EYELIDS AND LACRIMAL SYSTEM*

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I. Eyelids

The eyelids protect the eye by preventing contact with foreign materials and by preventing excessive drying of the cornea and conjunctiva. The palpebral fissure must be wide enough to allow light to enter the pupil and should close sufficiently to provide protection and moisture to the globe. The lid contours and palpebral fissures should be symmetric to avoid cosmetic deformity.

A. Eyelid anatomy

The eyelids are lamellar structures covered on their outer surfaces by skin and on their inner surfaces by conjunctiva. Between the skin and conjunctiva are the fibrous tarsal plates, the orbital septum, the upper lid elevators (levator muscle, levator aponeurosis, and Müller muscle), and the lower lid retractors (inferior rectus fascia and inferior tarsal muscle). The levator muscle is innervated by the third cranial nerve, whereas Müller muscle and the inferior tarsal muscle are innervated by sympathetic nerves. The lids and palpebral fissures are maintained in a stable position by periosteal attachments provided by the medial and lateral canthal tendons. The palpebral fissure is closed by the orbicularis muscle, which is innervated by the seventh cranial nerve.

B. Congenital and developmental eyelid anomalies

1. **Ptosis.** Most congenital ptosis is caused by a deficiency of the striated fibers in the levator muscle. Many cases of congenital ptosis are associated with other developmental abnormalities such as blepharophimosis and epicanthus. Congenital ptosis will be discussed with other eyelid malpositions (see sec. I.C.1., below).

2. **Blepharophimosis and epicanthus.** Blepharophimosis is a generalized narrowing of the palpebral fissure. This abnormality is frequently associated with congenital ptosis and epicanthus. Epicanthus is a semilunar fold of skin that crosses the medial canthus. Blepharophimosis and epicanthus should usually be repaired prior to surgical correction of ptosis.

3. **Colobomas** are usually full-thickness defects in the medial portions of the upper lids. Colobomas are often associated with other congenital defects such as facial dermoids. Unless exposure keratopathy occurs, surgical repair of most colobomas can be delayed until the child is several years old.

4. **Ankyloblepharon** is an abnormal fusion of the upper and lower eyelid margins, usually near the lateral canthus. The fused lids may be surgically divided if the
attachment is cosmetically disfiguring.

5. **Ectropion and entropion** are both uncommon congenital disorders (see sec. D. and sec. E., below). Congenital ectropion is usually minimal and may be associated with blepharophimosis. Congenital entropion is quite unusual but is frequently confused with epiblepharon.

6. **Epiblepharon** is a relatively common condition in which a prominent skin fold is present in front of the tarsus, usually near the medial margin of the lower lid. The lashes may be rotated inward without actual rotation of the eyelid margin (entropion). Surgical correction is seldom required, because epiblepharon usually resolves spontaneously.

C.

Ptosis is a malposition of the upper eyelid in which the lid margin is abnormally low because of insufficient upper eyelid retraction. Evaluation of a patient with ptosis should include measurement of palpebral fissure heights, distance from the pupillary light reflex to the upper eyelid margin (marginal reflex distance and levator function, excursion of the upper eyelid margin from downgaze to upgaze, while fixating the brow in a stable position). Complete ptosis evaluation should also include assessment of the protective mechanisms of the globe, including Bell phenomenon, corneal sensation, and baseline tear production (Schirmer test). The positions of the eyelid folds and any abnormalities of the extraocular muscles should be documented. The type of ptosis should be carefully established by history, because the treatment of congenital ptosis is usually different from that for acquired ptosis. Both congenital and acquired ptosis must be distinguished from pseudoptosis, a condition in which the upper eyelid appears to be low but lid elevation is adequate. Pseudoptosis of the lid margin may be due to descent of the brows (brow ptosis) or excessive skin and fat of the upper eyelid mechanically lowering the upper eyelid margin (dermatochalasis).

1. **Congenital ptosis** is usually unilateral, although approximately one-fourth of the cases involve both upper eyelids. It may be associated with other abnormalities:
   b. **Marcus Gunn “jaw-winking” syndrome**, a synkinesis in which the ptotic eyelid is elevated with movement of the mandible.
   c. **Extraocular muscle palsies**, particularly those involving the superior rectus and inferior oblique muscles ipsilateral to the ptosis.
   d. **Treatment** of congenital ptosis usually requires resection of part of the weak levator muscle and aponeurosis, or suspension of the lids from the frontalis muscle (brow).

