

Chapter 6

Examination of the Lids

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Key Concepts

- An anatomically and physiologically normal eyelid is vital in maintaining the health of the eye.
- Meticulous examination of the tear film yields valuable information in the diagnosis and treatment of dry eye.
- Anterior eyelid examination may reveal trichiasis, an often frustrating malady that may result in severe symptoms, chronic inflammation, and corneal scarring.
- Examination of the posterior eyelid may reveal significant meibomian gland dysfunction (MGD) which can alter the mucocutaneous junction.
- Meibomian gland expression is an important part of the eyelid examination and is helpful in distinguishing seborrheic from obstructive MGD.
- Meibography provides information on meibomian gland structure and may be a valuable clinical tool in the treatment of MGD.

General Principles

The health of the ocular surface is dependent on an adequately positioned and properly functioning eyelid. A systematic approach such as that outlined in [Box 6.1](#) is helpful but should be modified as necessary.

History of the Patient

Symptoms of eyelid disease may be vague and nonspecific. While history of disease onset, duration, severity, exacerbation, localization, and previous treatments is being obtained, it is helpful to observe unconscious behaviors such as eye rubbing, scratching, or wiping away excess tears. These behaviors may be more indicative of the true malady, especially if the patient is having difficulty verbalizing a chief complaint. Furthermore, important observations on orbicularis oculi function can be made by observing the strength and rate of blinking.

Dermatologic Examination

Examination of the facial and adnexal skin is best done with fairly bright, diffuse, indirect light. Dark exam rooms and harsh lighting may distort the color and translucency of tissues. Many patients are unaware of dermatologic conditions that may be clearly apparent to clinicians, thus clinical photos are helpful educational tools.

Many conditions affect the periorbital skin—focal or diffuse, local or systemic, congenital, infectious, inflammatory, and neoplastic. The full breadth of the discussion is beyond the scope of this chapter, but several entities are discussed below.

Contact dermatitis of the eyelids is quite common and is associated with other ocular allergies.¹ The periocular skin may be erythematous, edematous, and scaly. A history of lotions, creams, topical medications, or other exacerbants should be sought. Atopic dermatitis can result in keratoconjunctivitis and may also present with thickened, scaly, erythematous, and fissured periocular skin. Patients may be aware of lesions elsewhere on their body, but may not have associated their dermatitis with their eye condition.

Rosacea is a common dermatologic condition that affects up to 10% of the population and most commonly in those of northern European origin. Rosacea dermatitis is characterized by malar flushing, telangiectasias, papules, pustules, sebaceous gland hypertrophy, and rhinophyma.

Bacterial infections may be focal (hordeola, chalazia), diffuse (preseptal cellulitis), or potentially fatal (orbital cellulitis). Cutaneous malignancies are commonly seen on the periorbital skin, accounting for upwards of 18% of all eyelid lesions.² Basal cell carcinoma is by far the most frequent (86%), followed by squamous cell carcinoma (7%) and sebaceous carcinoma (3%).²

Eyelid Position

Alteration of eyelid position and function can lead to exposure keratopathy, which may go unnoticed by patients.³ Evaluation of eyelid position starts with the measurement of margin reflex distance (MRD). MRD1 is the distance from the central light reflex, congruent with the visual axis, to the upper eyelid margin. Conversely, MRD2 is the distance from

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Box 6.1 A recommended order for examination of the eyelids

- Take history and observe patient's unconscious behavior and habits.
- Examine face and eyelids in ambient lighting.
- Examine tear meniscus and puncta with slit lamp prior to administration of drops or dyes.
- Examine anterior and posterior eyelid.
- Express the meibomian glands.
- Re-examine mechanical properties of the lids.
- Instill dye (typically fluorescein, also lissamine green or rose Bengal).
- Use slit lamp again to identify the mucocutaneous junction and its position relative to the meibomian gland orifices.
- Consider imaging studies as appropriate (typically for research purposes).

the central light reflex to the lower eyelid margin (Fig. 6.1). Together, they comprise the interpalpebral fissure (IPF) height. Measurement of MRD along with IPF provides a more accurate clinical picture than measurement of IPF alone (Fig. 6.1). Eyes with a larger IPF have a greater surface area. Because tear evaporation rate is correlated to surface area, patients with a larger IPF are more susceptible to dry eye symptoms.

