## **Ophthalmic Pearls**

**External Disease** 

# The Tearing Patient: Diagnosis and Management

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xcessive tearing, also known as epiphora, is due to a disruption in the balance between tear production and tear loss. Numerous etiologies lead to an excess of tears, and there are a number of ways to diagnose and treat this condition.

Currently, there is not a firm consensus on the best way to evaluate the tearing patient. However, a simple algorithm may aid the general ophthalmologist in the evaluation and management of this common condition (see Fig 1).

### Anatomy and Physiology of the Lacrimal System

The main lacrimal gland, the accessory lacrimal glands and the conjunctival epithelium are responsible for producing tears. Tears are spread over the surface of the eye by blinking to establish the precorneal tear film. Each contraction of the orbicularis muscle helps move the tears across the ocular surface toward the lacrimal drainage system.

Ideally, the basal tear secretion rate equals the rate of tear drainage and evaporation. Basal tear secretion occurs at a rate of about 1.2  $\mu$ l/minute, although reflexive tear secretion can increase this up to 100-fold. Tears enter the puncta at a rate of 0.6  $\mu$ l/min; about 90 percent are reabsorbed through the nasolacrimal duct mucosa and 10 percent drain into the floor of the nasal cavity. Tears evaporate from the ocular surface at a variable rate,



but ideally tear evaporation roughly equals the difference between basal secretion and drainage. The ocular surface (including the lacrimal lakes in the conjunctival fornices, the marginal tear strip and the precorneal tear film) can hold only 8 µl of tears at any time.

### Clinical Causes and Associated Symptoms

The lacrimal system governs a delicate balance between tear production and loss with little reserve for disturbance. This balance is complicated by the fact that the system is subject to a constant

### Causes and Treatments for Epiphora

CAUSES	TREATMENTS
Punctal obstruction	Dilation, three-snip punctoplasty, sili- cone intubation
Canalicular obstruction	
Canalicular stenosis/constriction	Silicone intubation
Complete canalicular occlusion	Excision of occluded area and plastic repair of canaliculus
Canaliculitis	Antibiotics, warm compresses, curettage with canaliculotomy to remove con- cretions
Common canalicular obstruction or obstruction of both canaliculi	Conjunctivodacryocystorhinostomy (CJDCR) with Jones tube placement
Nasolacrimal duct obstruction (NLDO)	Silicone intubation with or without dacryocystorhinostomy (DCR)
With dacryocystitis	Antibiotics, allow acute infection to resolve, usually necessitates DCR
Recurrent NLDO	DCR
Poor pump function/lid malposition	
Involutional ectropion	Horizontal eyelid tightening with lateral tarsal strip or modified lateral can- thopexy
Involutional entropion	Retractor reinsertion with lateral tarsal strip or modified lateral canthopexy
Punctal ectropion	Medial spindle with or without horizontal eyelid tightening procedure
Ocular surface disorders	Correct underlying problem; if dryness is a contributing factor, consider arti- ficial tears, punctal plugs, Restasis, etc.

input of environmental, physical and biologic factors. An imbalance in the system in either direction eventually may lead to epiphora. Tear production in excess of loss directly leads to tearing, whereas tear loss in excess of production stimulates reflexive tear production, which may also lead to epiphora. Most imbalances fall into the categories described below.

**Appositional abnormalities and poor pump function.** Close apposition of the lids against the globe is an integral part of maintaining an adequate tear balance. This apposition prevents excessive exposure of the ocular surface and minimizes tear film evaporation. Lid-globe apposition is also important for directing tears across the ocular surface and into the lacrimal drainage system. The orbicularis muscle provides the pump mechanism for the excretion of tears. With eyelid closure, orbicularis contraction creates a negative pressure within the lacrimal drainage system, thus propelling fluid into the nasolacrimal sac. When the eye opens, the muscles relax, creating positive pressure within the system and forcing tears from the sac into the duct. Any abnormality that affects this pump mechanism or any condition in which the puncta are not in apposition to the globe can produce epiphora. The most common examples of this include ectropion, entropion, floppy eyelid syndrome, punctal eversion and seventh nerve palsy.

**Obstructive lacrimal drainage disorders.** Any condition that obstructs

the lacrimal drainage pathway can result in tearing. Punctal and canalicular stenosis can result from toxic medications (pilocarpine, epinephrine, phospholine iodide and idoxuridine), trauma, prior radiation therapy and chronic inflammation secondary to infections (e.g., Actinomyces israelii) or autoimmune disorders (e.g., ocular cicatrical pemphigoid, Stevens-Johnson syndrome). The nasolacrimal duct also may become obstructed secondary to involutional stenosis, trauma, prior surgery, post-irradiation, chronic sinus disease, dacryocystitis or granulomatous disease. In addition, neoplasms can affect and obstruct any portion of the nasolacrimal system.

