BMJ Best Practice

Evaluation of ptosis

Straight to the point of care

Last updated: Nov 21, 2023

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Summary

Ptosis, or blepharoptosis, refers to the drooping or downward displacement of the upper eyelid. The levator muscle, its aponeurosis, and the superior tarsal muscle are responsible for upper eyelid resting position and elevation. When these structures are compromised, the resultant depressed eyelid position can reduce the amount of light entering the eye, thereby degrading visual acuity. In pseudoptosis, aberrant structural relationships of the intact globe, bony, and soft-tissue attachments may cause secondary eyelid abnormalities.

Congenital myogenic, acquired aponeurotic, and involutional forms of ptosis represent the most common causes of ptosis among children and adults.[1] [2] Adults may be affected by associated involutional changes to the facial soft tissues that exacerbate or mask signs of ptosis. The vast majority of patients with ptosis do not present to the ophthalmologist or oculoplastic surgeon for evaluation and treatment. Of those who do, symptoms include headache, brow ache, and decreased visual acuity and visual field. Visual acuity improves with manual elevation of the eyelid and facial soft tissues. Superior visual field loss is most common; however, central vision can also be adversely affected. Any acute onset of ptosis, especially with other ocular or orbital symptoms, justifies further investigation with ophthalmologic consultation.[3]

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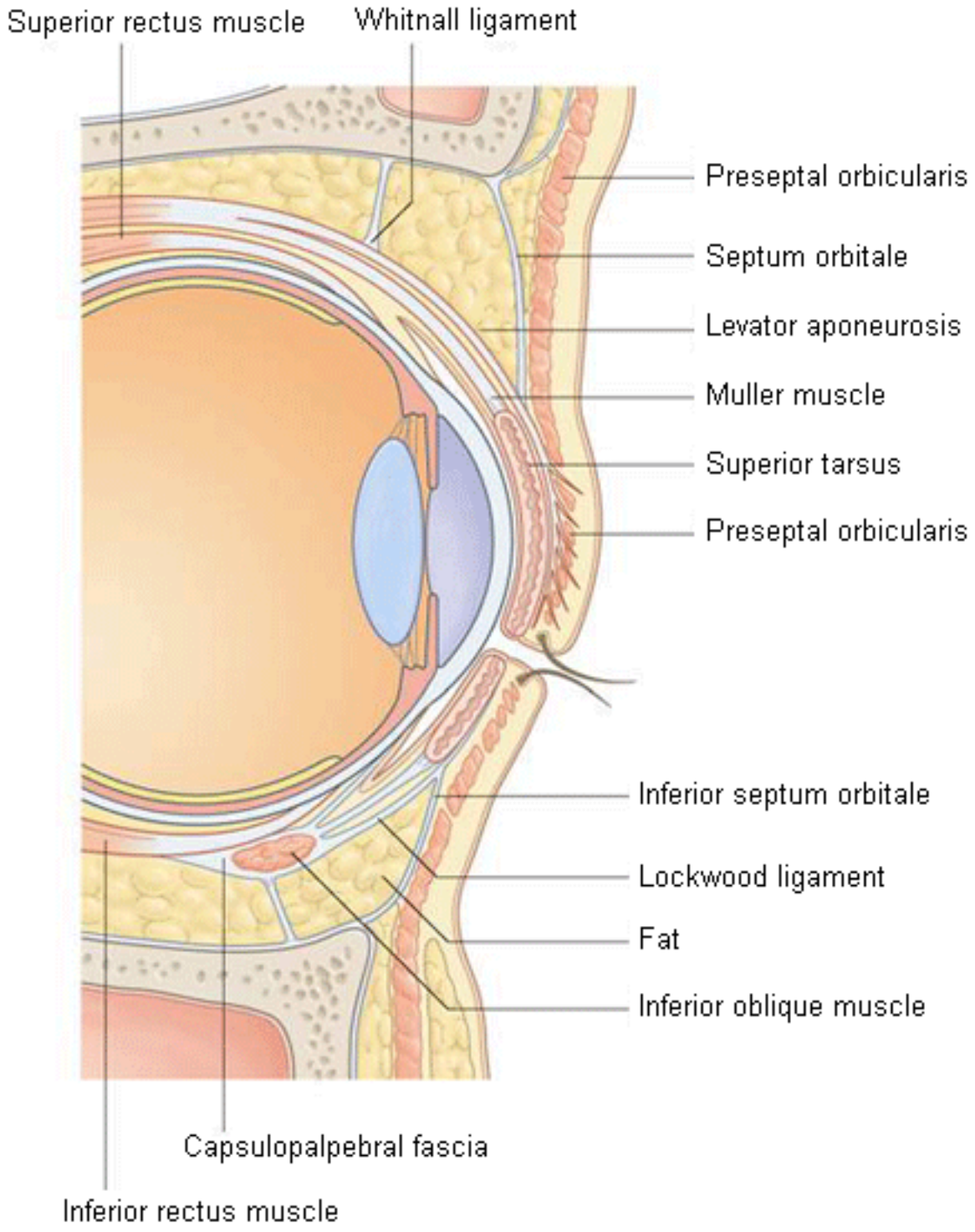

Sagittal view of eyelid anatomy

From the collection of Dr Allen Putterman

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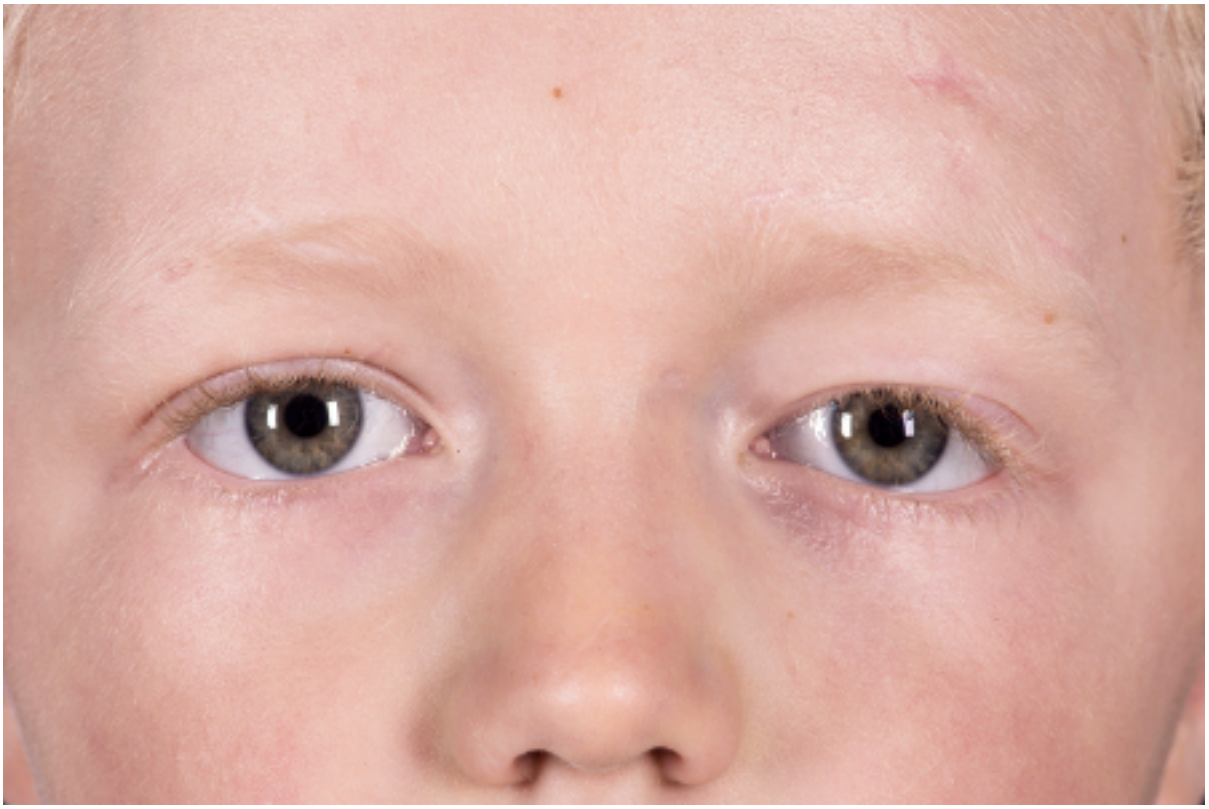

