

LAGOPHTHALMOS (LAGOPHTHALMUS)

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Introduction: Defining Lagophthalmos (Lagophthalmus)

Lagophthalmos, alternatively termed Lagophthalmus, is a significant ophthalmological disorder defined by the **inability to fully close the eyelids**. This failure to achieve complete apposition of the upper and lower eyelids leaves a portion of the ocular surface exposed, even during attempted blinking or sleep. The term derives from the Greek words *lagos* (hare) and *ophthalmos* (eye), referencing the folk belief that hares sleep with their eyes open. This condition is far more than a cosmetic issue; it represents a critical failure of the protective mechanisms designed to maintain the integrity and moisture of the cornea and conjunctiva, leading inevitably to severe complications if left unaddressed. Understanding lagophthalmos requires appreciating the delicate neuromuscular coordination necessary for complete palpebral closure, primarily mediated by the orbicularis oculi muscle.

The functional implications of lagophthalmos are profound because the eyelids perform several vital protective roles. Normal blinking distributes the tear film across the ocular surface, washing away debris and supplying essential nutrients and oxygen to the avascular cornea. When the eyelids cannot meet, the rate of tear evaporation accelerates dramatically, and the protective barrier of the tear film is compromised, creating a localized state of hypoxia and desiccation. Furthermore, lagophthalmos can manifest differently depending on its underlying cause and timing; some individuals experience **nocturnal lagophthalmos**, where the closure deficit is only apparent during sleep, while others suffer from constant, **paralytic lagophthalmos** due to facial nerve damage or mechanical obstruction. Accurate classification of the type and severity is the initial step toward effective therapeutic intervention, given the high risk of corneal damage associated with chronic exposure.

While the most common causes are related to motor nerve dysfunction, lagophthalmos is a symptom complex that can arise from diverse etiologies, including structural defects, inflammatory diseases, and iatrogenic injury. It is crucial for clinicians to differentiate between paralytic causes, which involve weakness or failure of the orbicularis oculi muscle (innervated by the Facial Nerve, CN VII), and mechanical causes, where the muscle function is intact but physical obstruction or retraction prevents full closure. The severity of lagophthalmos is often quantified by measuring the distance between the upper and lower lids during maximal attempted closure, providing an objective measure of exposure and risk. The long-term prognosis hinges entirely on the rapidity of diagnosis and the efficacy of subsequent treatment aimed at restoring corneal protection, either through lubrication or surgical correction.

Etiology and Underlying Causes of Eyelid Incompetence

The etiology of lagophthalmos can be broadly categorized into neurogenic, mechanical, and cicatricial causes. The most frequent neurogenic cause is **paralysis of the seventh cranial nerve**

(Facial Nerve), which supplies the motor function to the orbicularis oculi muscle. Conditions such as Bell's Palsy, although often temporary, are a primary driver of acute paralytic lagophthalmos. More severe and chronic neurogenic causes include trauma to the facial nerve (e.g., temporal bone fracture or surgical injury during parotidectomy), tumors compressing the nerve, or neurodegenerative diseases affecting motor pathways. When the orbicularis oculi muscle cannot contract effectively, the dynamic force required to pull the upper eyelid down and the lower eyelid up is lost, resulting in persistent ocular exposure, making this the most challenging form to manage without surgical intervention.

Mechanical lagophthalmos occurs when the eyelids are prevented from closing despite adequate function of the facial nerve and the orbicularis oculi muscle. A common mechanical cause is **severe proptosis (exophthalmos)**, typically associated with thyroid-related ophthalmopathy, also known as Graves' disease. In this condition, the volume increase in orbital tissues pushes the globe forward, stretching the eyelids over the prominent eye and making complete closure physically impossible. Other mechanical impediments include large orbital tumors or severe edema. Furthermore, iatrogenic causes, such as aggressive or excessive blepharoplasty (cosmetic eyelid surgery), can lead to over-resection of skin or muscle, causing the upper eyelid to retract and resulting in irreversible lagophthalmos.