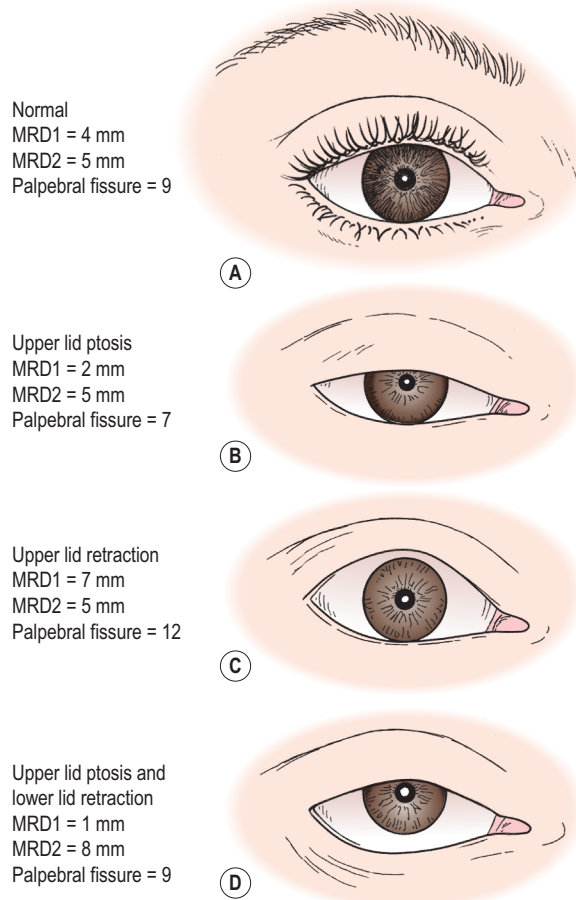
Both eyelid malposition and decreased force of contracture contribute to lagophthalmos. Forceful lid closure on exam may mask subtle degrees of incomplete lid closure. In these instances, it may be helpful to wait for one minute with the lids closed to mitigate forced lid closure.

Normal eyelid position is dependent on appropriate horizontal and vertical tension mediated by the lateral canthal tendon and lower eyelid retractors, respectively. Pulling the lid directly away from the ocular surface tests displacement, while pulling the lid inferiorly tests the ability of the lid to “snap back” into position. Although most abnormalities of lid tension are due to increased laxity, occasionally abnormalities due to increased tension are seen, such as superior limbic keratoconjunctivitis.

Increased horizontal laxity predisposes the lid to involutional ectropion (Fig. 6.2). Concomitant vertical laxity (via dehiscence of the lower lid retractors) predisposes the lid to involutional entropion (Fig. 6.3) as contraction of the pretarsal orbicularis oculi fibers forces the lower lid margin inward. In the absence of horizontal laxity, spastic entropion can occur due to vigorous contraction of the pretarsal orbicularis. The examination for ectropion and entropion is important as symptoms are often nonspecific.⁴ Ectropion may present insidiously with tearing, redness, irritation, tear film abnormalities, dry eye, and conjunctival keratinization. Entropion may present more acutely with pain, foreign body sensation and photophobia due to ocular surface contact.

Floppy eyelid syndrome (FES) results in excessive eyelid elasticity and usually presents with mucous discharge,

Margin Reflex Distance (MRD)



*Note palpebral aperture measurement is the same for examples A and D.

Fig. 6.1 Measurement of margin reflex distance (MRD1 and MRD2) along with the interpalpebral fissure (IPF) provides a much clearer view of the clinical picture. Note how IPF is equal in examples A and D. Whereas A is “normal,” the eye depicted in D presents with upper lid ptosis and lower lid retraction. (Courtesy of Jeffrey A. Nerad MD. From Nerad JA, Techniques in Ophthalmic Plastic Surgery, 2010, Elsevier Inc. Page 31. Figure 2.5.)

chronic irritation, papillary conjunctivitis, and keratopathy. Symptoms may be worse in the morning and patients may not be cognizant of any associated eyelid disease.⁵⁻⁸ FES patients are commonly obese and frequently report snoring or sleep apnea. Histological studies have demonstrated decreased tarsal elastin.^{10,11}

Markedly increased upper and lower eyelid laxity may be seen. Examination involves placing both thumbs on the superotemporal orbital rims and drawing the upper eyelid superotemporally. FES is diagnosed when the lid stretches excessively, often to the superior orbital rim, and the tarsal plate everts, exposing the palpebral conjunctiva. Treatment is aimed at correcting lid laxity and excising redundant tissue.



Fig. 6.2 Involutional ectropion of the lower eyelid due to increased horizontal lid laxity. Patients may present with tearing, redness, irritation, tear film abnormalities, and dry eye.