**Ocular surface disorders.** Ocular surface dryness or irritation stimulates the reflex arc of the fifth and seventh cranial nerves, producing excessive tear secretion. If the lacrimal drainage system is unable to handle the transient increase in tear volume, this results in an overflow of tears. The list of ocular surface disorders is extensive, but some of the more common etiologies include chronic blepharitis, keratoconjunctivitis sicca, trichiasis, allergic conjunctivitis, exposure keratopathy and medicamentosa.

**Neurogenic lacrimal hypersecretory disorders.** Hypersecretion of tears is rare but can occur in certain conditions. Compression of the parasympathetic lacrimal fibers from a tumor, aberrant regeneration of the seventh cranial nerve secondary to trauma, dacryoadenitis or certain medications (e.g., cholinergic agonists) can result in inappropriate lacrimation.

### **Examination and Diagnosis**

A pertinent history is essential to provide clues to the diagnosis. History of sinus disease, sinus surgery, midfacial or ocular trauma, or history of nasolacrimal duct probing as a child may all suggest obstructive problems. In addition, pus or blood in the tear film may indicate infection or malignancy, respectively. Associated symptoms such as pain, itching, burning, etc., are important to elicit, as they may provide further insight into the etiology. A full ocular examination is warranted to pinpoint the cause of tearing.

**Inspection.** The ophthalmologist should look for facial and periorbital asymmetry, eyelid malposition and midface ptosis. Any inflammation, discharge or fistulas should be noted. It is also necessary to evaluate the corneal surface, assess the blink reflex and check for lagophthalmos. A simple but very effective way to assess for nasolacrimal duct obstruction is to evaluate the size of the tear meniscus. Burkat and Lucarelli<sup>1</sup> demonstrated that the height of the tear meniscus, measured by slit-lamp examination, was a statistically useful indicator for nasolacrimal duct obstruction. They found that the median tear level in eves with obstructed nasolacrimal ducts was 0.6 mm compared with 0.2 mm in eves with unobstructed ducts.

**Palpation.** Fullness over the lacrimal sac region and/or reflux of mucopurulent drainage upon palpation of the lacrimal sac may indicate dacryocystitis. Nodules or firmness superior to the medial canthal tendon may suggest neoplasm.

**Functional testing.** Functional tests include:

• Assessing lid laxity. Horizontal lid laxity is assessed by pulling the lid down or away from the globe. If the lid can be stretched more than 8 mm, this is considered to be excessively lax. The lid is also considered lax if it takes more than 8 seconds for the lid to return to its normal position. The laxity is severe if the lid does not appose the globe before the first blink.

• Assessing for dry eyes and other tear film abnormalities. Evaluate tear breakup time (TBUT) by having the patient refrain from blinking after placing fluorescein in the conjunctival cul-de-sac. If TBUT is less than 10 seconds, there may be a problem with tear film stability. There is some debate about the reliability of testing to evaluate tear production, but we find it useful to assess the basal tear secretion. This is done by placing a strip of filter paper in the conjunctival fornix after administering topical anesthetic drops. Less than 10 mm of tear wetting in five minutes is considered subnormal, while less than 5 mm is pathologic.

Assessing for lacrimal obstruction. While there is no consensus regarding the best way to assess for lacrimal obstruction, we present our method. The dye disappearance test (DDT) can help determine whether a lacrimal outflow obstruction is present, especially in unilateral cases. Fluorescein is instilled into the conjunctival cul-de-sac bilaterally. Persistence of significant dye and asymmetric clearance of the dye from the tear lake over five minutes indicates a relative obstruction on the side with the retained dye. We do not routinely perform the Jones I and II tests when evaluating the patency of the lacrimal system. Instead, we irrigate the lacrimal system to determine the level of the obstruction. A 27-gauge anterior chamber cannula on a 3-cc syringe with normal saline allows the ophthalmologist to irrigate without having to dilate the puncta. After irrigant is introduced into the lacrimal system, resistance, reflux and delay or lack of clearance into the nasopharynx suggests the presence of obstruction. The degree of resistance and reflux suggests the severity of obstruction, whereas the location of reflux helps

to localize the obstruction. Reflux through the same punctum suggests canalicular obstruction, whereas reflux through the opposite punctum suggests distal obstruction.

#### Management

Successful management of the tearing patient requires the clinician to determine the underlying cause of the epiphora. Unfortunately, this can be difficult to discern, at least in part because the causes are often multifactorial. We begin all epiphora evaluations by explaining the normal tear balance to patients and pointing out that any disruption to one part of the system can cause changes in other parts of the system. The "art" of the evaluation of the tearing patient is to try to determine what processes are contributing most to the tearing and then direct treatment accordingly (see "Causes and Treatments for Epiphora").

1 Burkat, C. N. and M. J. Lucarelli. *Ophthalmology* 2005;112:344–348.

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**C.** Send at least one photo or illustration.

**D.** Write an introduction letting readers

- know why this topic is relevant.
- E. Use subheadings to help readers easily navigate the 1,500-word article.

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\*Send topics to Pearls Editors Ingrid U. Scott, MD, MPH, iscott@psu.edu, or Sharon Fekrat, MD, fekra001@mc.duke.edu.