Bilateral, asymmetric, congenital myogenic ptosis

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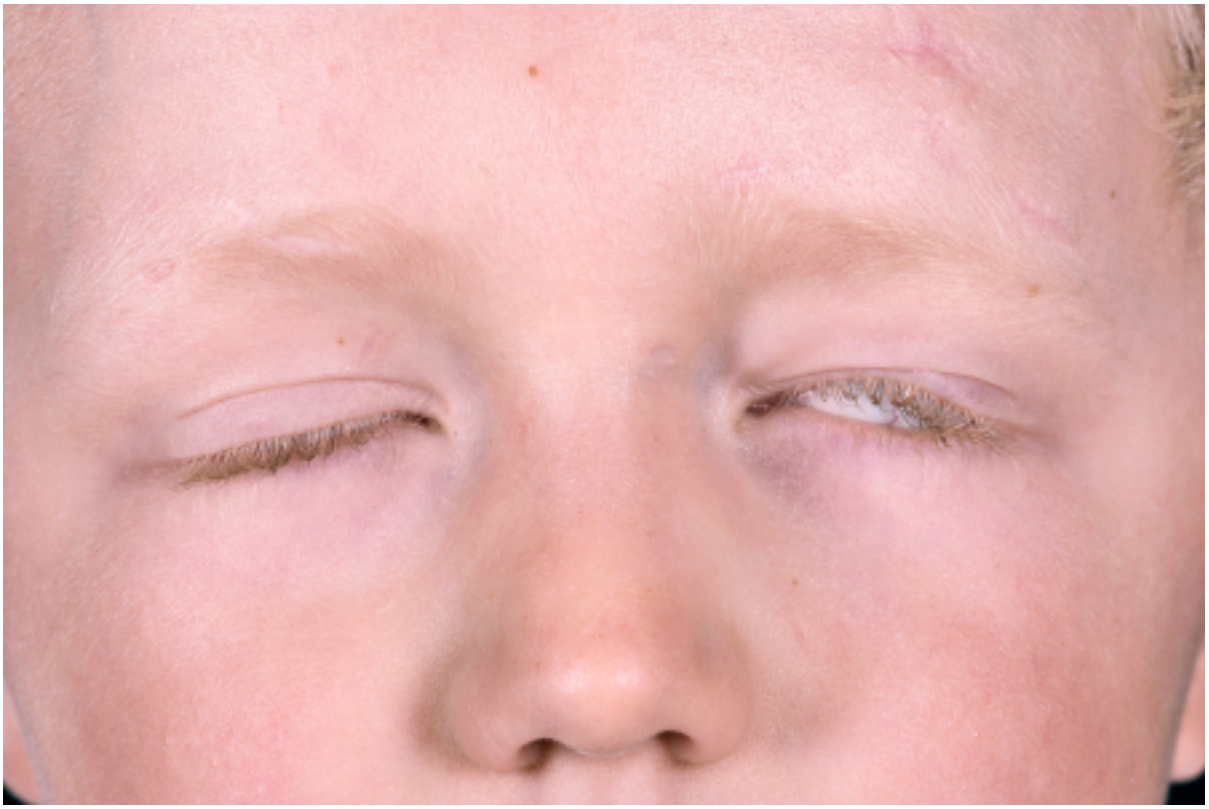

Ptosis in a 6-year-old boy. Ptosis is normally due to weakness of the levator muscle of the upper eyelid, here of the left eye (at right). This patient has had this condition since birth, and has had three operations aimed at correcting the condition

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Etiology

Ptosis is an important and often under-recognized cause of vision loss. The condition may be a manifestation of a number of local and systemic conditions that require further evaluation. Understanding the cause guides further evaluation and enhances the utility of diagnostic testing.[4] [5] [6]

Infectious or inflammatory

The presence of decreased vision, globe proptosis (displacement of eyeball from eye socket), and significant pain suggest an infectious or inflammatory process.

- Chalazion is a very common cause of ptosis resulting from focal inflammation of an obstructed meibomian gland. Lesions may be associated with pain and local or diffuse erythema.
- Stye (hordeolum) presents in similar fashion to chalazion, and represents an acute infection of secretions within eyelid sebaceous glands.
- Uveitis represents inflammation of the iris, ciliary body, or choroid, resulting from traumatic, infectious, autoimmune, or neoplastic causes.[7] Ptosis is a secondary response to ocular pain, photophobia, and eyelid edema. Uveitis has no direct relation to the onset of ptosis; however, patients may present with ptosis as a compensatory mechanism related to photophobia. Mild periorbital edema and erythema may be present. Prompt ophthalmologic evaluation is necessary for appropriate diagnosis and treatment.
- Blepharochalasis is a rare familial variant of angioneurotic edema that occurs during adolescence or young adulthood. It is characterized by recurrent episodes of inflammatory eyelid edema. After several episodes, the eyelid skin has a crepe-paper-like appearance. True ptosis may coexist secondary to attenuation of the levator aponeurosis.[8]
- The presence of headache, vision loss, and a third nerve palsy is suspicious for giant cell (temporal) arteritis. Urgent ophthalmologic evaluation is necessary to prevent permanent loss of vision.
- Preseptal cellulitis is an inflammation of the structures anterior to the orbital septum. It is a relatively common local or diffuse infectious process of the periorbital tissues resulting from superficial trauma, chalazia, hordeola, and styes, or adjacent sinus infection. Visual acuity and extraocular movement are not affected.
- Orbital cellulitis is characterized anatomically by infectious involvement of tissues posterior to the orbital septum. Orbital cellulitis is an ophthalmic emergency with vision-threatening implications from adjacent sinus infection, recent trauma, facial surgery, or immunocompromised state.
- Orbital inflammatory syndrome (OIS) is an inflammatory process of orbital soft tissue affecting children and adults. The subtype is specified by nature of involvement, such as dacryoadenitis (most common) or myositis. Systemic infectious, autoimmune, and neoplastic etiologies are often implicated, while some cases are idiopathic.

Autoimmune

Ptosis may be a result of an underlying autoimmune disorder, such as myasthenia gravis, multiple sclerosis (MS) or thyroid eye disease.

- Myasthenia gravis is an immunologic condition characterized by blockage of neuromuscular transmission resulting in local or diffuse weakness. Ptosis is the most common sign. Generalized weakness associated with dysphonia, dysphagia, and dyspnea is highly suspicious for airway and

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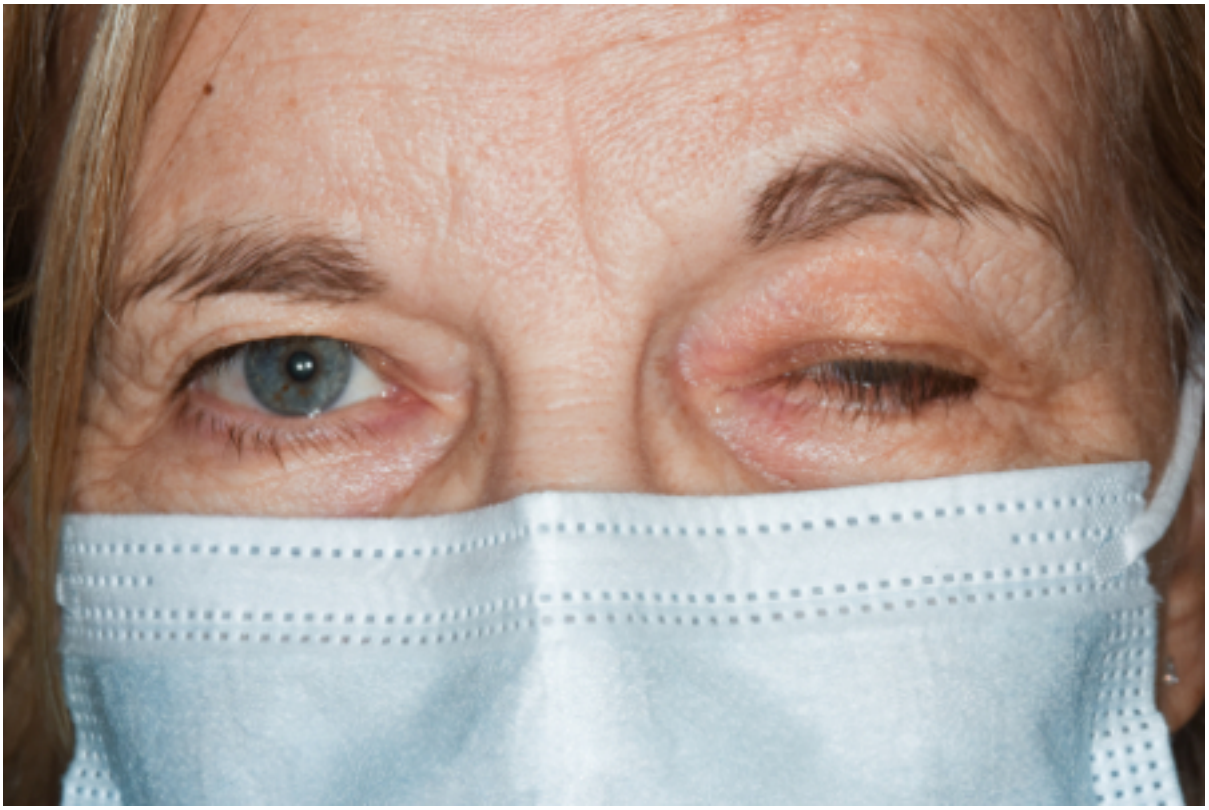
respiratory compromise, findings that require urgent evaluation. Diagnosis is verified through clinical history, provocative testing, serum assays, or electromyogram studies.[9]



Drooping eyelid (ptosis) in 69 year old female patient due to myasthenia gravis (MG). MG is a rare autoimmune neuromuscular disorder that weakens and fatigues the body's voluntary muscles, which include the muscles that control movement of the eyes and eyelids

Dr P Marazzi/Science Photo Library; used with permission

- MS is rarely associated with isolated ptosis, as diagnosis requires recognizing neurologic signs and symptoms over time, and identifying lesions affecting different areas of the central nervous system. Ophthalmologic symptoms, when they occur, reflect optic nerve or brainstem white matter involvement. Decreased vision, ocular pain with eye movement, and ocular dysmotility are common signs and symptoms of MS.
- Thyroid eye disease (Graves disease) is an autoimmune inflammatory disorder with potential vision-threatening complications. It is the most common cause of eyelid retraction and both unilateral and bilateral proptosis. Orbital disease does not necessarily accompany thyroid disease, and patients may be hyperthyroid, hypothyroid, or euthyroid. Strabismus and diplopia secondary to restrictive extraocular myopathy is common. Women are affected more than men, in a bimodal age distribution (middle-aged and older adults). Decreased visual acuity, visual field abnormalities, and relative afferent pupillary defects suggest compressive optic neuropathy for which urgent ophthalmologic or oculoplastic referral is indicated.[10]

Iatrogenic

Iatrogenic causes of ptosis include previous eye or eyelid surgery.