Cicatricial, or scarring, lagophthalmos is another significant category, arising from conditions that cause contraction and retraction of the eyelid tissues themselves. This often follows severe trauma, burns, chronic inflammatory diseases like ocular pemphigoid, or certain infectious diseases. For example, severe infections of the eyelid or conjunctiva can lead to significant fibrosis, creating tension that pulls the lids away from the globe. The original observation linking the condition to **leprosy (Hansen's disease)** falls under the neurogenic category but often develops secondary cicatricial components; the bacterial infection damages the peripheral nerves, including the facial nerve branches, leading to muscle atrophy and subsequent scarring, which pulls the eyelids into a retracted state, severely compounding the exposure risk.

Pathophysiology of Incomplete Closure

The pathophysiology of lagophthalmos centers on the breakdown of the tear film and the subsequent damage to the corneal epithelium. The primary protective mechanism is the action of the orbicularis oculi muscle, which closes the eye, and the levator palpebrae superioris muscle, which opens it. In paralytic lagophthalmos, the unopposed action of the levator muscle, combined with the complete failure of the orbicularis oculi to depress the upper lid, leads to constant exposure. This exposure initiates a vicious cycle: rapid evaporation of the aqueous component of the tear film leads to hyperosmolarity on the ocular surface. This hyperosmolar stress damages the superficial corneal epithelial cells, leading to a condition known as **exposure keratopathy**, which is the hallmark complication of untreated lagophthalmos.

Crucially, the protective reflex known as **Bell's phenomenon**--the upward and outward rotation of the globe upon attempted lid closure--is often insufficient to protect the cornea in cases of significant lagophthalmos. While Bell's phenomenon attempts to tuck the sensitive cornea under the protection of the upper eyelid, if the palpebral aperture remains too wide, a substantial portion of the inferior cornea and limbus remains exposed to the environment, particularly during sleep. This inferior exposure is particularly problematic because the inferior cornea is the area most prone to desiccation and subsequent breakdown. Chronic desiccation impairs the barrier function of the epithelium, making the eye highly susceptible to bacterial invasion, ulceration, and eventual corneal melting.

The degree of risk is directly proportional to the extent of the residual opening, measured in millimeters, and the quality of the patient's tear production. Patients with pre-existing dry eye syndrome (keratoconjunctivitis sicca) who develop lagophthalmos are at an extremely high and accelerated risk of severe corneal damage. The continuous cycle of drying, epithelial damage, inflammation, and potential infection necessitates immediate intervention to artificially replace the protective function of the failed eyelids. This intervention must address both the lack of physical closure and the deficiency in tear film stability, often requiring high-viscosity lubricants to maintain a protective layer over the vulnerable corneal surface throughout the day and night.

Clinical Presentation and Subjective Symptoms

The clinical presentation of lagophthalmos spans a spectrum from subtle, chronic discomfort to acute, debilitating ocular pain and vision loss. The subjective symptoms experienced by the patient are primarily related to the exposure and drying of the cornea. Patients frequently report a persistent **foreign body sensation**, often described as grit or sand in the eye, which is exacerbated in dry, windy environments or upon waking. Other common complaints include intense ocular burning, itching, and significant photophobia (sensitivity to light). Paradoxically, patients often report excessive tearing (epiphora); this is usually a reflex tearing response triggered by the severe irritation and dryness of the corneal surface, rather than an indication of adequate lubrication.

Objectively, the most defining sign is the visible gap between the upper and lower eyelids during attempted forced closure. Clinicians can observe **scleral show**, particularly in the lower portion of the eye, which is normally covered by the eyelids. Upon slit-lamp examination, the signs of exposure keratopathy become evident. Initially, this manifests as fine, punctate epithelial erosions (PEE), typically staining with fluorescein dye in the interpalpebral zone (the area between the lids). If the condition progresses, these erosions coalesce into larger epithelial defects, leading to persistent epithelial defects (PEDs) and, eventually, a sterile or infectious corneal ulcer. The location and pattern of corneal damage are highly diagnostic for lagophthalmos, concentrated precisely in the zone of maximal exposure.