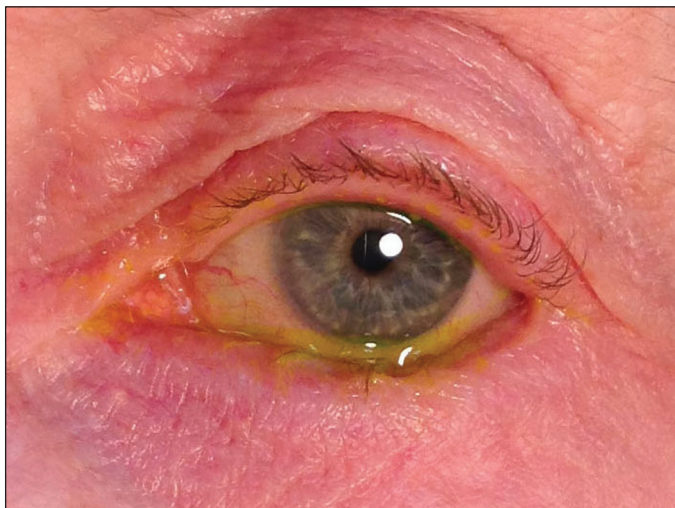


Fig. 6.3 Involutional entropion of the lower eyelid due to increased horizontal and vertical lid laxity. Symptoms of pain, foreign body sensation, and photophobia are typically more acute than those seen in ectropion.

Tear Meniscus and Puncta

The slit lamp exam should begin with the lamp off and just enough ambient light to measure the tear meniscus. Manipulation of the eyelids should be avoided. Slit lamp illumination can then be turned on to assess reflex tearing. Patients with a small tear meniscus who are unable to generate reflex tears are much more likely to have difficulty with dry eye.^{9,12,15–17} Foamy tears generally indicate meibomian gland dysfunction (MGD). An ocular surface interferometer (LipiView, TearScience Inc.) has been developed to quantify the tear film lipid layer thickness. Studies suggest that decreased lipid layer thickness may correlate with obstructive MGD.^{13,14}

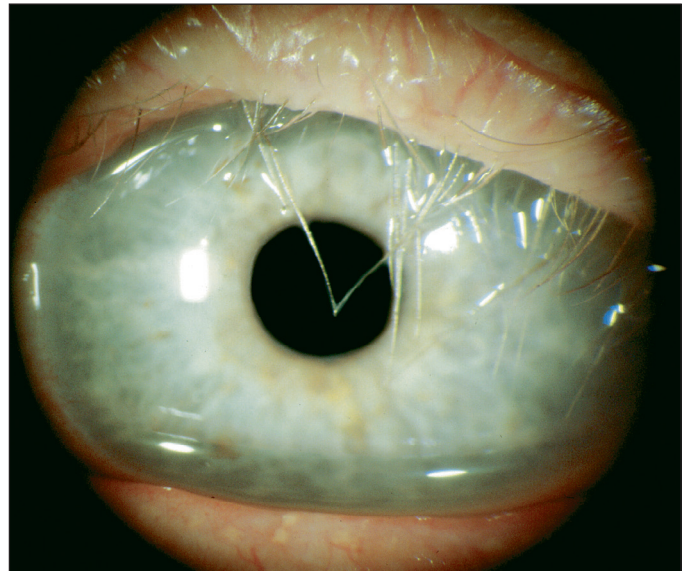


Fig. 6.4 Trichiasis from marginal entropion can be seen in blepharitis as well as other scarring diseases of the conjunctiva (mucous membrane pemphigoid, Stevens–Johnson syndrome, drug induced cicatrizing conjunctivitis, chemical or thermal injury).

Punctal position and patency are important for normal tear drainage. Punctal ectropion, even with a well-positioned central eyelid, prevents access to the nasolacrimal system and can lead to epiphora. Puncta may be scarred from a variety of conjunctival diseases (pemphigoid, chemical injury, blepharitis) or as a treatment for dry eye.^{18,19}

Anterior Eyelid

The anterior lamella comprises the skin and orbicularis oculi muscle. The eyelid should first be examined in ambient light with attention to color, transparency, induration, and other general characteristics. Lesions suspicious for a cutaneous malignancy should be examined with the biomicroscope. Potentially malignant characteristics such as a nodular pearly consistency, ulceration, induration, irregular borders, suspicious telangiectasias, madarosis, and loss of lid architecture are more easily seen with magnification.