- Previous eye or eyelid surgery may cause disinsertion and dehiscence of the levator aponeurosis from its tarsal attachments.

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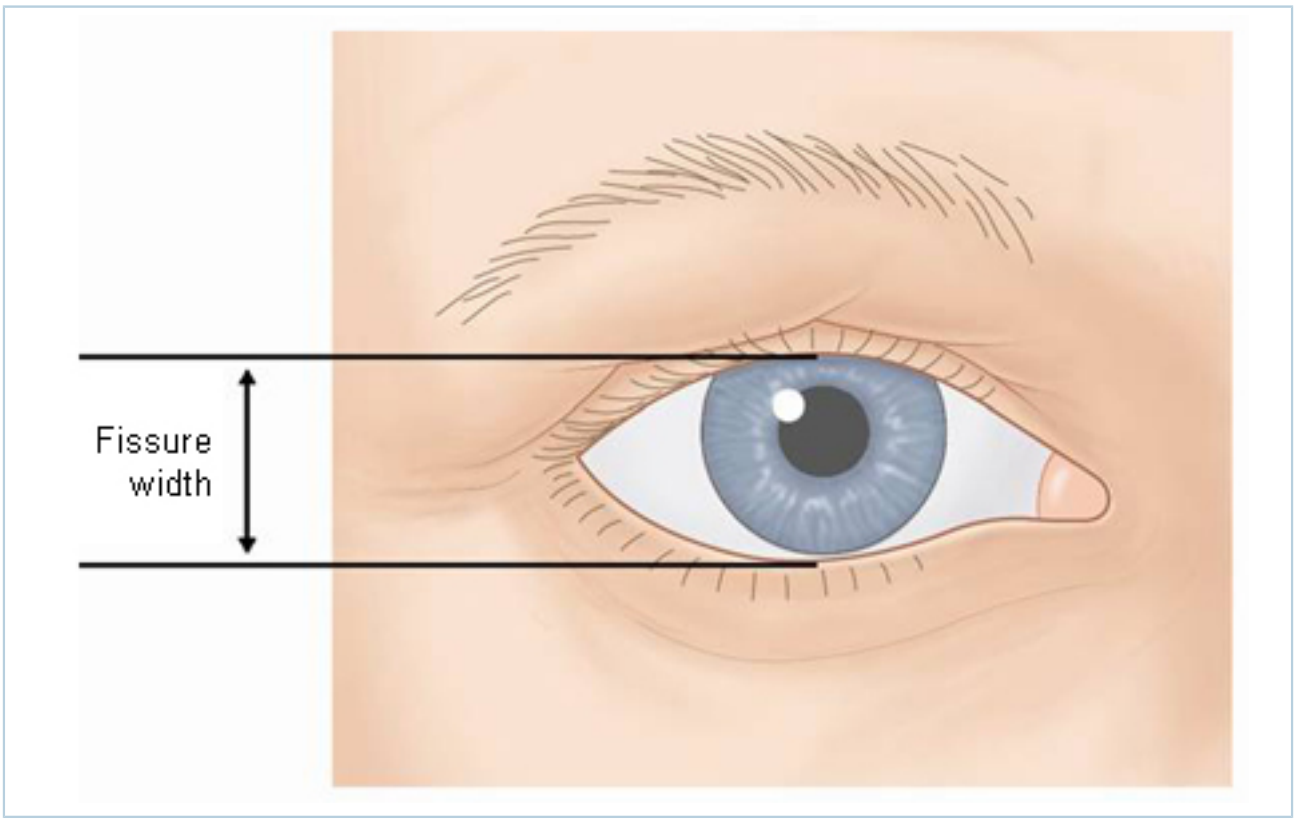
- Surgical implants may restrict action of the extraocular muscles or ultimately lead to globe malposition.
- Some patients may develop ptosis from gold weight eyelid implants for paralytic lagophthalmos or a scleral buckle for retinal detachment repair.
- Depending on the nature of the implant, measurements of palpebral fissure (widest point between the upper and lower eyelid in primary gaze), margin to reflex distance (distance from a corneal light reflex in primary gaze to the upper eyelid), and levator function (excursion of the upper eyelid in downgaze to upgaze with the frontalis muscle immobilized) can be variable.



Measurement of vertical interpalpebral fissure

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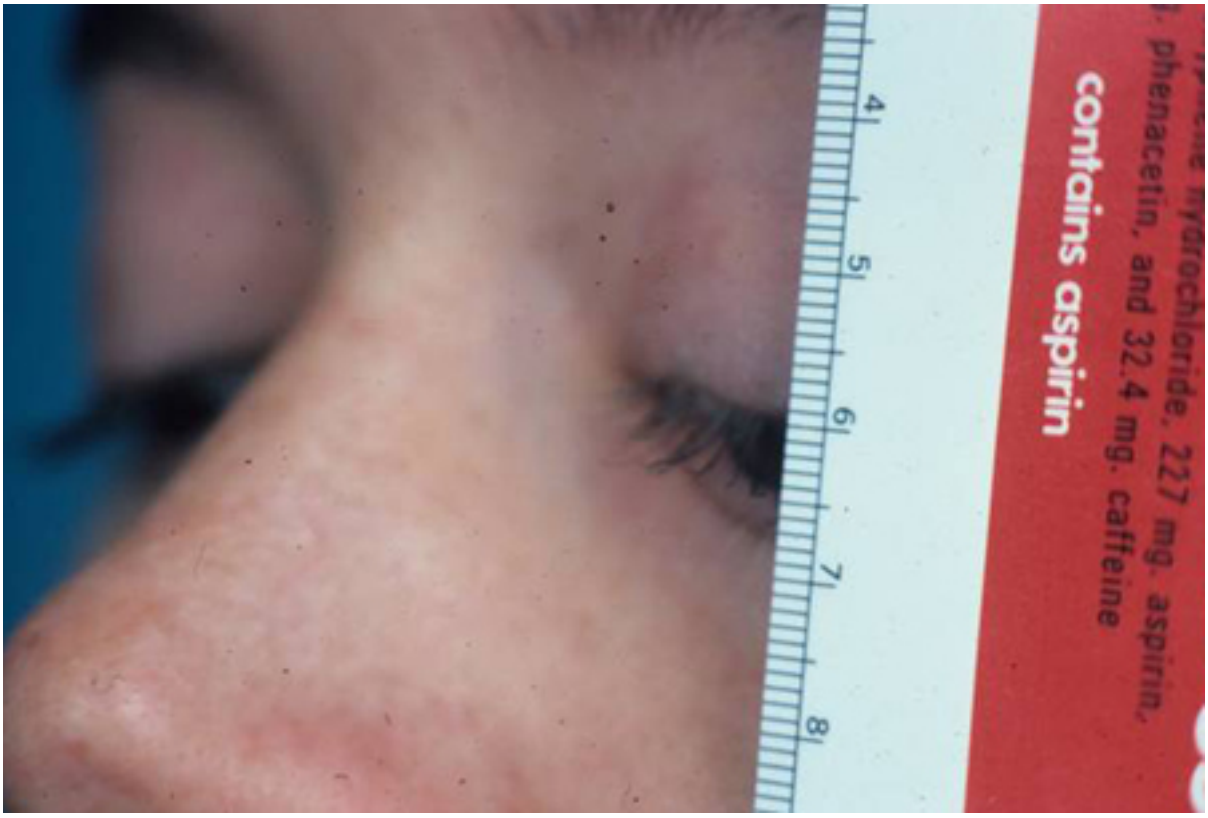

