial alteration.

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. The use of tear supplements can make symptoms tolerable in the milder forms of the condition. Specifically, nonpreserved tear supplements also play a role in the relief of moderate and advanced cases. Lid hygiene 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. More 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. Staphylococcal infection of the lid margin produces dermatitis, and there is often an aqueous tear deficiency.[37]

The severity of the symptoms is also 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 an alteration of the POTF with consequent signs and symptoms. In severe forms, secondary conjunctival and corneal inflammation may occur.

c. Early Detection and Prevention

Steps to prevent blepharitis are aimed at controlling the severity of the inflammation and preventing secondary complications. 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 acne rosacea, should be treated. In the event of exacerbation, early diagnosis and treatment can help minimize the degree of inflammation and infection.

II. CARE PROCESS

This Guideline describes the 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. Some components of care may be delegated.

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 blepharitis includes many of the elements of a comprehensive eye and vision examination[1] and a more in-depth evaluation of the ocular surface and adnexa. The evaluation for ocular surface disorders includes a carefully detailed patient history, an assessment of associated risk factors, and an 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, 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.[56] The history should document associated conditions that make an individual more likely to develop tear film abnormalities. Common ocular complaints include burning or stinging, itching, scratchiness, irritation, tearing, increased

[1] Refer to the Optometric Clinical Practice Guideline on Comprehensive Adult Eye and Vision Examination.

mucous and reduced contact lens tolerance. An index specific to ocular surface disorders has been proposed and validated.[57]

Due to the visible nature of some forms of 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 Dry Eye

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 and rose bengal or lissamine green staining.

In moderate manifestations of ocular surface disorders, there may be obvious changes in tear film instability, subtle corneal superficial punctate keratopathy, and more apparent conjunctival staining. In more severe cases, the cornea may reveal 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 with folds (conjunctivochalasis) in the exposed bulbar portion. The POTF may have increased viscosity, debris and foamy secretion, and 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 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.[58] 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 measures the tear secretion of the accessory lacrimal glands; without anesthetic, it measures the tear production of the lacrimal gland. Although it is controversial because the results are often inconsistent, the Schirmer tear test can provide useful clinical information.

- **Fluorescein staining**. After adding fluorescein, a water-soluble, inert dye (not fluorescein-anesthetic solution) to the ocular surface, the clinician can observe the rate of dilution[59] of the aqueous component of the POTF, especially with enhancement by cobalt-filtered illumination. Acceptance of this method has been hampered by lack of a standard.

- **Evaluation of the tear prism.** The tear meniscus height can be assessed with biomicroscopic examination both with and without instilling fluorescein dye.[60] A tear meniscus height of 0.5-0.6 millimeters (mm) is considered normal. A scanty or absent tear meniscus is an indication of an aqueous tear deficiency.[61]

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

- **Rose bengal staining**. Perhaps the most useful test for identification of ocular surface disorders specific to aqueous deficiency is 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.[62] 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[63] enables description of the intensity and extent of involvement and may be more useful in documenting subtle changes in response to treatment strategies. 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.[64,65]

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

Schirmer II (irritation)	Cotton thread test[66]
Lissamine green staining[67]	Phenol red thread test[68]
Tear volume measurements	Fluorophotometry; fluorescein dilution
Lacrimal equilibration time[69]	Temporary punctal occlusion

Several procedures are commonly used to evaluate tear film stability.

- **Tear film breakup time (BUT)**. The time required for a tear film to break up following a blink[70] is normally 15-20 seconds; a BUT of less than 10 seconds seconds is a practical index for an abnormal tear film. Because lipid contamination of the mucin layer decreases the surface tension and eliminates the aqueous portion of the tear film in that area, a decreased BUT may also indicate a mucin deficiency.

- **Tear thinning time.** This noninvasive test involves a keratometer to view the mire image and measuring the time from a complete blink to distortion of the image.[71]

- **Lactoferrin concentration tests: LactoPlate® and LactoCard®.** Each of these clinical tests estimates the lactoferrin content of tears by different means. The LactoPlate test uses a radial immunodiffusion technique, whereas the LactoCard® test uses the enzyme-linked immunosorbent assay (ELISA).[72,73] Determining tear lactoferrin with the LactoCard® assay requires only 10-15 minutes, significantly less than the 3 days required for the LactoPlate® test.[74] Tear lactoferrin concentrations below 0.9 mg/ml are considered diagnostic of dry eye.