In cases of nocturnal lagophthalmos, patients often wake up with severe pain and difficulty opening their eyes, as the drying has been uninterrupted throughout the sleep cycle. The nocturnal form is frequently missed during routine clinical examination unless specifically investigated by observing the patient during sleep or using specialized monitoring devices. Chronic, low-grade lagophthalmos, even without immediate ulceration, contributes significantly to poor quality of life, visual fatigue, and impaired daily function. The continuous irritation and inflammation can lead to chronic conjunctival injection (redness) and thickening of the eyelid margins, further complicating the maintenance of a stable tear film and requiring careful management to prevent long-term structural changes.

Diagnosis and Assessment Protocols

Diagnosis of lagophthalmos is primarily clinical but requires specialized testing to quantify severity and determine the underlying etiology. The essential diagnostic maneuver is the direct observation of the patient attempting to gently close their eyes. The clinician measures the **palpebral fissure width** in millimeters during maximal attempted closure. A measurement greater than 2 mm is generally considered significant lagophthalmos. This measurement provides a baseline for monitoring treatment efficacy and assessing the immediate risk level. In cases of suspected nocturnal lagophthalmos, the patient may be instructed to film themselves sleeping or undergo an overnight observation protocol.

Further assessment includes evaluation of the corneal surface using a slit lamp after applying vital dyes, such as fluorescein and lissamine green. Fluorescein staining highlights epithelial defects and ulcers, while lissamine green staining reveals damaged or devitalized conjunctival and corneal epithelial cells, particularly those suffering from chronic desiccation. The pattern of staining--confined to the exposed area--confirms the diagnosis of exposure keratopathy secondary to lagophthalmos. Additionally, comprehensive assessment of tear film quality and quantity is mandatory, often utilizing the **Schirmer test** or tear break-up time (TBUT) to ascertain if the patient also suffers from concomitant aqueous tear deficiency, which significantly increases the risk profile.

To determine the root cause, particularly in paralytic cases, a complete neurological examination focusing on the facial nerve (CN VII) is necessary. This involves testing the strength of other facial muscles (e.g., forehead wrinkling, smiling) to classify the level of facial paralysis (e.g., House-Brackmann grading system). In mechanical cases, orbital imaging, such as computed tomography (CT) or magnetic resonance imaging (MRI), may be required to visualize and measure proptosis or identify orbital masses or fractures contributing to the mechanical obstruction. For patients with thyroid eye disease, Hertel exophthalmometry is used to accurately measure the degree of globe protrusion, providing crucial data for surgical planning and risk stratification.

Management and Therapeutic Interventions

Management of lagophthalmos is staged, prioritizing immediate ocular surface protection before considering definitive, often surgical, correction of the underlying cause. The immediate goal is to prevent irreversible corneal damage.

Non-Surgical Management (Lubrication and Moisturization):

Initial treatment involves aggressive lubrication using preservative-free artificial tears applied hourly during waking hours, and the use of high-viscosity lubricating ointments, especially before sleep. These ointments provide a physical barrier against evaporation overnight. Furthermore, moisture retention techniques, such as the use of **moisture chamber spectacles** or specialized goggles, help create a humid microenvironment around the eye, reducing the evaporative stress. For severe acute cases, temporary measures like taping the eyelids shut (taping and patching) at night can be implemented, provided the patient can tolerate the application and removal process without causing further skin irritation or trauma.

Temporary and Reversible Surgical Options:

If the lagophthalmos is secondary to an acute cause expected to recover (e.g., Bell's Palsy), temporary procedures may be utilized. A **temporary lateral tarsorrhaphy** involves surgically fusing the outer portions of the upper and lower eyelids. This narrows the palpebral fissure, effectively reducing the exposed surface area and allowing the cornea to heal. While cosmetically intrusive, this procedure is highly effective and can be reversed once facial nerve function returns, protecting the eye during the recovery phase.