Examination of the lashes is most readily performed with the biomicroscope. Length, number, and absence of lashes should be noted. Particular attention should be paid to the presence of trichiasis—posteriorly misdirected lashes (Fig. 6.4). This is commonly due to conditions that cause posterior lamella shortening, such as blepharitis, mucous membrane pemphigoid, Stevens–Johnson syndrome, chemical burns, and drug-induced cicatrizing conjunctivitis. Trauma is also a common cause, since soft tissue scarring distorts eyelash orientation. Less commonly this is due to epiblepharon (Fig. 6.5), a congenital condition in which redundant skin and muscle override the lid margin and force the eyelashes against the eye. Rarely is this due to distichiasis—growth of lashes from the meibomian gland orifices. Trichiasis may be a difficult problem that can lead to severe keratopathy, inflammation, and corneal scarring.²⁰

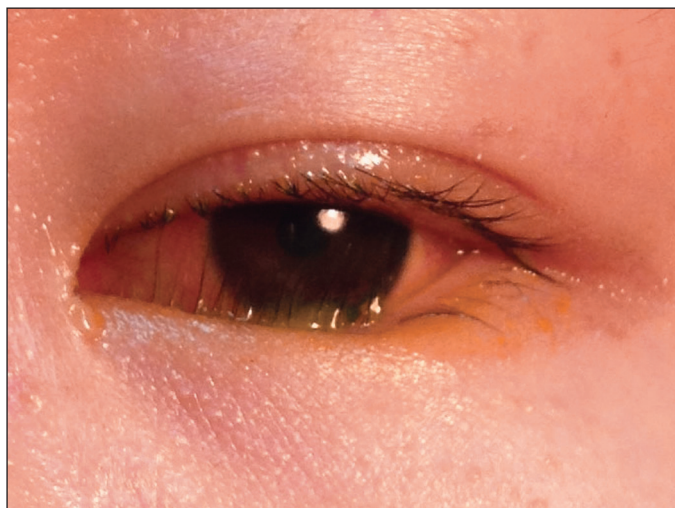


Fig. 6.5 Epiblepharon may cause trichiasis as an extra roll of skin and muscle overrides the lid margin and pushes the lashes towards the ocular surface.

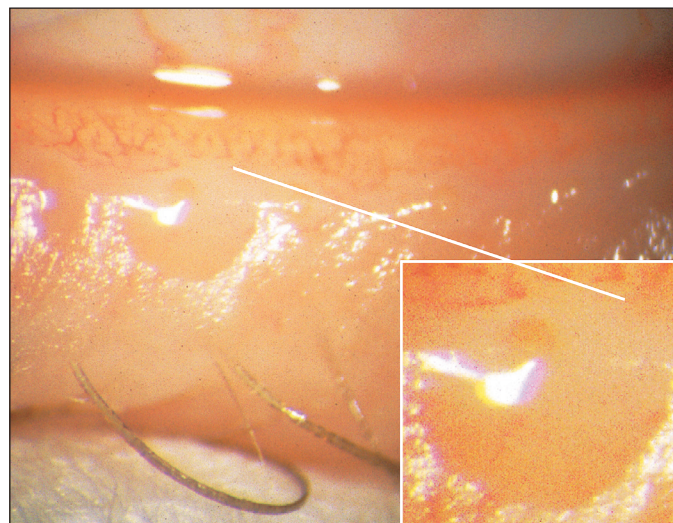


Fig. 6.7 Lipid expressed with digital pressure on an eyelid with seborrheic meibomian gland dysfunction reveals semitransparent liquid of increased volume.



Fig. 6.6 Vascularization and hypertrophy along the lid margin alters the normal contours and obscures landmarks.

The lashes should also be examined for signs of inflammation, infestation, or infection. Collarettes are mucous debris and desquamated epithelium adherent to the lash base and are a nonspecific sign of inflammation. *Phthirus pubis* are easily seen, whereas *Demodex* mites are smaller and more difficult to identify.^{21–24} Infectious processes may occur and are usually evident by swelling and pus at the lash base. Such hordeola of the lash follicles may be associated with a more generalized bacterial infection the eyelid.^{25,26}

Posterior Eyelid

The posterior lamella comprises the tarsus and conjunctiva. An uninflamed eyelid has a square edge and fine capillaries.²⁷ Inflammatory and infectious stimuli may induce rounding of the posterior lid margin.²⁷ Atrophy of the lid margin may result in hypervascularity as deeper vessels become visible. Although relatively nonspecific, these changes are often associated with obstructive MGD, rosacea, and infections (Fig. 6.6). Chalazia are indicative of obstructive MGD and

commonly cause lid scarring. Resolution of chalazia may result in notching and trichiasis.