2. **Acquired ptosis** is frequently associated with good levator muscle function and may
be categorized according to etiology.

a. **Involuntary** ptosis is the most common ptosis encountered, often involves both upper lids of older patients, and may occur following cataract extraction. This is the most common form of acquired ptosis and is caused by stretching of the levator aponeurosis or disinsertion of the levator muscle from its insertion onto the tarsus.

b. **Myogenic** ptosis may be associated with a variety of muscular disorders, including myasthenia gravis, oculopharyngeal muscular dystrophy, and progressive external ophthalmoplegia.

c. **Neurogenic** ptosis can be caused by deficient innervation of the third cranial nerve to the levator muscle or deficient sympathetic innervation to Müller muscle.

d. **Traumatic** ptosis may result from lacerations of the levator muscle or aponeurosis and may sometimes follow severe blunt trauma with eyelid edema.

e. **Mechanical** ptosis may be associated with lid tumors such as neurofibromas and may result from scars or foreign bodies.

f. **Treatment** of acquired ptosis involves correcting the cause of the ptosis, typically repair of the levator aponeurosis (tendon) if possible. If levator function is not good, this involves a larger resection of the levator aponeurosis, including the distal levator muscle; in more severe cases, the levator aponeurosis may be suspended from the frontalis muscle if levator function is poor.

3. **Pseudoptosis** is a condition in which the upper eyelid appears to be abnormally low without insufficiency of the lid retractors. Causes of pseudoptosis include:

   a. Epicanthus and facial asymmetry.
   
   b. Excessive upper eyelid skin, as found in dermatochalasis (very common).
   
   c. Contralateral palpebral fissure widening.
   
   d. Palpebral fissure narrowing associated with adduction in Duane retraction syndrome.
   
   e. Hypertropia or contralateral hypotropia.
   
   f. Enophthalmos or contralateral exophthalmos.

**D. Ectropion**

Ectropion is a malposition of the eyelid in which the lid margin is rotated away from the globe. The lower lid is involved much more commonly than the upper lid. Ectropion sometimes leads to exposure keratopathy and conjunctival
hypertrophy. Tearing may result from eversion of the lacrimal punctum if the ectropion involves the medial lid.

1. **Congenital ectropion** is quite uncommon, although it may be found with blepharophimosis. Treatment is rarely required because the eversion is usually minimal.

2. **Acquired ectropion** is categorized on the basis of etiology.
   
   a. **Involutional** ectropion is relatively common and is a frequent cause of tearing *(epiphora)*. This abnormality is caused by attenuation of the lower eyelid retractors, the orbicularis muscle, and the canthal tendons. Treatment involves horizontal eyelid shortening and canthal suspension. If punctal eversion is the most significant feature, conjunctival shortening and a punctoplasty may reduce tearing.

   b. **Paralytic** ectropion usually results from seventh nerve injury, with resulting drooping of the lower lid and widening of the palpebral fissure. Treatment may require tarsorrhaphy, horizontal lid shortening, canthoplasty, or suspension of the upper cheek. A flaccid brow and upper lid may be surgically elevated if they partially cover the palpebral fissure.

   c. **Mechanical** ectropion may be caused by abnormalities that push or pull the lid away from the eye. Treatment usually involves treatment of the underlying abnormality.

   d. **Cicatricial** ectropion occurs when the anterior lamella of the eyelid (skin and orbicularis muscle) is contracted by a variety of possible causes (e.g., burns, tissue loss, traumatic scars, or inflammation). Linear and circumscribed scars may respond to massage or relaxing operations. More extensive cicatricial ectropion usually requires a skin graft.

**E. Entropion**

Entropion is a malposition of the eyelid in which the lid margin is rotated toward the globe. Entropion is functionally important because inturned lid margins may damage the cornea and produce keratitis or ulceration. Related conditions that should be differentiated from entropion are epiblepharon, trichiasis, and distichiasis.

1. **Congenital entropion** is rare and is usually associated with other abnormalities such as tarsal hypoplasia or microphthalmia. Congenital entropion may be confused with epiblepharon, a mild deformity that usually resolves spontaneously. Depending on severity, this condition may be treated similarly to acquired entropion.