- **Lysozyme radial diffusion assay: Quantiplate.** This test estimates the amount of lysozyme in tears. Low tear protein levels have been shown to be sensitive for KCS.[75] The lysozyme test is a more subtle measure of lacrimal gland function than either tear flow or the Schirmer test.

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

Tear osmolarity test[76]
Conjunctival scraping and biopsy
Mucin assay test (tear ferning)[79]
Specular reflection of the tear surface[81,82]
Impression cytology[77]
Tear protein analysis[78]
Lipid layer interference patterns[80]
ELISA tear protein profile.[83]

Despite nearly a century of research attempting to characterize clinical signs among patients with dry eye, no consensus has emerged. 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 two or more abnormal test results.[84] True dry eye, whether caused by aqueous, mucus, or lipid deficiency, must be diagnosed and treated as early as possible to prevent further changes in the delicate, exposed ocular surface. Table 1 summarizes normal values that have been established for selected tests.

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 (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 break-up 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
Lysozyme	Lacrimal gland function	Total lysozyme reactivity (TLR) <1.0

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 the inflammation. Differentiating among the various presentations of blepharitis requires the use of the 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 acne 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
- Examination of the tear film for lipid layer abnormalities.
- Evaluation of the palpebral and bulbar conjunctiva

Each type of 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, madarosis, ectropion, or entropion may result. The lower third of the cornea may have 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**. Meibomian openings are dilated in this condition associated with seborrheic dermatitis. A distinguishing clinical feature is increased meibum, which causes a foamy tear film, especially at the lateral canthus. The bulbar conjunctiva is injected, and there may be concurrent KCS.
- **Seborrheic blepharitis with secondary meibomianitis**. Chronic with exacerbations, this condition 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 acne rosacea. The gland openings are obstructed by desquamated epithelial cells, resulting in a poor POTF that can be identified by rose bengal staining. The meibomian secretions have a higher melting point than the ocular surface temperature which results in constipated sebum and inspissated plugs of free fatty acids at the gland openings that are often inflamed and pouted. The tear film is very unstable.
- **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.[85] 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.

B. Management of Ocular Surface Disease

The authority for an optometrist to provide treatment for ocular surface disorders is determined by state law. Treatment strategies may require consultation with or referral to the patient's primary care physician, a dermatologist, an ophthalmologist, or other health care provider, as appropriate. Appendix Figure 1 presents a flowchart describing the management of patients diagnosed with dry eye or blepharitis.

l. General Considerations

A comprehensive approach to 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.[85] 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. Typically, anterior blepharitis does not have the dry eye component of posterior blepharitis (meibomianitis).[87,88] In diagnosing blepharitis, the clinician must pay special attention to the lid margins and the preocular tear film.

Contact lens wear may pose a threat 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 tear BUT 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 acne rosacea may complicate contact lens wear for patients with chronic dry eye.

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:

- 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)

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 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. 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

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

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 provide 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 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.

- **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.[47,89] Recently, a large, placebo-controlled that the immunosuppressive agent cyclosporine can both ameliorate symptoms and reduce the clinical signs of dry eye.[90] In the spring

* 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 2003, Restasis (cyclosporine ophthalmic solution 0.05%) became available, by prescription, for the treatment of KCS.

Tear supplements can be designed to mimic the tonicity, pH, retention time, mucomimetic properties, and lubricating features of the POTF, and to increase the size of the tear meniscus. Available in a variety of formulations, tear supplements have active ingredients representing a wide spectrum of components (Table 2). The U.S. Food and Drug Administration (FDA) requires all multidose ophthalmic solutions to be preserved against contamination from a standard group of pathogens. With chronic use, these preservatives may cause adverse effects, including reduction of the desired effect, allergic response, or toxic reaction. Unpreserved unit-dose containers prevent the preservative problem but are more costly. Recently, tear supplements use preservatives that, when instilled into the tears, 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 are:

Thimerosal – hypersensitivity reaction in an estimated 10-25 percent of users[91]

Benzalkonium chloride – POTF instability, lowered BUT, and disrupted corneal epithelial cell functions when dosed at commercial concentrations more frequently than three times daily.[92]

Chlorobutanol – evaporation, corneal epithelial cell changes

Ethylenediaminetetraacetic acid (EDTA) – contact allergy

Chlorhexidine digluconate – storage in the corneal and conjunctival epithelium.[93]

Effective management of dry eye may require the instillation of tear supplements as often as l drop every 30 minutes or as infrequently as l drop daily at bedtime. Only evaluation and continual monitoring can establish the frequency and duration of treatment.