Allergic processes may cause thickening of the conjunctiva and chronic changes to the lid margin. In severe cases, deep furrows develop in the skin and conjunctiva and may become secondarily infected or ulcerated.²⁸

The openings of the meibomian glands should be inspected carefully for signs of chronic disease. Periglandular atrophy renders the glands more evident as the lid margin recedes around the keratinized duct.²⁹ Hyperkeratinization of the ductal epithelium may partially or completely occlude the meibomian gland orifices.^{30–32} Partial occlusion from hyperkeratinization may augment obstruction from dry and hardened inflammatory debris and exacerbate obstructive MGD. Chronic aging changes also occur and are exacerbated by the effects of long-term obstructive MGD and dry eye.³³

Meibomian Gland Expression

Meibomian gland expression is an important part of the lid examination.^{33,34} With the patient in upgaze, firm sustained pressure (via a finger or cotton tipped applicator) is applied to the lower eyelid inferior to the lid margin until meibomian gland excreta is seen. Approximately 20–25 meibomian glands are typically present in the lower lid; two or three can be compressed at one time. The entire lid margin should be examined and the volume and viscosity of the excreta noted.

The volume can be recorded as the diameter of the lipid dome that forms after several seconds of pressure. Normal diameter is 0.5–0.7 mm. Diameters of 0.8 mm or larger are associated with increased lipid volume and are diagnostic of seborrheic MGD (Fig. 6.7). Decreased lipid volumes or inexpressible glands are associated with obstructive MGD. Meibomian gland lipid production may also be measured by evaluating the area of increased transparency on a paper

strip placed against the meibomian orifices, a technique termed “meibometry.”^{35,36}

The viscosity and opacity of the expressed meibomian lipid are important signs of eyelid disease. Normal lipid flows easily and remains transparent at body temperature. Seboreic MGD is associated with increased lipid opacity. Obstructive MGD demonstrates increased lipid viscosity and opacity. The most viscous lipid will emerge slowly like toothpaste, and will be totally opaque with a white or light yellow tint (Fig. 6.8).³³ Although typically associated with obstructive MGD, this may also be seen in rosacea.³⁴ The differences in the consistency of meibomian excreta have been attributed to variations in lipid composition.³⁷

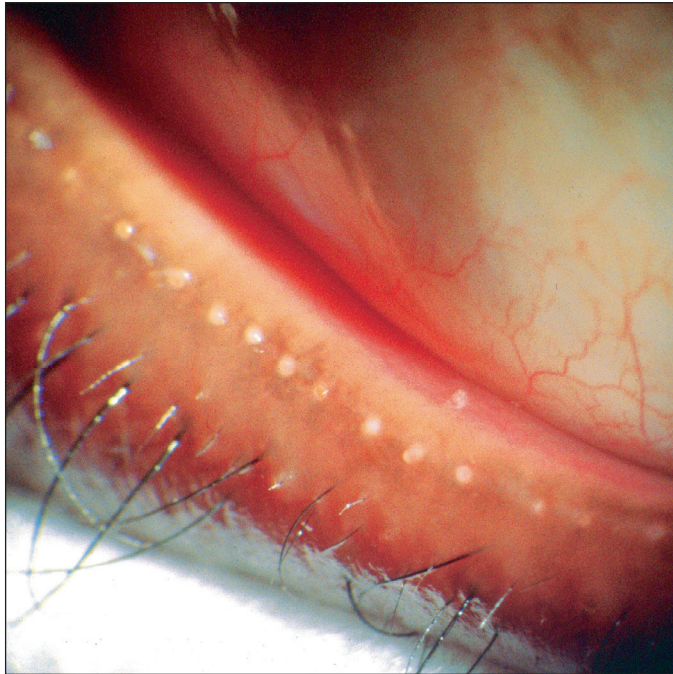


Fig. 6.8 Meibomian gland expression from a lid with obstructive meibomian dysfunction showing thickened and opaque lipid (toothpaste).

In cases of infection, meibomian glands may be tender and may yield pus on expression. This may be difficult to distinguish from staphylococcal blepharitis.³⁸ Although *Staphylococcus* and *Streptococcus* organisms are typically responsible, there is evidence that different strains of bacteria may be involved.³⁹ Culturing the eyelid for antibiotic sensitivity may be helpful, but because of the ubiquity of these organisms, the clinical significance is equivocal.^{39–41} The relative contribution of bacterial overgrowth, infection, bacterial toxins, and abnormal immune responses towards the development of blepharitis and meibomian gland dysfunction is a subject of controversy.^{42,43} In practice, this distinction is moot as current regimens employ strategies that reduce both infection and inflammation.^{42,44} It is important, however, to recognize the presence of meibomian gland disease to direct appropriate treatment.