2. **Acquired entropion** is a common disorder that is usually either involutional, as a result of aging, or cicatricial, resulting from tarsconjunctival shrinkage.
a. **Involutional** entropion usually involves the lower lid and is caused by degenerative changes similar to those that cause involutional ectropion. With aging, atrophy of the orbital tissues can lead to a relative enophthalmos and a tendency for inward rotation of already attenuated eyelid structures. Treatment should be directed toward correction of those abnormalities that are most prominent. Penetrating pretarsal cautery or three Quickert lid eversion sutures may temporarily correct a moderate entropion, but may be followed by recurrence. Many operations have been devised for correction of involutional entropion. The most physiologic procedures are those that restore the action of attenuated eyelid retractors and that tighten a lax lower lid.

b. **Spastic** entropion is a temporary or intermittent accentuation of involutional changes caused by irritation and vigorous lid closure. Treatment can be directed toward removing the cause of irritation or treating the underlying involutional abnormalities.

c. **Cicatricial** entropion is usually the result of tarsoconjunctival shrinkage. This may be caused by a wide variety of disorders, including trachoma, Stevens-Johnson syndrome, pemphigus, ocular pemphigoid, and mechanical, thermal, or chemical injury. Cicatricial changes are often accompanied by trichiasis, reduced tear production, mucosal epithelialization, and punctal occlusion. Treatment may consist of marginal rotation of the lid margin and grafts of mucosa or other tissue to replace contracted tarsus and conjunctiva.

F. **Blepharospasm**

Blepharospasm is a disorder of unknown cause that involves involuntary closure of the eyelids. The severity of this closure ranges from mild increased frequency of blinking to severe spasms that completely occlude the eyes. Essential blepharospasm, in which the eyelids are chiefly involved, is distinguished from conditions such as Meige's disease, in which lower face and neck muscles also spasm, and hemifacial spasm, which may be caused by facial nerve compression. Excision of facial muscles (myectomy) and nerves (neurectomy) has been used in the past as treatment for severe cases of essential blepharospasm. Currently, **botulinum toxin injections** are considered the most effective treatment for the majority of patients with this condition. Multiple small amounts of the toxin are injected into the muscles around the eyelids. Blepharospasm is usually relieved within several days, but the effect is temporary and additional injections are often necessary within 3 months. Side effects include ptosis, double vision, and drying of the eyes from inability to close the lids (see Chapter 12, sec. XII.).

G. **Eyelash disorders**

The eyelashes normally emerge from the lid margin anterior to the mucocutaneous junction and are directed away from the surface of the eye. In a number of conditions the lashes
either arise abnormally posterior or are directed toward the eye (Table 3.1).

**Table 3.1. DIFFERENTIAL DIAGNOSIS OF EYELASHES DIRECTED AGAINST THE GLOBE**

1. **Distichiasis** is an abnormality in which extra lashes arise from the lid margin behind the mucocutaneous junction, frequently from the meibomian gland orifices. These lashes are usually small and cause few symptoms, but occasionally they may produce severe corneal damage. Treatment is not required in mild cases. If the eye is being injured, the lashes may be destroyed with cryotherapy, electrolysis, or surgery.

2. **Trichiasis** is an acquired condition in which the lashes are directed posteriorly, toward the surface of the eye. Although the lid margin is not necessarily inverted, trichiasis can occur in association with entropion. Trichiasis often accompanies chronic blepharoconjunctivitis or cicatricial conjunctivitis. Electrolysis is usually effective in treating focal areas of abnormal lashes. Surgical excision of the lashes and replacement with a mucous membrane graft may be used to treat severe cases of trichiasis in which retention of some normal lashes is desired.