Position of upper eyelid in downgaze

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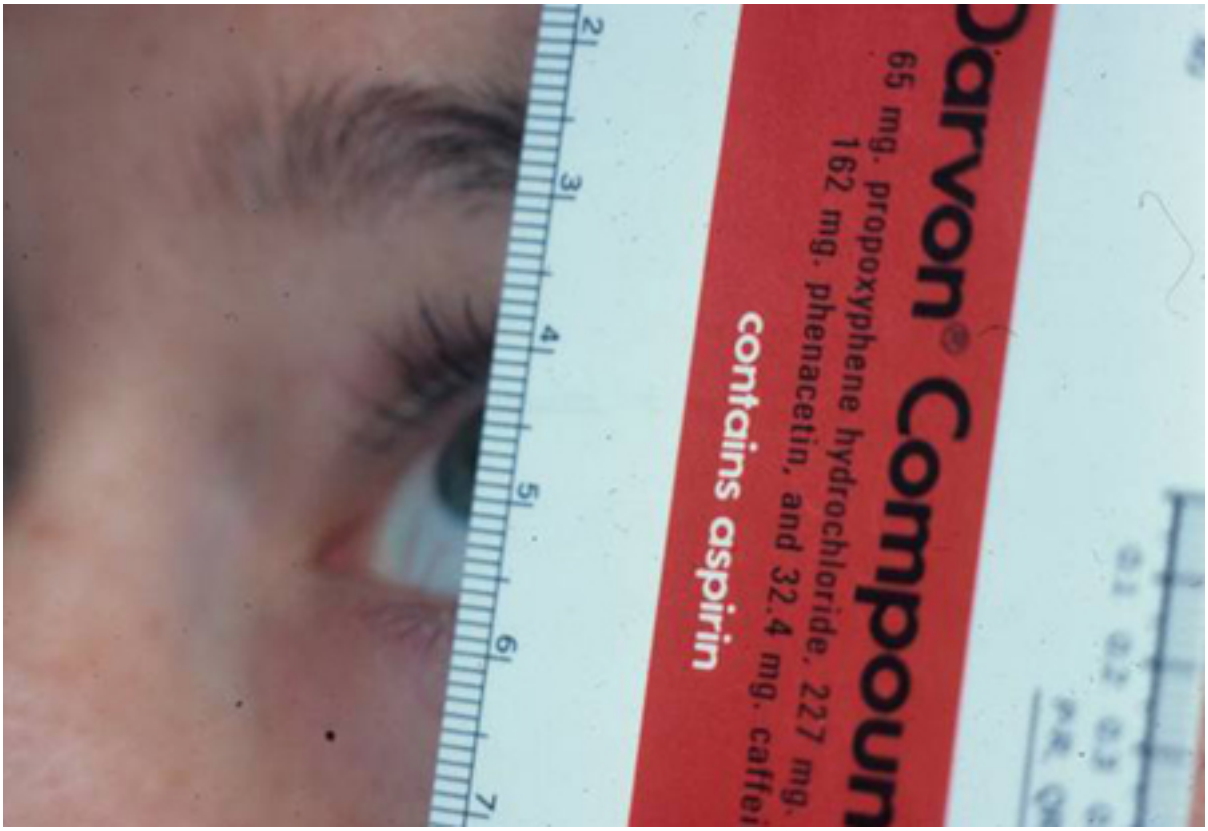

Position of upper eyelid in upgaze

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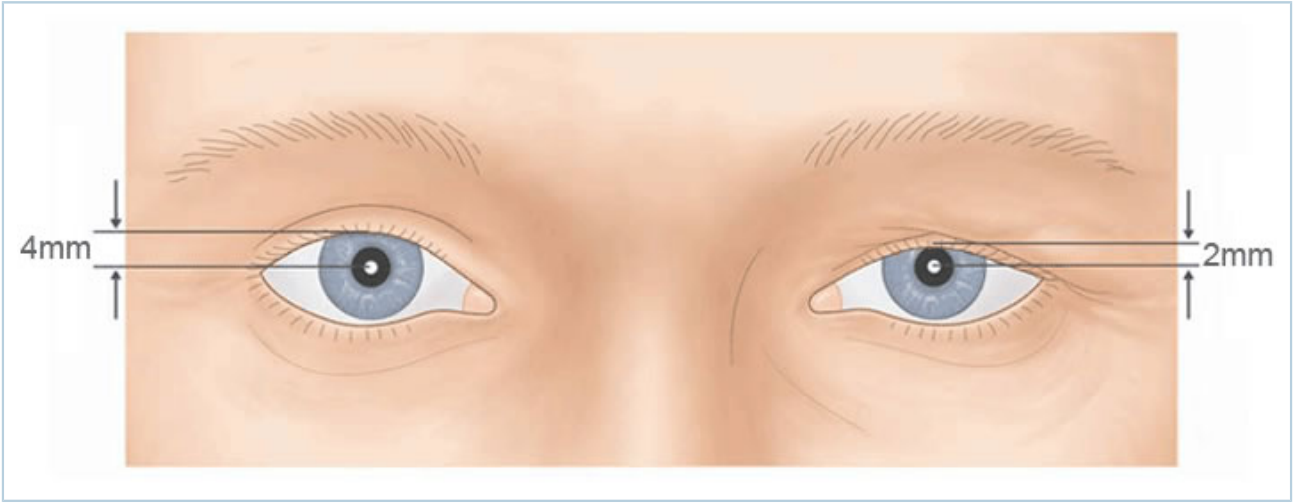

Measurement of margin-reflex distance

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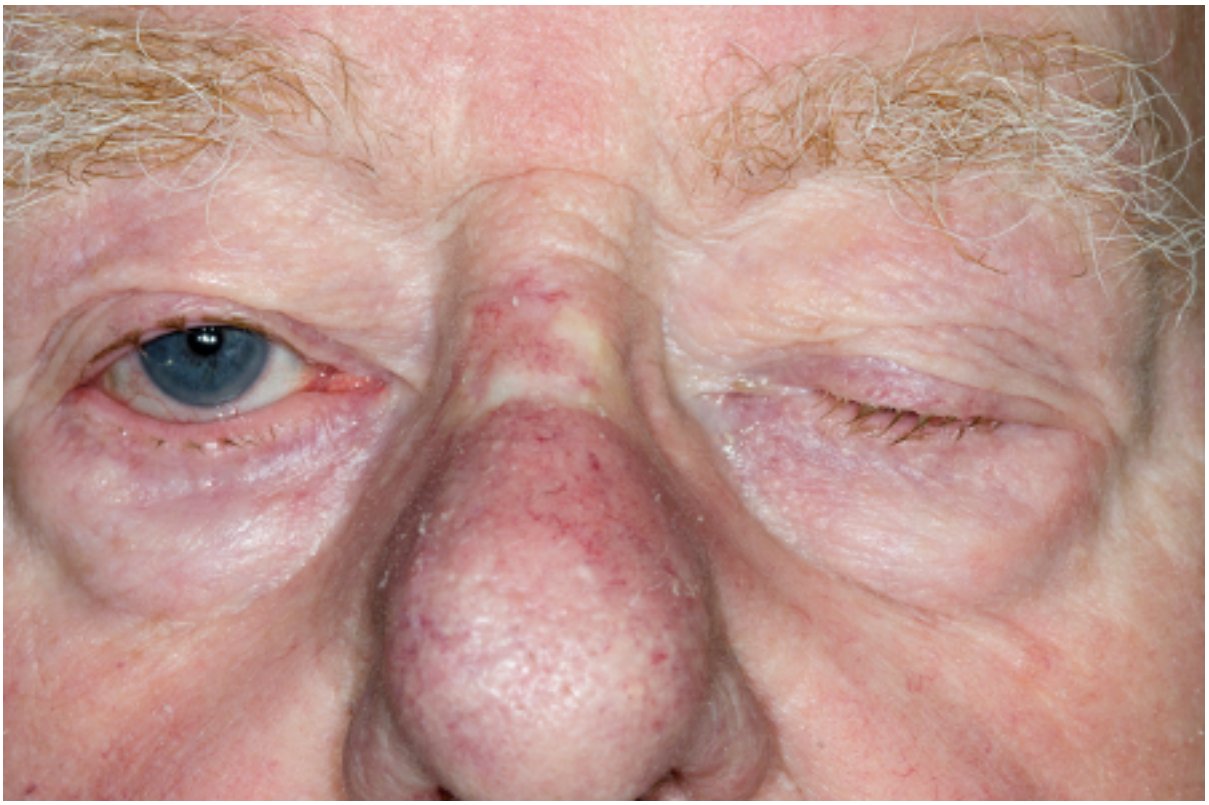

Ptosis in an 89 year old male patient following a botox injection to correct double vision (diplopia)

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Mechanical

Mechanical ptosis involves a mass lesion (tumor or foreign body) governing eyelid position and motility.

- Globe malposition refers to any process that alters the normal anatomic position of the globe within the orbit, including volume changes of the globe. Alterations of this relationship result in eyelid malposition.
- Eyelid tumors cause a mechanical form of ptosis. The location and size of the lesion determine the severity of eyelid malposition. Symptomatic benign and malignant lesions are often treated with surgical excision and eyelid reconstruction. Lesions of significant size, resulting in disruption of eyelid architecture or involving the medial and lateral canthi, are more likely to invade the orbit and surrounding structures. All neoplastic eyelid lesions are referred to surgeons experienced in eyelid reconstruction to prevent ocular complications.
- Orbital tumors cause neurogenic and mechanical forms of ptosis, and disrupt orbital architecture resulting in pseudoptosis. The type of lesion and its location determine the clinical manifestations. Certain tumors cause profound vision loss, proptosis, strabismus, and pain. Others are asymptomatic. All orbital lesions require evaluation by an ophthalmologist or oculoplastic surgeon.

Neurogenic

Neurogenic causes of ptosis should raise suspicion for vision-threatening and life-threatening conditions.