Mucocutaneous Junction

The mucocutaneous junction is the confluence of the keratinized squamous epithelium of the skin and the nonkeratinized squamous epithelium of the conjunctiva. Normally just posterior to the meibomian gland orifices, visualization is aided by mucosal staining with lissamine green, rose Bengal or fluorescein (the Marx line).^{27,45,46} Anterior displacement of the mucocutaneous junction relative to the meibomian gland orifices may correlate with MGD, although this has been debated.^{27,45}

Meibomian Gland Imagery

Meibography is a noninvasive in vivo study of the gross and microscopic structure of meibomian glands that provides valuable adjunctive information in the evaluation and treatment of MGD. Studies on infrared (IR) photography of the meibomian glands date back to the late 1970s.⁴⁷ In 1994, Mathers et al. introduced video IR meibography with resolution approximately equal to that of IR film.^{47,48}

Contact meibography involves direct application of a light probe for eversion and transillumination of the eyelid (Fig. 6.9). Less invasive noncontact meibography techniques

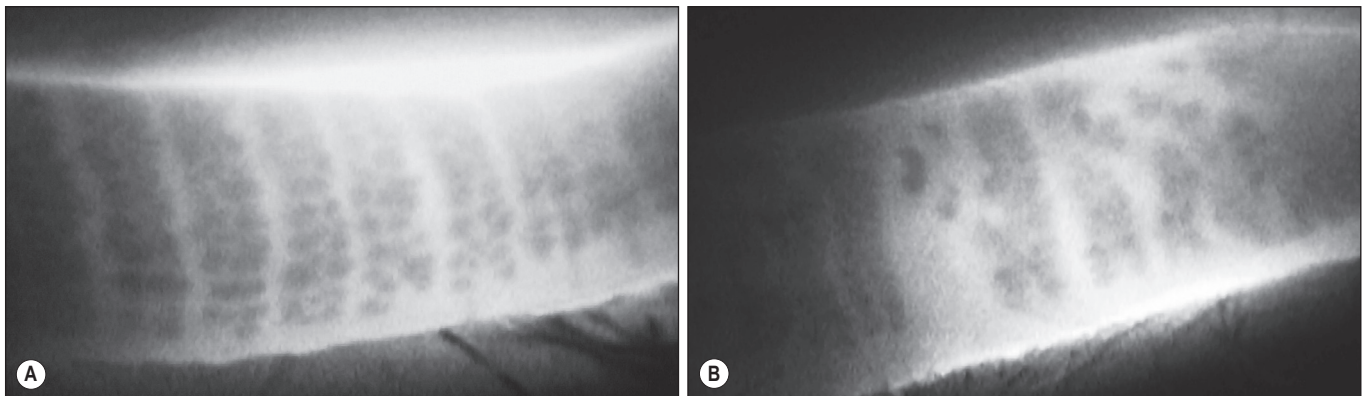


Fig. 6.9 Meibomian gland imagery. (A) Transillumination of a normal eyelid showing evenly spaced glands. (B) An infrared image with transillumination of the lower lid showing loss of glands.

have been developed involving biomicroscope-mounted and hand held devices.^{49,50}

Both techniques most commonly utilize IR meibography. Newer technologies (laser confocal microscopy [LCM] and optical coherence tomography [OCT]) provide valuable structural and volumetric information previously only available via ex vivo studies. In obstructive MGD, IR meibography demonstrates gland enlargement, duct dilation and gland dropout. In addition, LCM meibography demonstrates increased acinar unit diameter, decreased acinar unit density, periglandular inflammation, and fibrosis.⁵¹ OCT meibography also provides volumetric information but is still under development.

Meibography has great potential as a diagnostic tool but is limited by the lack of a widely accepted standardized grading system.⁵² The meiboscore and meibograde methods are two promising candidates. In the meiboscore method, meibographs of the upper and lower eyelid are quantified by the degree of glandular dropout. A score of 0 is given to a lid with no missing glands. Scores of 1 to 3 are assigned based on the relative area of gland loss: (1) for <33%, (2) for 33–66%, and (3) for >66%. The scores are summed by laterality for a total score of 0 to 6 per eye. Although methodical, this fails to account for changes in gland architecture that may precede dropout. These “pre-dropout” stages are better incorporated in the meibograde method. In this method, meibographs are assessed for gland distortion, shortening and dropout on a scale of 0 to 3 also based on the area involved (similar to the meiboscore method), resulting in a total score of 0 to 18 per eye.⁵³