Table 2
Components in Tear Supplements

- **Cellulose ethers**
  Hydroxypropyl cellulose
  Hydroxypropyl methylcellulose
  Methylcellulose
  Hydroxyethyl cellulose
  Carboxymethyl cellulose

- **Polyvinyl polymers**
  Polyvinyl alcohol (PVA)
  Polyvinyl pyrrolidone (PVP)
  Polyvinyl methyl ether

- **Mucolytic polymers**

- **Hyposmotic artificial tears**
  Glycerin dextran

- **Vitamin A**
  Retinol

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 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.

Lacrisert® is a rod of water-soluble hydroxypropyl cellulose that is placed in the lower cul-de-sac. During the 12-24 hours in which the preservative-free polymer is released into the tears, vision may be blurred and the patient may experience a foreign-body sensation.

- **Punctal occlusion**. When surface treatments do not relieve symptoms, preocular moisture can be retained by blocking the outflow of tears to the nasolacrimal system. This blockage can be accomplished by dissolvable, removable, or permanent punctual occlusion (Table 3). The clinical efficacy of silicone punctual plugs may be limited in both duration (>2 years) and rate of retention (~50%).[94]

Table 3
Procedures for Punctal Occlusion

- Collagen implants—dissolvable plugs that provide insight into the effectiveness of using silicone plugs
- Tapered shaft silicone punctal plugs[95,96]
- Cylindrical shaft silicone punctal plugs
- Herrick intracanalicular implants
- Thermal cautery and other forms of permanent occlusion—may indicated when the patient's predisposing condition is permanent
- Electrodesiccation using the Hyfrecator electrocautery unit—permanently scars the punctum and canaliculus
- Laser punctal occlusion[97] (punctoplasty) using the argon laser—less efficacious than thermal or electric cautery
- Surgical repositioning of the punctum[98] 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**.[99,100] Used with sporadic success, and especially useful when filaments or mucus strands are present, they may offer an alternative in cases of severe ocular surface disease.[101]
- **Moisture chamber goggles**.[102] 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.
- **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.[103] 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).[26]
- **Salivary gland transplant**.[104] The replacement of salivary gland tissue in the conjunctiva has been attempted as a means of producing preocular secretion. Autologous submandibular gland transplantation to the temporal fossa has also been suggested.[105]
- **Limbal grafts**. Proposed for severe cases of ocular surface disease, limbal grafts remain experimental while guidelines for their implemention evolve.106,107

3. Management of Blepharitis

a. Basis for Treatment

Acute forms of blepharitis are usually the direct result of infection of the lipid-producing glands that open to the lid margin. Their clinical presentation includes internal and external hordeola. The treatment is relatively straightforward. Though essential, lid hygiene alone may not resolve the problem. Depending upon the clinical findings, an appropriate anti-infective drug can be administered topically, systemically, or in combination. On the other hand, chronic blepharitis is a disease for which there is no complete cure. Aggressive therapy should initially include a minimum of 6 weeks of lid hygiene and appropriate anti-infective medications to gain control of the condition, followed by continuing treatment to maintain control of chronic blepharitis.

b. Available Treatment Options

Because every category of blepharitis is actually a separate condition, each needs to be addressed individually.