- Benign essential blepharospasm (BEB) is a bilateral facial dystonia affecting the orbicularis oculi, corrugator, and procerus muscles. Patients present with progressive, involuntary eyelid twitching, blinking, or closure. [Benign Essential Blepharospasm Research Foundation] (http://

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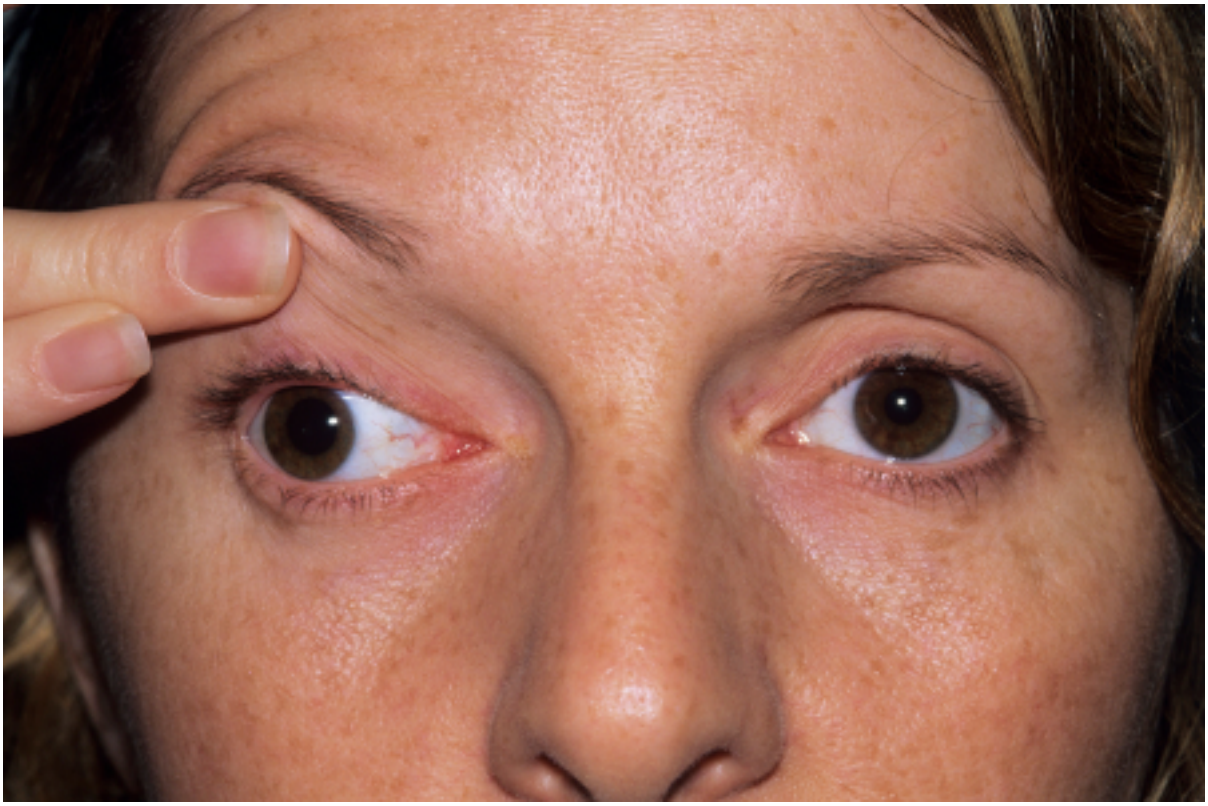
www.blepharospasm.org) Symptoms are often worse during visual tasks (e.g., reading or driving), rendering patients functionally blind.

- Chronic progressive external ophthalmoplegia (CPEO) is an inherited condition manifest by progressive, symmetric extraocular muscle paresis and ptosis. Patients often do not complain of diplopia despite obvious strabismus. Potential life-threatening systemic findings, such as cardiac conduction defects in Kearns-Sayre syndrome, may coexist.[11]
- Third nerve (oculomotor) palsy is an acute-onset dysfunction of eyelid and extraocular motility resulting in ptosis and strabismus. Involvement may be partial or complete. Most cases are ischemic in origin, reflecting comorbid hypertension, diabetes mellitus, and arteriosclerotic disease. Pupillary abnormalities (e.g., dilated pupil that responds poorly to light) suggest nerve compression, neoplastic, and aneurysmal processes.[12]



Oculomotor nerve palsy. Face of a 36-year-old woman with third nerve palsy after surgery to treat a subarachnoid haemorrhage. A berry aneurysm, a common localised dilation of an intercranial artery, caused the subarachnoid hemorrhage. Third nerve palsy is a dysfunction of the third cranial nerve, the oculomotor nerve, which controls the movement of the eyes. It leads to an inability to move the eye, double vision, a fixed and non-reactive pupil and eyelid drooping (ptosis, seen here, right eye)

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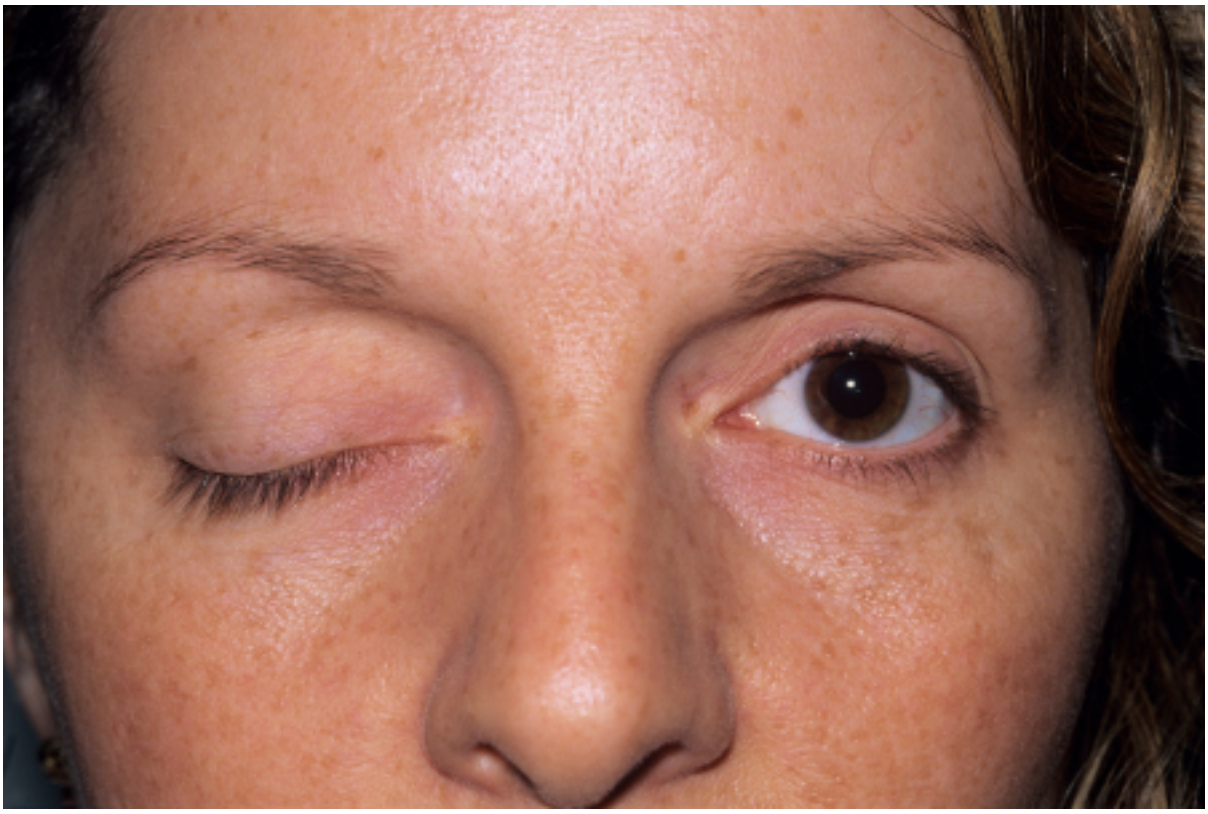

Oculomotor nerve palsy. Face of a 36-year-old woman with third nerve palsy after surgery to treat a subarachnoid haemorrhage. A berry aneurysm, a common localised dilation of an intercranial artery, caused the subarachnoid hemorrhage. Third nerve palsy is a dysfunction of the third cranial nerve, the oculomotor nerve, which controls the movement of the eyes. It leads to an inability to move the eye, double vision, a fixed and non-reactive pupil and eyelid drooping (ptosis, seen here, right eye)

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- Horner syndrome is a lesion in the sympathetic pathway resulting in ptosis, miosis, and variable degrees of anhidrosis. Involvement of the inferior tarsal muscle (analog to the upper eyelid, superior tarsal muscle) results in lower eyelid elevation with a further decrease in vertical interpalpebral fissure and pseudo-enophthalmos. Evaluation for an occult malignancy, vascular dissection, or an aneurysm is recommended in these cases.[13]
- A stroke results in a constellation of neurologic findings determined by the location of ischemia or hemorrhage. Clinical observations and measurements may be difficult to interpret and often are inconsistent. Ptosis may be prominent following a stroke involving the vertebrobasilar circulation. Management of resultant ischemic injury and edema is paramount.

Traumatic

Traumatic ptosis results from direct trauma to the levator muscle, its aponeurosis, or the superior tarsal muscle.

- Levator muscle trauma may result in partial and complete transection of the levator aponeurosis. Ptosis results from disruption of the soft-tissue and bony attachments, or globe malposition. Globe inspection is mandatory, with a low threshold of action in pursuing head and orbital imaging or seeking ophthalmologic consultation. Eyelid function depends on the extent of injury incurred.