**H. Eyelid tumors**

The first priority in treating any tumor of the eyelids is to establish the diagnosis. Except for inflammatory lesions such as chalazia, any tissue removed from the eyelids should be examined histologically. Treatments such as cauterization that destroy tissue and make histologic evaluation impossible should be avoided. In the case of a small skin lesion, excisional biopsy can be performed with removal of all clinical evidence of the tumor. If a benign tumor is suspected, the margins of the incision may be within 1 or 2 mm of the lesion. If a malignant tumor is suspected, 3 to 5 mm of clinically uninvolved tissue should be removed with the lesion. When a tumor is histologically malignant, the pathologist should be asked to examine all margins of the specimen, including the deep surface, for evidence of tumor that has been cut across. If the tumor is found to have been transected, additional tissue should usually be removed. The management of each kind of tumor should obviously depend on its individual growth characteristics and on the requirements for reconstructing functional eyelids. The most common benign and malignant tumors of the eyelids are listed below:

<table>
<thead>
<tr>
<th>Benign tumors</th>
<th>Malignant tumors</th>
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<tr>
<td>Keratoses</td>
<td>Basal cell</td>
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1. **Basal cell carcinomas** are by far the most common malignant tumors of the eyelids. These tumors most frequently arise on the sun-exposed lower lids and medial canthal areas. Most basal cell carcinomas are nodular with a pearly surface and telangiectatic vessels. Some are flat and leathery and are described as morpheaform or sclerosing basal cell carcinomas. This latter type of tumor is particularly likely to be infiltrative. Treatment usually consists of histologically confirmed surgical excision. Although cryotherapy and radiation are sometimes used to destroy basal cell carcinomas, their use does not provide microscopic confirmation of complete tumor excision. Metastatic spread of basal cell carcinomas is exceedingly uncommon. Concern that the tumors may invade into the orbit occur when the tumor is located adjacent to the bony orbital rim in the region of the medial or lateral canthi, hence it is critical that the deep margins are carefully assessed during the primary excision of these tumors to ensure that the entire tumor is excised.

2. **Squamous cell carcinomas** usually arise among older patients and commonly develop from actinic keratoses. Differentiation from benign keratoacanthomas is sometimes difficult. Treatment of these potentially metastasizing tumors should consist of wide surgical excision that is histologically confirmed to be adequate by careful examination of all margins.

3. **Malignant melanomas** of the eyelids are uncommon and may extend from melanomas of the conjunctiva. These potentially metastasizing tumors may occur de novo or may evolve from preexisting nevi or from areas of acquired melanosis. The histologic feature of greatest prognostic importance seems to be the tendency toward vertical growth (deep invasion below the epithelial surface). Conjunctival melanomas that grow deeply tend to be more likely to metastasize than those that grow peripherally. Complete wide excision is the most common therapy, and radical surgery such as exenteration may be advised if there is extensive conjunctival disease or evidence of orbital invasion.

4. **Sebaceous cell carcinomas** arise most often from the meibomian glands within the tarsal plates. These highly malignant tumors may also develop from the sebaceous glands of the eyelashes, the caruncle, and the eyebrow. Their growth may mimic chalazia and the contents of a presumably recurrent chalazion should always be examined histologically. Fat stains should be performed on fresh tissue whenever sebaceous carcinoma is suspected.
These tumors may be multifocal and can spread peripherally by intraepithelial or pagetoid growth. Metastasis and orbital extension frequently occur. Treatment consists of wide surgical excision, with histologic confirmation of complete removal. Because of insidious intraepithelial growth, multiple areas of excision or exenteration may be required.

I. Eyelid inflammation and degeneration

The most common inflammations of the eyelids are those involving the lashes and the lid margins (blepharitis) and those that arise within the meibomian glands as an acute lesion (hordeolum) or evolve into a chronic lesion (chalazion). Diffuse inflammatory eyelid atrophy (blepharochalasis) should be distinguished from involutional degeneration of the eyelids (dermatochalasis).

1. Blepharitis is the most common inflammation of the eyelid and may present as anterior, posterior, or both forms.

   a. Anterior blepharitis (AB) involves the lashes and anterior lid margin. Seborrheic AB shows inflammation and flaking of the skin with oily scruff (cuffing) at the base of the lashes, and often lash loss or misdirected growth. Eczematous AB has dry, roughened, flaking skin with some inflammation, whereas bacterial AB has inflammatory, purulent changes and discharge. The meibomian glands and tarsus are not primarily involved.