- **Staphylococcal blepharitis**. Treatment includes an antibiotic ointment to control the infection, as well as lid hygiene.[108] Lid hygiene can be performed with a standard lid scrub or by using diluted (1:10) baby shampoo applied with a facial cloth. Erythromycin, bacitracin, polymyxin B-bacitracin, gentamicin, and tobramycin are all effective antibiotics for treatment of staphylococcal blepharitis. Antiobiotic 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**. The application of warm, moist compresses to soften and loosen the crusts is followed by washing with a commercial lid scrub or diluted (1:10) baby shampoo on a facial cloth or cotton swab, taking care not to involve the globe. The scalp and eyebrows should be washed with a selenium antidandruff shampoo.[109]

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

- **Meibomian seborrheic blepharitis**. The treatment includes the same warm compress and shampoo regimen as for seborrheic blepharitis. In addition, the meibomian glands are 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.[88,110]

- **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 may require systemic tetracycline (up to 1g/day) or doxycycline (100 mg/day) for at least 6 weeks.[111,112] It is not unusual for patients with this condition to require lower maintenance doses after tapering. Neither tetracycline nor its derivatives should not be given to children or pregnant or nursing women.

- **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. Oral tetracycline may be beneficial, by inhibiting lipolytic enzymes, especially when acne rosacea is present. The condition should be stable or improved in 6 weeks;[116] however, some patients may need a lower maintenance dose for a longer period.[114]

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

- **Demodicosis**. Treatment with a 4% pilocarpine gel (b.i.d. x 2 wk) may, in some cases, be supplemented by the application of antibiotic ointment.[114,115] 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.[116,117]

4. Patient Education

Contemporary health care ethics dictate that patients be kept informed about their status and encouraged to participate in their health care management. This concept is applicable in 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.

When topical treatment for dry eye is prescribed, the patient should be given 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 blepharitis requires close, ongoing cooperation between 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 blepharitis have a 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 blepharitis, patients must actively participate in steps to control the inflammatory process. 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.

5. Prognosis and Follow-up

In many cases of dry eye, 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. Multiple evaluations may be necessary to establish the diagnosis and to determine the minimum treatment regimen that produces results. Once a treatment plan has been shown to be effective, the clinician should provide follow-up care at appropriate intervals to encourage compliance and continued effectiveness (See Appendix Figure 2).

Follow-up visits for treatment of blepharitis 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 Figure 3). 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 blepharitis may be controlled with daily hygiene and topical medication, and, when indicated, courses of systemic medication.

CONCLUSION

The clinical challenges of ocular surface disorders frequently confront the optometrist. Because dry eye and blepharitis constitute the largest components of the ocular surface disorder classification, the primary care optometrist needs to understand, examine, diagnose, treat, and manage each condition with a careful view toward each treatment's effect on the ocular surface. 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 l
Optometric Management of the Patient with Ocular Surface Disorders: A Brief Flowchart

Patient history and examination

Assessment and diagnosis

Dry eye

Evaluate per Guideline:

Slit-lamp biomicroscopy
Ocular surface staining
Tear quantity tests
Tear stability tests

Treat and manage per Guideline

- Reduce symptoms and maintain normal ocular surface
- Prescribe appropriate regimen of tear supplements and ocular hygiene
- Consider Punctual Occlusion
- Identify and eliminate environmental factors
- Comanage with primary care physician or other health care provider for treatment of associated medical conditions

Provide patient counseling and education

Schedule for periodic re-evaluation per Guideline

Blepharitis

Evaluate per Guideline:

External examination of lids and other adnexa
Slit-lamp biomicroscopy

Treat and manage per Guideline

- Prescribe anti-infective medications or antibiotic/steroid combination drugs and lid hygiene appropriate for specific type of condition
- Provide patient counseling and education
- Schedule for periodic re-evaluation per Guideline

Figure 2
Frequency and Composition of Evaluation and Management Visits for Dry Eye*

Degree of Involvement	Frequency of Examination	History	External Evaluation/ Slit Lamp Biomicroscopy	Supplemental Testing Plan*	Management
Mild	Annual or p.r.n.	Yes	Yes	Fluorescein, rose bengal staining, BUT	Preserved or unpreserved tear supplements q.d. up to p.r.n.; Patient counseling and education
Moderate	Every 6-12 mo or p.r.n.	Yes	Yes	Fluorescein, rose bengal staining, BUT, Schirmer test	Unpreserved tear supplements 4-5 times a day up to p.r.n.; Patient counseling and education
Severe	Every 3-6 mo or p.r.n.	Yes	Yes	Fluorescein, rose bengal staining, BUT, Schirmer test	Unpreserved tear supplements p.r.n.; ointment h.s.; punctal occlusion; consider Tarsorrhaphy; Patient counseling and education
Associated with systemic disease	Every 1-6 mo or p.r.n.	Yes	Yes	Fluorescein, rose bengal staining, BUT, Schirmer test	Unpreserved tear supplements p.r.n.; ointment h.s.; punctal occlusion; Refer to primary physician; consider Tarsorrhaphy; Patient counseling and education

* See Guideline for other management strategies.