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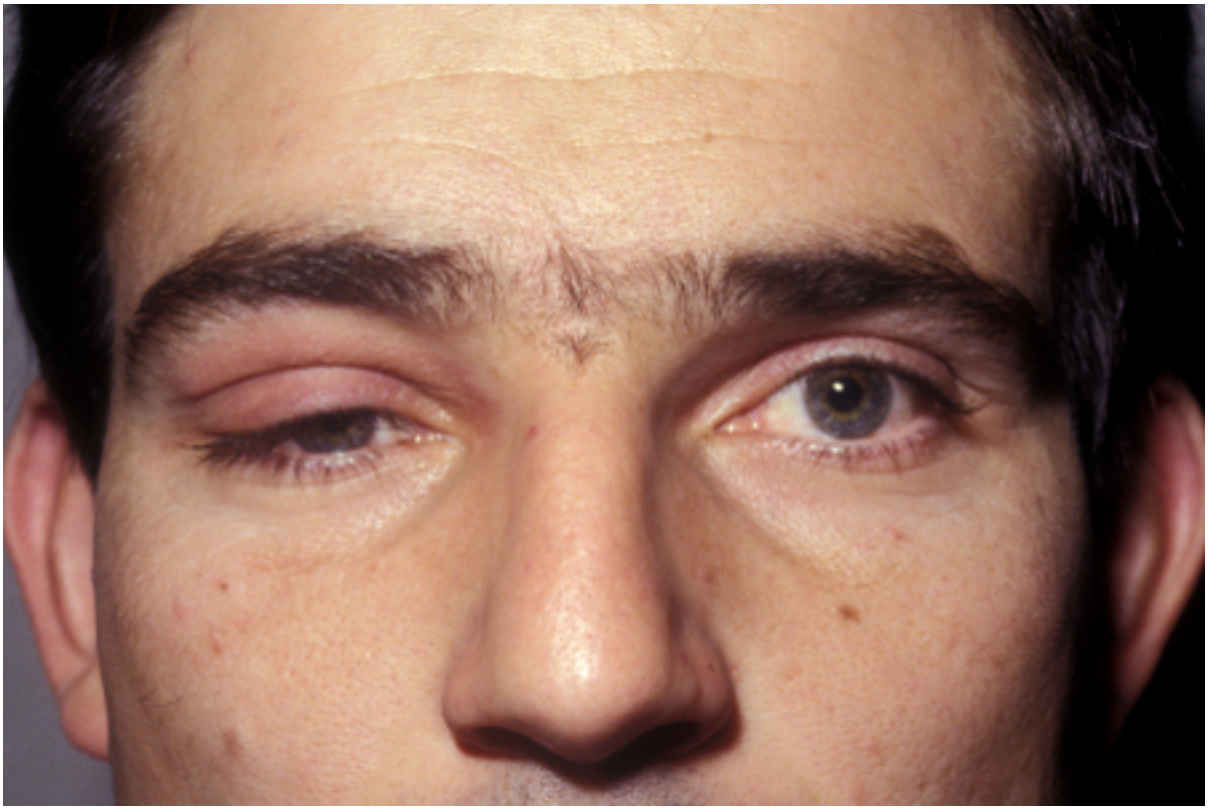
- An eyelid foreign body may reflect previous trauma or surgical intervention. Eyelid laceration is evident by clinical history and physical exam. Determination of levator function may be difficult secondary to soft-tissue edema. Wound characteristics are important to identify (e.g., prolapse of orbital fat). Urgent surgical referral is necessary for primary wound evaluation and repair.
- Orbital and facial fractures may be associated with trauma to orbital and intracranial structures and adjacent paranasal sinuses. Surgical reduction and repair is indicated for significant enophthalmos and ptosis, diplopia, or soft-tissue and extraocular muscle entrapment. Prosthesis fitting may be indicated in congenital micro-ophthalmos, or postenucleation (total globe removal) and postevisceration (partial globe removal with sclera and ocular musculature intact) patients.



Male patient suffering from post-traumatic acquired ptosis, likely caused by an injury to the eyelid

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Age-related or congenital

Age-related laxity of the periorbital and orbital soft tissue is often a corollary to aponeurotic and involutional forms of ptosis. Eyelid pathology with changes to the eyelid retractors and supportive soft tissue represents the vast majority of conditions that cause ptosis and pseudoptosis.

- Involutional (age-related) changes to the forehead skin result in brow ptosis, headache, brow ache, and prominent transverse forehead furrows with chronic contracture. Orbital fat prolapses forward secondary to attenuation of the orbital septum.
- Dermatochalasis (redundancy and laxity of eyelid skin) more commonly affects older adults and often coexists with true eyelid ptosis. Patients may present with similar symptoms of brow ache, eyelashes in the visual field, and loss of superior visual field. The upper eyelid crease may be obscured.
- Congenital myogenic ptosis results from dysgenesis of the levator muscle, originating from birth. Muscle fibers are replaced by fibrofatty tissue, which decreases the ability of the muscle to contract

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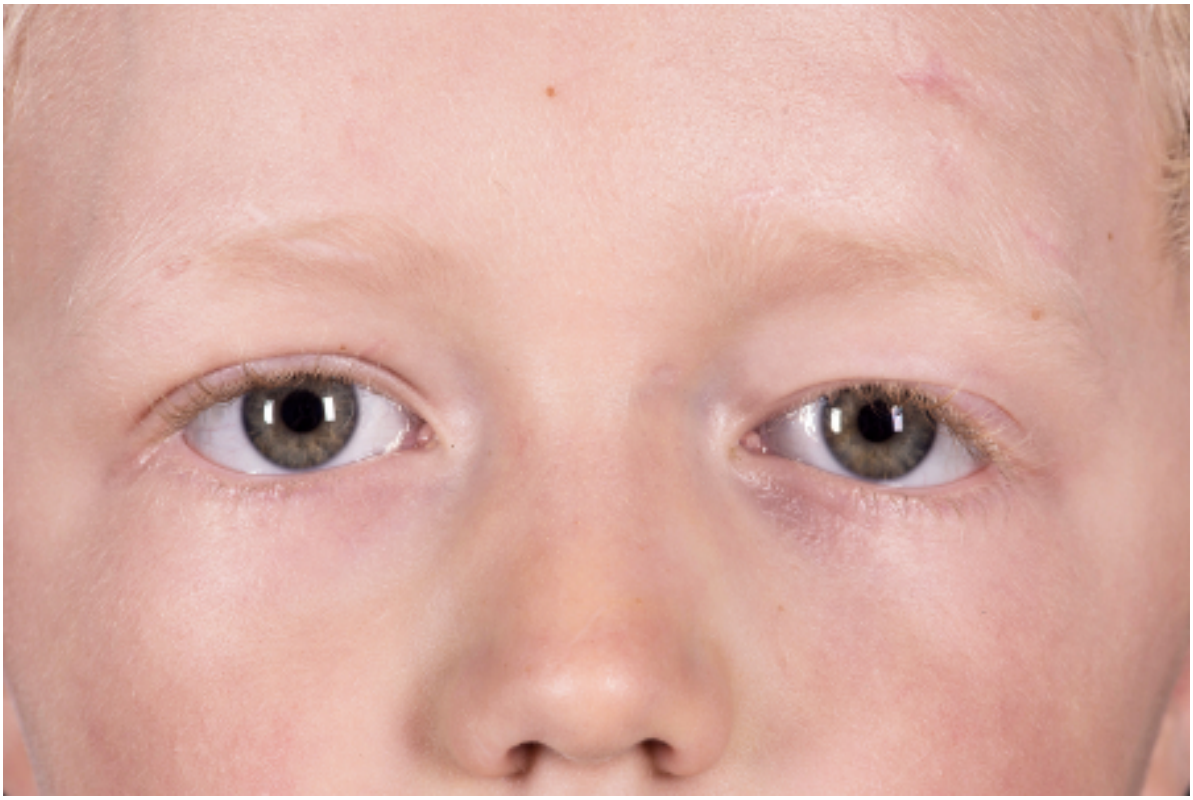
effectively.[14] Children with congenital myogenic ptosis may have amblyopia, especially in cases of bilateral asymmetry. Acquired forms (rare) may be a manifestation of local or diffuse disease.