Definitive Surgical Correction:

For chronic or permanent paralytic lagophthalmos, definitive surgical solutions are necessary to restore closure function. The most common and effective procedure is the implantation of a **gold weight or platinum weight** into the upper eyelid. The weight utilizes gravity to assist the eyelid in closing, supplementing the failed orbicularis oculi muscle. Alternatively, palpebral springs or fascia lata slings may be used to provide dynamic closure, pulling the eyelid down when the levator muscle relaxes. In mechanical cases (like proptosis), orbital decompression surgery may be required to reduce the protrusion of the globe, allowing the lids to cover the eye naturally. In cicatricial cases, complex lid reconstruction and skin grafting are often necessary to release contractures and restore lid elasticity and length.

Complications and Long-Term Sequelae

The most significant and feared complication of untreated or inadequately managed lagophthalmos

is progressive **exposure keratopathy**, which represents a continuum of corneal damage. Beginning as superficial punctate erosions, the damage can progress rapidly to persistent epithelial defects, where the epithelium fails to heal due to continuous desiccation. This compromised barrier function invites opportunistic infection. Once a microbial organism breaches the corneal stroma, a sight-threatening **infectious corneal ulcer** develops, which requires aggressive, immediate antimicrobial treatment. If the ulcer is deep or poorly managed, it can lead to corneal perforation, endophthalmitis, and ultimately, permanent vision loss or structural collapse of the eye.

Beyond acute complications, chronic lagophthalmos imposes severe long-term sequelae. The continuous inflammatory response associated with chronic dryness and irritation can lead to scarring of the conjunctiva and the corneal stroma. Stromal scarring results in opacification, permanently reducing visual acuity even after the lagophthalmos itself is corrected. Furthermore, the persistent need for lubrication and the risk of recurrent irritation significantly impact the patient's psychological well-being and quality of life, requiring constant vigilance and management to prevent recurrence of erosions.

In cases of severe mechanical lagophthalmos due to proptosis, the constant stretching of the eyelid tissues can lead to secondary changes, including irreversible stretching of the lateral canthal tendon and atrophy of the orbicularis muscle fibers, making subsequent surgical correction more complex. Therefore, the goal of long-term management is not merely to alleviate symptoms but to achieve a stable, functional ocular surface that minimizes the risk of infection and maintains maximal visual potential, often requiring a multidisciplinary approach involving ophthalmologists, neurologists, and plastic surgeons.

Specific Association: Lagophthalmos in Leprosy

The historical connection of lagophthalmos to **leprosy (Hansen's disease)** remains a critically important example of neurogenic paralysis. Leprosy, caused by the bacterium *Mycobacterium leprae*, is unique in its affinity for peripheral nerves, particularly those located superficially and in cooler parts of the body, such as the facial nerve branches around the orbit. The bacterium infiltrates the Schwann cells of the nerves, causing progressive demyelination and axonal degeneration. This damage leads to chronic paralysis of the muscles innervated by the affected nerve branches.

In the context of the eye, damage to the zygomatic and temporal branches of the facial nerve results in profound weakness or complete paralysis of the **orbicularis oculi muscle**. This paralysis leads directly to paralytic lagophthalmos. Leprosy-related lagophthalmos is particularly dangerous because the disease often occurs in regions with limited access to specialized eye care, and the patient may also suffer from reduced corneal sensation due to concurrent damage to the trigeminal nerve (CN V), masking the pain that would normally serve as a protective warning

signal of corneal damage.

The combination of lagophthalmos (exposure) and corneal anesthesia (lack of warning pain) creates a devastating environment for the eye, leading rapidly to severe exposure keratopathy, corneal ulceration, and often, secondary blindness. This highlights why early diagnosis and aggressive ocular protection--involving frequent lubrication, protective eyewear, and surgical procedures like tarsorrhaphy or gold weight implants--are essential interventions in the management of leprosy-affected individuals to prevent preventable visual impairment and maintain functional vision throughout their lives.

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