Figure 3
Frequency and Composition of Evaluation and Management Visits for Blepharitis*

Type of Disorder	Frequency of Examination	History	Exernal Evaluation/ Slit-Lamp Biomicroscopy	Management Plan*[*]
Seborrheic blepharitis	Weekly until stable, then p.r.n.	Yes	Yes	Lid hygiene t.i.d. until improved, then daily; Patient counseling and education
Staphylococcal blepharitis	Twice a week until cleared, then p.r.n.	Yes	Yes	Antibiotic or antibiotic/ steroid ung. h.s. to t.i.d.; tear supplements p.r.n.; steroid gtt. or ung. if infiltrates; lid hygiene t.i.d. until improved, then q.d.; Patient counseling and education
Seborrheic/ staphylococcal blepharitis	Twice a week until controlled, then q. 6 mo or p.r.n.	Yes	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
Meibomian seborrheic blepharitis	Twice a week until stable, then as part of preventive care	Yes	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
Seborrheic blepharitis with secondary meibomianitis	Twice a week until stable (up to 8 wk), then as part of preventive care	Yes	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
Meibomian keratoconjunc-tivitis	Twice a week until stable (up to 2 wk) then as part of preventive care	Yes	Yes	Lid hygiene; antibiotic or antibiotic/ steroid ung. h.s. to t.i.d.; oral tetracycline or doxycycline (taper); Patient counseling and education

[*] See Guideline for other management strategies.

Figure 4
ICD-9-CM Classification of Dry Eye and Blepharitis

Inflammation of eyelids	373
Blepharitis *Excludes: blepharoconjunctivitis (372.20-372.22)*	373.0
Blepharitis, unspecified	373.00
Ulcerative blepharitis	373.01
Squamous blepharitis	373.02
Hordeolum and other deep inflammation of eyelid	373.1
Hordeolum externum Hordeolum NOS Stye	373.11
Hordeolum internum Infection of meibomian gland	373.12
Abscess of eyelid Furuncle of eyelid	373.13
Chalazion Meibomian (gland) cyst *Excludes: infected meibomian gland (373.12)*	373.2
Noninfectious dermatoses of eyelid	373.3
Eczematous dermatitis of eyelid	373.31
Parasitic infestation of eyelid *Code first underlying disease, as:* pediculosis (132.9) demodex follicularum (133.8)	373.6

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 Dry eye syndrome	375.15
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 Keratoconjunctivitis sicca Sjögren disease	710.2

Abbreviations of Commonly Used Terms

BUT	-	Breakup time
DE	-	Dry eye
EDTA	-	Ethylenediaminetetraacetic acid
ELISA	-	Enzyme-linked immunosorbent assay
gtt.	-	Drops
h.s.	-	Bedtime
HRT	-	Hormone replacement therapy
KCS	-	Keratoconjunctivitis sicca
MGD	-	Meibomian gland dysfunction
mm	-	Millimeters
NIBUT	-	Noninvasive breakup time
OCP	-	Ocular cicatricial pemphigoid
POTF	-	Preocular tear film
p.r.n.	-	As necessary
q.	-	Every
q.d.	-	Daily
SS	-	Sjögren syndrome
SJS	-	Stevens-Johnson syndrome
t.i.d.	-	Three times per day
TLR	-	Total lysozyme reactivity
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.

Dry eye A group of anterior segment eye conditions manifested by a deficiency of the preocular tear film.

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

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

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

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.

Ocular surface disorder Any condition that reduces the production, alters the composition, or impedes the distribution of the preocular tear film.

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.

[redacted]

Schirmer test Measurement 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 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.

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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