   b. Posterior blepharitis (PB) is frequently associated with acne rosacea. Malar and nasal bridge skin findings are tiny telangiectasia and often slightly raised, rough macules. Lid margin telangiectasia is common. Meibomitis causes stenosis of the orifices with bloating of the glands beneath the inferior tarsal conjunctiva, injection, and cyst formation. Early on, oil can still be expressed from the glands by pressure on the lids, but chronic meibomitis causes scarring and lid thickening with lash loss, loss of tear film surface oil, and tear film destabilization.

   c. Treatment of AB eczematous and seborrheic disease is steroid antibiotic ointment and lid hygiene with warm compresses bid to tid and baby shampoo scrubs with lathered fingertips daily. In addition, antiseborrheic shampoo applied to the scalp qd is useful for seborrhea. Therapy is essentially life-long. Bacterial AB is treated with topical antibiotic ointment or drops (bacitracin ointment, Polytrim, drops bid to qid for 10 days) along with warm compresses. Two or three forms of AB may occur together; adjust treatment accordingly.

   d. Treatment of PB includes lid hygiene plus doxycycline or minocycline 100 mg p.o. qd for 3 weeks, then 50 to 100 mg p.o. qd for 3 months or indefinitely if needed. For pregnant women or children under 10 ears, erythromycin 250 mg p.o. qd may be used instead. The facial rosacea will benefit from the antibiotic, but may need additional treatment with metronidazole 0.75% cream or ointment bid for 6 months to the involved facial area (see Chapter 5, sec.

2. **Hordeolum** is a focal acute infection arising within the meibomian glands or other glands at the eyelid margins. These lesions are commonly caused by *Staphylococcus* and usually respond to conservative therapy. Treatment with warm, moist compresses and topical antibiotics usually produces resolution of the inflammation (see Chapter 5, sec. III.B. and sec. III.D.).

3. **Chalazion** is a focal chronic inflammation of a meibomian gland. It is a common disorder that may occur as the result of a chronic hordeolum. Treatment with warm compresses is effective in most cases, with topical antibiotics used to prevent secondary spread of infection. If the lesion fails to resolve with compresses, incision and curettage through the conjunctiva may be necessary in some instances to facilitate resolution of the lesion. Recurrent lesions should be examined histologically because of the possibility that a malignancy such as sebaceous cell carcinoma may be present.

4. **Dermatochalasis** is a redundancy of the skin of the eyelids that is often accompanied by herniation of fat through the orbital septum. This condition usually occurs as an involutional change among older or middle-aged people. A familial predisposition is common, although there is no sex predilection. The excessive skin may cause a pseudoptosis, although a true involutional ptosis may be present. Treatment is blepharoplasty with optional fat excision and repair of acquired ptosis, if present.

5. **Blepharochalasis**, unlike dermatochalasis, is a very rare condition that results from repeated idiopathic episodes of eyelid edema and inflammation. These acute attacks occur most frequently among younger individuals and are more common among women than men. The inflammation often results in wrinkling of the skin, atrophy of fat, and ptosis. Treatment of acute attacks is usually not necessarily because they are self-limited, although systemic steroids may be of some value. The chronic atrophic changes may respond to blepharoplasty and to repair of the acquired ptosis.

**J. Eyelid trauma**

Injuries to the globe may be relatively occult and may be overshadowed by obvious lid damage. Therefore, the eye should be carefully examined and the visual acuity should be documented before injured eyelids are treated. During lid repairs the globe should be protected to prevent additional injury. The possible occurrence of orbital fractures or of embedded foreign bodies should always be considered. If necessary, x-rays and computed tomography scan may be used to evaluate these possibilities.

1. **Burns** of the lids may be chemical, thermal, or electrical (see Chapter 2, sec. I. and sec. II.). The first priority is to treat associated ocular injury. Lid retraction and cicatricial ectropion may result from burns and require skin grafting or other surgical
repair after a period of time.

2. **Lacerations** of the eyelids may be repaired primarily even as long as 12 to 24 hours after injury, because of their rich vascular supply and the rarity of infections. Treatment should involve minimum débridement and retention of as much tissue as possible. The wound should be explored to rule out foreign bodies, and all tissues should be replaced in their anatomic positions. The tarsus should be separately approximated by sutures tied away from the surface of the eye. Tissue loss can be replaced by lid advancement or a skin graft. When the deep tissues of the upper eyelid are involved by a laceration, the levator aponeurosis should be examined and repaired if it is damaged.