Bilateral, asymmetric, congenital myogenic ptosis

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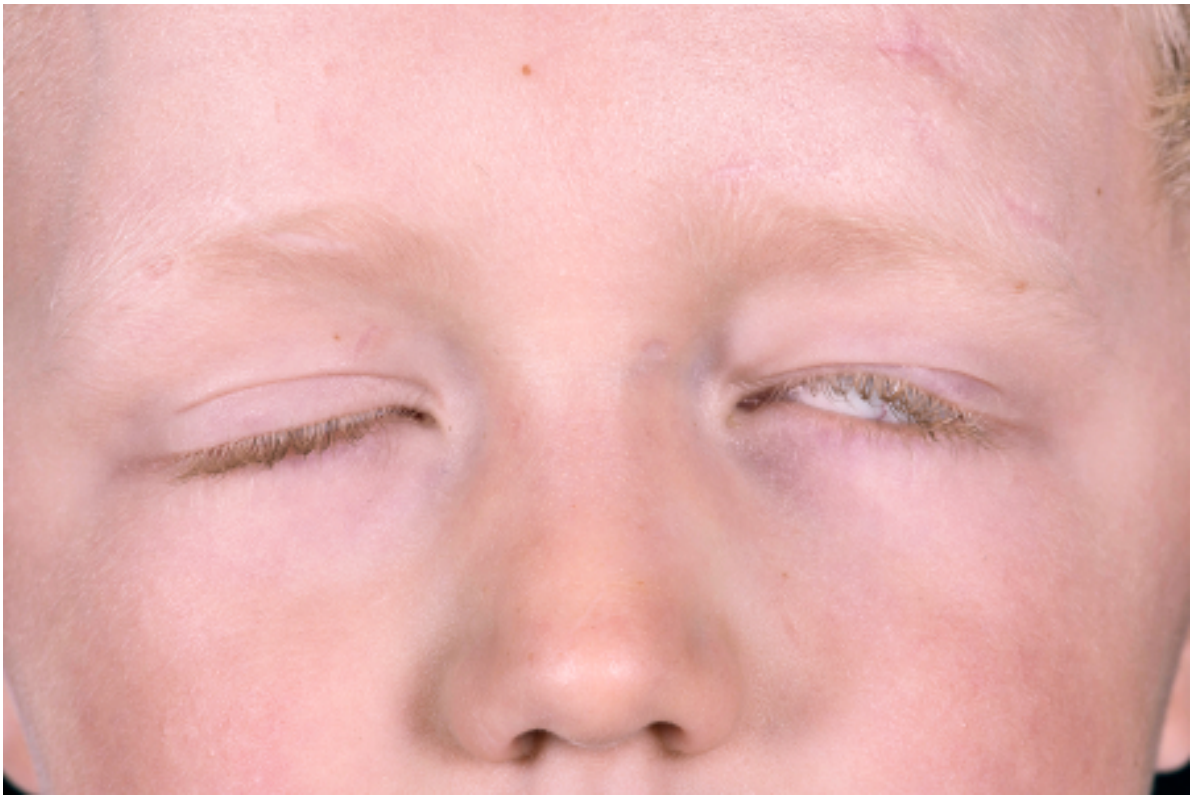

Ptosis in a 6-year-old boy. Ptosis is normally due to weakness of the levator muscle of the upper eyelid, here of the left eye (at right). This patient has had this condition since birth, and has had three operations aimed at correcting the condition

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- Blepharophimosis is an autosomal-dominant, congenital eyelid syndrome presenting with severe ptosis, telecanthus, and epicanthus inversus.

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Urgent considerations

(See **Differentials** for more details)

Uveitis

Uveitis may have an acute or insidious onset with recurrent exacerbations. Ocular inflammation may be caused by traumatic, infectious, autoimmune, and idiopathic factors.[7] Common symptoms include decreased vision, redness, ocular discomfort, and photophobia. Loss of vision can be significant with retinal and optic nerve involvement. Inflammation can be prolonged, and require topical or systemic immunosuppression. Topical cycloplegics may be used for ocular discomfort. Patients with significant symptoms, profoundly decreased vision, a relative afferent pupillary defect, or an unknown etiology that requires topical or systemic corticosteroids should receive prompt ophthalmologic evaluation.

Uveitis has no direct relation to the onset of ptosis, but ptosis may occur in people with this condition as a secondary response to ocular pain, photophobia, and eyelid edema.

Globe malposition

Globe malposition refers to any process that alters the normal anatomic position of the globe within the orbit, including volume changes of the globe. Alterations of this relationship result in eyelid malposition.

The cause of globe malposition is often elucidated by careful clinical history (e.g., congenital versus acquired, acute versus chronic, traumatic versus surgical). Inflammatory conditions, such as thyroid eye disease and orbital inflammatory syndrome, may result in soft-tissue fibrosis causing pseudoptosis. Facial trauma resulting in globe perforation or significant orbital fracture requires urgent surgical intervention.

Thyroid disease

Thyroid eye disease can result from restrictive extraocular myopathy or eyelid edema, although eyelid retraction with proptosis is characteristic. Severe extraocular muscle enlargement may result in loss of vision due to compressive optic neuropathy. Systemic corticosteroids, orbital radiation, or surgical decompression by a specialist may be indicated.[10]

Myasthenia gravis

Fluctuation and fatigability are hallmark features. Ptosis is the most common sign and >50% have ocular signs and symptoms at disease onset.[15]

Systemic disease associated with dysphagia and dyspnea may have life-threatening implications and necessitates prompt treatment with anticholinesterases and immunotherapy.[9]

Traumatic transection of levator muscle or aponeurosis

Direct trauma to the levator muscle or aponeurosis, or previous eyelid surgery, may cause disinsertion or dehiscence of the levator aponeurosis from its tarsal attachments. Salient wound characteristics, such as prolapse of orbital fat with or without decreased vision, are suspicious for levator transection or globe perforation. Determination of levator function may be difficult secondary to soft-tissue edema. Urgent surgical referral is necessary for primary wound evaluation and repair.

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Orbital and facial fracture

Trauma to orbital and intracranial structures and adjacent paranasal sinuses may be associated with significant enophthalmos and ptosis, and diplopia. Pediatric orbital fractures may be associated with minimal swelling and other signs and symptoms of orbital injury. Prompt ophthalmologic evaluation is recommended.[16]

Eyelid laceration

Eyelid laceration with herniation of orbital fat requires evaluation for levator muscle and aponeurosis laceration, and for globe perforation. A laceration with extension medial to the lacrimal punctum suggests lacrimal canalicular injury. Any eyelid trauma with decreased vision or a relative afferent pupillary defect requires urgent ophthalmologic consultation with possible surgical repair.

Third nerve palsy

The presence of a relative afferent pupillary defect with partial or complete third nerve palsy should prompt neuroimaging, with urgent neurologic consultation for a compressive lesion or an aneurysm.[17] Third nerve palsy with headache and vision loss suggests giant cell (temporal) arteritis. Ophthalmologic evaluation with possible temporal artery biopsy by a specialist may be indicated.

A complete third nerve palsy in a patient with evidence of hypertension, diabetes mellitus, or atherosclerosclerotic disease, is most likely due to microvascular ischemic disease. Therefore, in this patient cohort, a complete third nerve palsy without pupillary abnormalities may be cautiously observed.[12]

Horner syndrome

Congenital Horner syndrome is associated with trauma to the brachial plexus during birth. Extraocular motility and levator function are preserved. Children with a new-onset Horner syndrome are evaluated for possible neuroblastoma. Acquired Horner syndrome in adults warrants a careful history for other symptoms that may aid in localization of the lesion (e.g., apical lung tumor compressing the sympathetic trunk). Ipsilateral head pain or temporary loss of vision suggests possible carotid dissection or aneurysm. Neuroimaging or arteriography with neurologic consultation are appropriate.[13] [17] [18]

Stroke

Acute onset of symptoms may correlate with route of circulation and region of ischemia. Urgent neurologic consultation for evaluation and treatment is mandatory.

Orbital cellulitis

Patients present with orbital pain and discomfort, decreased vision, proptosis, and diplopia. The history is significant for recent concurrent sinus infection, recent dental and facial surgery, trauma, preseptal cellulitis, or immunocompromised state. Orbital cellulitis represents a vision-threatening, potentially life-threatening, emergency requiring appropriate neuroimaging and broad-spectrum, intravenous antibiotics.[17] Ophthalmologic consultation is mandatory early in the clinical course. Poor initial response to antibiotics may require addition of antifungals or surgical intervention.[19] [20]

Eyelid foreign body

An eyelid foreign body may reflect previous trauma or surgical intervention. Clinical history is contributory. Orbital imaging is indicated in traumatic forms of ptosis if eyelid laceration is associated with a deeply

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embedded foreign body or frank globe trauma.[17] Treatment includes surgical removal of the embedded foreign body from the eyelid.

Giant cell arteritis

Giant cell arteritis is an inflammatory condition that affects branches of the carotid artery. Patients are typically older adults, with a history of a temporal headache, vision loss, and variable ocular or orbital discomfort. Associated symptoms include jaw claudication, ptosis, fatigue, lethargy, loss of appetite, and weight loss. Associated signs include palpable tenderness along the course of the superficial temporal artery and lack of pulsation of this artery. Vision loss may be significant and symptoms may develop in the contralateral eye without urgent identification and appropriate intervention. Urgent ophthalmologic evaluation is necessary to prevent permanent loss of vision.

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Approach

Acute onset of ptosis requires urgent evaluation. Insidious onset is usually related to age-related involutional changes of the eyelid and support structures, or progression of local or diffuse muscular disease.

History

The patient history is the most important aspect of the evaluation of a patient with ptosis. This information will alert the examiner to potentially vision-threatening and life-threatening conditions prior to the physical exam.

A traditional order of interview, beginning with chief complaint and followed by a thorough past medical and surgical history with systemic review of symptoms, is the most fruitful approach.