References

- Fonacier L, Luchs J, Udell I. Ocular allergies. *Curr Allergy Asthma Rep* 2001;**1**(4):389–96.
- Deperez M, Uffer S. Clinicopathologic features of eyelid skin tumors. A retrospective study of 5504 cases and review of literature. *Am J Dermatopath* 2009;**31**(3):256–62.
- Cosar CB, Cohen EJ, Rapuano CJ, et al. Tarsorrhaphy: clinical experience from a cornea practice. *Cornea* 2001;**20**(8):787–91.
- Vallabhanath P, Carter SR. Ectropion and entropion. *Curr Opin Ophthalmol* 2000;**11**(5):345–51.
- Madjlessi F, Kluppel M, Sundmacher R. [Operation of the floppy eyelid. Symptomatic cases require surgical eyelid stabilization]. *Klin Monatsblatt Augenheilkde* 2000;**216**(3):148–51.
- Culbertson WW, Tseng SC. Corneal disorders in floppy eyelid syndrome. *Cornea* 1994;**13**(1):33–42.
- van den Bosch WA, Lemij HG. The lax eyelid syndrome. *Br J Ophthalmol* 1994;**78**(9):666–70.
- Boulton JE, Sullivan TJ. Floppy eyelid syndrome and mental retardation. *Ophthalmology* 2000;**107**(11):1989–91.
- Doughty MJ, Laiquzzaman M, Button NF. Video-assessment of tear meniscus height in elderly Caucasians and its relationship to the exposed ocular surface. *Curr Eye Res* 2001;**22**(6):420–6.
- Schlötzer-Schererhardt U, Stojkovic M, Hofmann-Rummelt C, et al. The pathogenesis of floppy eyelid syndrome: involvement of matrix metalloproteinases in elastic fiber degradation. *Ophthalmology* 2005;**112**(4):694–794.
- Netland PA, Sugrue SP, Albert DM, et al. Histopathologic features of the floppy eyelid syndrome. Involvement of tarsal elastin. *Ophthalmology* 1994;**101**(1):174–81.
- Yaylali V, Ozyurt C. Comparison of tear function tests and impression cytology with the ocular findings in acne rosacea. *Eur J Ophthalmol* 2002;**12**(1):11–17.
- Finis D, Pischel N, Schrader S, et al. Evaluation of lipid layer thickness measurement of the tear film as a diagnostic tool for Meibomian gland dysfunction. *Cornea* 2013;**32**(12):1549–53.
- Eom Y, Lee JS, Kang SY, et al. Correlation between quantitative measurements of tear film lipid layer thickness and meibomian gland loss in patients with obstructive meibomian gland dysfunction and normal controls. *Am J Ophthalmol* 2013;**155**(6):1104–10.
- Tomlinson A, Blades KJ, Pearce EI. What does the phenol red thread test actually measure? *Optom Vis Sci* 2001;**78**(3):142–6.
- Tsubota K, Kaido M, Yagi Y, et al. Diseases associated with ocular surface abnormalities: the importance of reflex tearing. *Br J Ophthalmol* 1999;**83**(1):89–91.
- Yokoi N, Kinoshita S, Bron AJ, et al. Tear meniscus changes during cotton thread and Schirmer testing. *Invest Ophthalmol Vis Sci* 2000;**41**(12):3748–53.
- McNab AA. Lacrimal canalicular obstruction associated with topical ocular medication. *Aust NZ J Ophthalmol* 1998;**26**(3):219–23.
- Sakol PJ. Tearing: lacrimal obstructions [Review]. *Pa Med* 1996;**99**(Suppl.):99–104.
- Lehman SS. Long-term ocular complication of Stevens–Johnson syndrome. *Clin Pediatr* 1999;**38**(7):425–7.
- Key JE. A comparative study of eyelid cleaning regimens in chronic blepharitis. *CLAO J* 1996;**22**(3):209–12.
- Demmler M, de Kaspar HM, Mohring C, et al. Blepharitis. Demodex folliculorum-associated pathogen spectrum and specific therapy. *Ophthalmologie* 1997;**94**(3):191–6.
- Junk AK, Lukacs A, Kampik A. Topical administration of metronidazole gel as an effective therapy alternative in chronic Demodex blepharitis – a case report. *Klin Monatsblatt Augenheilkd* 1998;**213**(1):48–50.
- Burkhart CN, Burkhart CG. Oral ivermectin therapy for phthiriasis palpebrum. *Arch Ophthalmol* 2000;**118**(1):134–5.
- Kiratli HK, Akar Y. Multiple recurrent hordeola associated with selective IgM deficiency. *J AAPOS* 2001;**5**(1):60–1.
- Lederman C, Miller M. Hordeola and chalazia. *Pediatr Rev* 1999;**20**(8):283–4.
- Hykin PG, Bron AJ. Age-related morphological changes in lid margin and meibomian gland anatomy. *Cornea* 1992;**11**(4):334–42.