### II. Lacrimal system

The tear film is composed of mucin, oil, and watery lacrimal fluid. The mucin component is the product of the conjunctival goblet cells, whereas oil is secreted by the sebaceous meibomian glands within the tarsal plates and by the glands of Zeis and Moll, which lie near the lid margins. The bulk of the tear film is made up of lacrimal fluid from the main lacrimal gland and from the accessory lacrimal glands of Krause and Wolfring (see Chapter 1, sec. II.J. and sec. II.K. for the clinical tests used to measure tear film adequacy, including tests of secretion). The lacrimal secretions are distributed over the surface of the eye by gravity, capillary action, and the eyelids. Tears leave the eye by evaporation and by flow through the lacrimal excretory system, composed of the puncta, the canaliculi, the lacrimal sac, and the nasolacrimal duct.

#### A. Lacrimal excretory anatomy and physiology

1. **Puncta.** The puncta are small openings approximately 0.3 mm in diameter that lie at the edge of each eyelid. Each upper punctum is approximately 6 mm temporal to the medial canthal angle among adults; the lower punctum is slightly more temporal than the upper.

2. **Canaliculi.** The canaliculi are composed of short vertical segments that begin at the puncta, and horizontal segments approximately 8 mm long that empty into the lacrimal sac. The upper and lower canaliculi usually empty into a common canaliculus (sinus of Maier) before communicating with the sac. In approximately 10% of patients, the upper and lower canaliculi open separately into the lacrimal sac.

3. **Lacrimal sac.** The sac is a cystic structure lined with columnar epithelium. The medial canthal tendon passes in front of the sac, and the lacrimal diaphragm and Horner muscle (the deep head of the pretarsal muscle) pass behind the sac.

4. **Nasolacrimal duct.** This duct is a vertically oriented tube that is continuous with the lower end of the sac. It passes through the bony nasolacrimal canal to drain into the nose beneath the inferior turbinate via the nasal ostium. At the junction between the duct and the nasal fossa, a mucosal fold (valve of Hasner) may
be found.

5. Lacrimal excretion. Passage of tear fluid from the surface of the eye through the excretory system depends on the anatomic patency of each segment of the pathways. For fluid to enter the system, the puncta must be in anatomic apposition to the tear film meniscus on the surface of the eye. Entrance of tears into the canaliculi is aided by capillary action. Movement of fluid through the pathways is aided by a lacrimal pump mechanism. This pumping action results from eyelid blinking, during which the muscles and diaphragm around the lacrimal sac move to create internal pressure changes and propel tears. The patency and function of the excretory system can be evaluated by dye tests, irrigation, and dacryocystography.

B. Congenital and developmental lacrimal anomalies

1. Nasolacrimal duct obstruction is the most common congenital abnormality of the lacrimal system. As many as 30% of newborn infants are believed to have closure of the duct at birth. This obstruction is usually located at the nasal mucoperiosteum, near the site of the valve of Hasner. In most cases, this obstruction is transient, and patency occurs within 3 weeks of birth. Tears and mucus may accumulate in the lacrimal sac, causing distention of the sac and sometimes leading to dacryocystitis. Treatment of a distended sac in an infant consists of sac massage and application of topical antibiotics. In the absence of an acute infection, if the obstruction is not relieved by the first birthday, nasolacrimal duct probing and irrigation is usually curative.

2. Punctal and canalicular abnormalities include absence, stenosis, duplication, and fistulization. Imperforate or absent puncta can sometimes be opened by a sharply pointed dilator and a microscope. Fistulas can be surgically excised. Absent canaliculi can be bypassed by performing a conjunctivodacryocystorhinostomy with insertion of a glass or plastic tube into the nose.

3. Diverticula may arise from the lacrimal sac, the canaliculi, or the nasolacrimal duct. These cystic outpouchings may accumulate fluid and therefore simulate a mucocele of the lacrimal sac. They may become infected and mimic dacryocystitis. Chronic epiphora, however, is usually not a prominent symptom of diverticula. Treatment is surgical excision.