- Inoue Y. Ocular infections in patients with atopic dermatitis. *Int Ophthalmol Clin* 2002;**42**(1):55–69.
- Bron AJ, Benjamin L, Snibson GR. Meibomian gland disease. Classification and grading of lid changes. *Eye* 1991;**5**(Pt 4):395–411.
- Jester JV, Rife L, Nii D, et al. In vivo biomicroscopy and photography of meibomian glands in a rabbit model of meibomian gland dysfunction. *Invest Ophthalmol Vis Sci* 1982;**22**(5):660–7.
- Robin JB, Jester JV, Nobe J, et al. In vivo transillumination biomicroscopy and photography of meibomian gland dysfunction. A clinical study. *Ophthalmology* 1985;**92**(10):1423–6.
- Jester JV, Rajagopalan S, Rodrigues M. Meibomian gland changes in the rhino (hrrhrrh) mouse. *Invest Ophthalmol Vis Sci* 1988;**29**(7):1190–4.
- Mathers WD, Shields WJ, Sachdev MS, et al. Meibomian gland dysfunction in chronic blepharitis. *Cornea* 1991;**10**(4):277–85.
- Mathers WD, Lane JA, Sutphin JE, et al. Model for ocular tear film function. *Cornea* 1996;**15**(2):110–19.
- Chew CK, Jansweijer C, Tiffany JM, et al. An instrument for quantifying meibomian lipid on the lid margin: the Meibometer. *Curr Eye Res* 1993;**12**(3):247–54.
- Chew CK, Hykin PG, Jansweijer C, et al. The casual level of meibomian lipids in humans. *Curr Eye Res* 1993;**12**(3):255–9.
- Shine WE, McCulley JP. Association of meibum oleic acid with meibomian seborrhea. *Cornea* 2000;**19**(1):72–4.
- Groden LR, Murphy B, Rodniti J, et al. Lid flora in blepharitis. *Cornea* 1991;**10**(1):50–3.
- Dougherty JM, McCulley JP. Bacterial lipases and chronic blepharitis. *Invest Ophthalmol Vis Sci* 1986;**27**(4):486–91.
- Dougherty JM, McCulley JP. Comparative bacteriology of chronic blepharitis. *Br J Ophthalmol* 1984;**68**(8):524–8.
- McCulley JP, Dougherty JM, Deneau DG. Classification of chronic blepharitis. *Ophthalmology* 1982;**89**(10):1173–80.
- Pflugfelder SC, Karpecki PM, Perez VL. Treatment of blepharitis: most recent clinical trials. *Ocul Surf* 2014;**12**(4):273–84.
- Jackson WB. Blepharitis: current strategies for diagnosis and management. *Can J Ophthalmol* 2008;**43**(2):170–9.
- Dougherty JM, McCulley JP, Silvany RE, et al. The role of tetracycline in chronic blepharitis. Inhibition of lipase production in staphylococci. *Invest Ophthalmol Vis Sci* 1991;**32**(11):2970–5.
- Yamaguchi M, Kutsuna M, Uno T, et al. Marx line: fluorescein staining line on the inner lid as indicator of meibomian gland function. *Am J Ophthalmol* 2006;**141**(4):669–75.
- Bron AJ, Yokoi N, Gaffney EA, et al. A solute gradient in the tear meniscus: I. A hypothesis to explain Marx's line. *Ocul Surf* 2011;**9**(2):70–91.
- Wise RJ, Sobel RK, Allen RC. Meibography: A review of techniques and technologies. *Saudi J Ophthalmol* 2012;**26**(4):349–56.

48. Mathers WD, Daley T, Verdick R. Video imaging of the meibomian gland [letter]. *Arch Ophthalmol* 1994;**112**(4):448–9.
49. Arita R, Itoh K, Inoue K, et al. Noncontact infrared meibography to document age-related changes of the meibomian glands in a normal population. *Ophthalmology* 2008;**115**(5):911–15.
50. Arita R, Itoh K, Maeda S, et al. A newly developed and noninvasive mobile pen-shaped meibography system. *Cornea* 2013;**32**(3):242–7.
51. Matsumoto Y, Sato E, Ibrahim O, et al. The application of in vivo laser confocal microscopy to the diagnosis and evaluation of meibomian gland dysfunction. *Mol Vis* 2008;**14**:1263–71.
52. Matsumoto Y, Shigeno Y, Sato EA, et al. The evaluation of the treatment response in obstructive meibomian gland disease by in vivo laser confocal microscopy. *Graefe's Arch Clin Exp Ophthalmol* 2009;**247**(6):821–9.
53. Call CB, Wise RF, Hansen MR, et al. In vivo examination of meibomian gland morphology in patients with facial nerve palsy using infrared meibography. *Ophthal Plast Reconstr Surg* 2012;**28**(6):396–400.