C. Lacrimal sac tumors

Neoplasms of the lacrimal sac are unusual and therefore may go undiagnosed for a long time because they are confused with inflammations and other causes of nasolacrimal obstruction. Tumors of the sac typically cause epiphora with a subcutaneous mass superior to the medial canthal tendon. Blood may reflux from the puncta and saline irrigation may pass into the nose, despite a history suggestive of dacryocystitis. Squamous papillomas are the most common benign tumors of the sac, whereas epidermoid
carcinomas are the most frequent malignancies. Treatment of lacrimal sac tumors usually requires dacryocystectomy. Removal of the medial canthal tissues and adjacent bone may be necessary to eradicate malignant epithelial lesions. Lymphoid tumors frequently respond to radiation therapy.

D. Lacrimal inflammations and degenerations

Acquired obstructions of the lacrimal excretory system may result from infections, other inflammations, and involutional changes.

1. Dacryocystitis is an infection of the lacrimal sac that usually results from obstruction of the nasolacrimal duct. Dacryocystitis usually produces localized pain, edema, and erythema over the lacrimal sac. This clinical pattern must be distinguished from acute ethmoid sinusitis, although purulent discharge from the puncta almost always indicates an infection within the sac. Irrigation and probing should usually not be performed during an acute infection. This disorder usually responds to warm, moist compresses, together with topically and systemically administered antibiotics. A distended lacrimal sac should be incised and drained only if the infection does not respond to conservative therapy or if an abscess begins to point.

2. Lacrimal sac obstructions are uncommon and generally result from dacryoliths. Solid concretions within the sac may be caused by infection with Actinomyces israelii (Streptothrix). Although such infections sometimes respond to irrigation with antibiotics, the sac must frequently be opened and a dacryocystorhinostomy performed.

3. Nasolacrimal duct obstruction usually occurs among older individuals and is commonly idiopathic. Most such involutional cases are probably the result of mucosal degeneration with stenosis. The most common sequelae of duct obstruction are epiphora and a mucocele of the sac. Dacryocystitis often follows a chronic mucocele. Probing of obstructed nasolacrimal ducts among adults rarely restores patency. Partial obstruction may respond to intubation of the entire excretory system with Silastic tubing. A dacryocystorhinostomy may be considered in cases in which the canaliculi are patent. Such an operation is usually not indicated unless tearing and mucous discharge are extremely bothersome, or unless the patient suffers repeated attacks of dacryocystitis. It is possible that a duct obstruction may result from a mass within the nose. Therefore, the nasal fossa should always be examined before a dacryocystorhinostomy is performed.

4. Punctal and canalicular obstructions may occur in association with conjunctival disorders such as Stevens-Johnson syndrome, pemphigus, ocular pemphigoid, and mechanical, thermal, or chemical injury. Systemic chemotherapy (e.g., 5-fluorouracil or docetaxel [Taxotere]) that is preferentially concentrated in the tear film is caustic to the canalicular epithelium and may result in canalicular or punctal occlusion. Canaliculitis may sometimes result from infections caused by A. israelii. Stenotic or
obstructed puncta can be dilated and incised if necessary. If canalicular stenosis can be opened by probing, Silastic tubing can sometimes be passed through the entire excretory system into the nose to maintain patency. In cases of complete and irreversible punctal or canalicular stenosis when epiphora is severe, the obstruction can be bypassed by performing a conjunctivodacryocystorhinostomy with insertion of a glass or plastic tube into the nose (Jones tube).

**E. Lacrimal trauma**

Although the lacrimal excretory system may be obstructed by trauma to any of its components, the most common injuries are lacerations of the canaliculi or puncta and nasolacrimal duct obstructions associated with medial orbital fractures (see Chapter 4, sec. VIII.).

1. **Lacerations** of the canaliculi usually need not be repaired as emergencies. Because of the rich vascular supply and the infrequency of infections near the eyelids and medial canthus, primary repair can sometimes be delayed for as long as 12 to 36 hours after injury. Such a delay may actually be beneficial because transected canaliculi can occasionally be better identified after a period of time, and because nighttime surgery may be avoided. If only one canaliculus is severed, it should be repaired, but unnecessary damage to the uninjured canaliculus should be carefully avoided. In many individuals, no significant tearing occurs even after complete obstruction or loss of a single canaliculus. A wide variety of sutures, wires, and tubes have been described for the support of lacerated canaliculi during surgical approximation and healing. If possible, these supports should remain within the canaliculus 6 to 8 weeks after injury.

**Footnote**


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