- Pain is sought as a sentinel sign at presentation. The presence of decreased vision, globe proptosis, and significant pain suggests an infectious or inflammatory process. Orbital pain, headache, mental status changes, and vertigo may indicate occult or uncontrolled hypertension and diabetes mellitus, conditions suggestive of stroke as the cause of ptosis.[12] Patients with aponeurotic and involutional forms of ptosis present with symptoms of headache, brow ache, and decreased vision that worsen over the course of the day.[4] Difficulty reading is a common complaint, as ptosis is worse on downgaze.[21]
- Adults with the acquired myogenic forms of ptosis may have excessive production of tears, ocular irritation, and corneal exposure secondary to inability to fully close the eye (lagophthalmos) or poor Bell phenomenon (normal upward orbital rotation with eyelid closure).
- A history of implanted ophthalmic devices, such as scleral buckles and glaucoma implants, can cause a mechanical ptosis related to implant size or a pseudoptosis secondary to effects on extraocular muscles. Aponeurotic and involutional forms of ptosis are worsened with use of eyelid retraction devices during ocular surgery.[22] Loss of orbital volume secondary to globe pathology (neoplasm) or orbital radiation therapy may also cause ptosis. Previous head and neck or chest surgery (as well as lesions along the sympathetic pathway) suggest Horner syndrome.[13]
- Past medical history in young patients or patients with a strong hereditary predilection to basal cell carcinomas may suggest systemic syndromes, such as basal cell nevus syndrome or xeroderma pigmentosum.
- Numerous medications can exacerbate ptosis by causing eyelid swelling or through disruption of normal sympathetic tone. Transient ptosis may occasionally complicate botulinum toxin type A treatment of strabismus, blepharospasm, and facial wrinkles.[23] [24] [25]
- Cardiopulmonary symptoms of lethargy, palpitations, chest pain, and dyspnea may be presenting signs of diffuse muscular disease as the cause of ptosis. Vasculopathic diseases (e.g., diabetes mellitus, hypertension, and atherosclerosis) may be accompanied by third nerve palsy and ptosis. Stroke may cause ptosis if the vertebrobasilar circulation is involved.[12]
- Congenital forms of ptosis may result from innervational defects during development, such as oculomotor nerve palsy and Horner syndrome. Localized or diffuse muscular dystrophy, such as myotonic dystrophy or oculopharyngeal dystrophy, and mitochondrial myopathies, such as Kearns-Sayre syndrome, can result in acquired myogenic forms of ptosis.[11]
- Ptosis may be a result of an underlying autoimmune disorder. Myasthenia gravis often presents with generalized weakness; patients may have undergone thymoma excision.[9] Dysphonia, dysphagia, or shortness of breath and dyspnea should raise suspicion for generalized myasthenia gravis in

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any patient with inconsistent upper eyelid position and variable diplopia.[9] Systemic autoimmune conditions can present as eyelid infiltration or orbital inflammation with ptosis (e.g., thyroid disease).

- Cutaneous dermatologic malignancies can result in ptosis. Patients with prolonged sun exposure are at risk for these cancers, and the eyelid is a common location.

Physical exam

Initial assessment of patients with ptosis includes vital signs and careful inspection of the periorbital area for evidence of infection, trauma, and visual disturbance.

- Preseptal soft-tissue edema and erythema with overlying induration is common in infectious and inflammatory etiologies.[26]
- Lacrimal gland enlargement associated with orbital inflammatory syndrome, sarcoidosis, and other autoimmune conditions can exacerbate ptosis and present with temporal upper eyelid fullness or a discrete upper eyelid mass. Production of tears may be functionally impaired secondary to ocular irritation or tear outflow obstruction. Orbital cellulitis and orbital malignancies can be secondary to adjacent sinus infections and malignancies, respectively.[20]
- The presence of significant orbital trauma may compromise the interrelationship of the eyelid retractors and the soft-tissue and bony attachments. Herniation of orbital fat from an eyelid laceration with or without decreased vision is suspicious for levator muscle and aponeurosis injury and globe perforation. Orbital wall fractures increase the volume of the orbit resulting in enophthalmos and pseudoptosis. Patients with confirmed orbital trauma and high suspicion for globe perforation require urgent ophthalmologic consultation.
- Accurate visual acuity readings (distance and near) must be obtained with habitual correction (glasses or contact lenses). If glasses or contact lenses are unavailable, pinhole occlusion devices may be used. If standardized vision screening materials are unavailable, newspaper print, identification badges, or confrontation with fingers or hand movements can be used. Bright colored objects or a light source can be used for preverbal children.
- Careful attention is given to a precise pupillary exam under dim illumination. Anisocoria (discrepancy in size of pupils) may reflect disruption of either sympathetic or parasympathetic input. A relative afferent pupillary defect implies significant retinal and optic nerve injury often requiring urgent ophthalmologic consultation. Disruption of the sympathetic input to the superior tarsal muscle induces mild ptosis (1-3 mm), pupillary miosis, and variable anhidrosis. The associated neurologic findings depend on the nature and location of the lesion affecting the sympathetic input.[13] Interruption of normal oculomotor nerve innervation presents with moderate to severe ptosis, pupillary mydriasis, ocular dysmotility, and diplopia secondary to inflammatory, infectious, ischemic, traumatic, and compressive lesions.
- Ocular motility should be evaluated using a specific focus of interest (e.g., examiner's finger, pen, light source) in all positions of gaze while recording relative limitations of gaze and subjective diplopia. Dysmotility suggests restriction of movement or paresis secondary to disruption of normal innervational input. Children with myogenic forms of ptosis often adopt a chin-up position to gaze under the ptotic eyelid.
- Slit-lamp exam is important for evaluation of intraocular inflammation. Presence of cell and flare (white blood cells floating in milky substance), hypopyon (frank pus), or hyphema (blood) in the anterior chamber may require ophthalmologic referral. Optic disk and funduscopic evaluation should be attempted using a direct ophthalmoscope, if available.
- An estimate of the vertical interpalpebral fissure (PF), margin-reflex distance (MRD), and levator function (LF) is helpful to assist in determining the cause of ptosis.[27] In myogenic forms of ptosis, PF, MRD, and LF are decreased. In aponeurotic and involutional forms of ptosis, PF and MRD are

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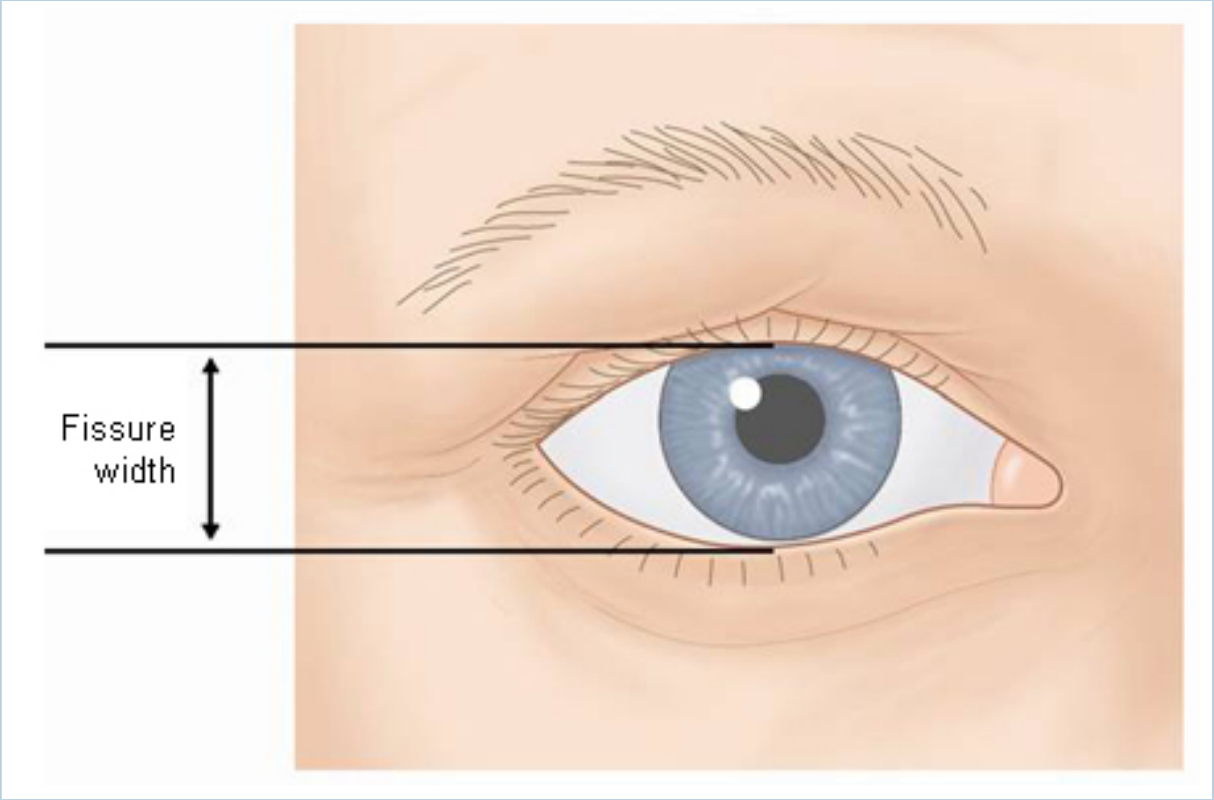
decreased and LF is intact. In neurogenic, mechanical, and traumatic forms of ptosis PF, MRD, and LF may be minimally to severely decreased.



Measurement of vertical interpalpebral fissure

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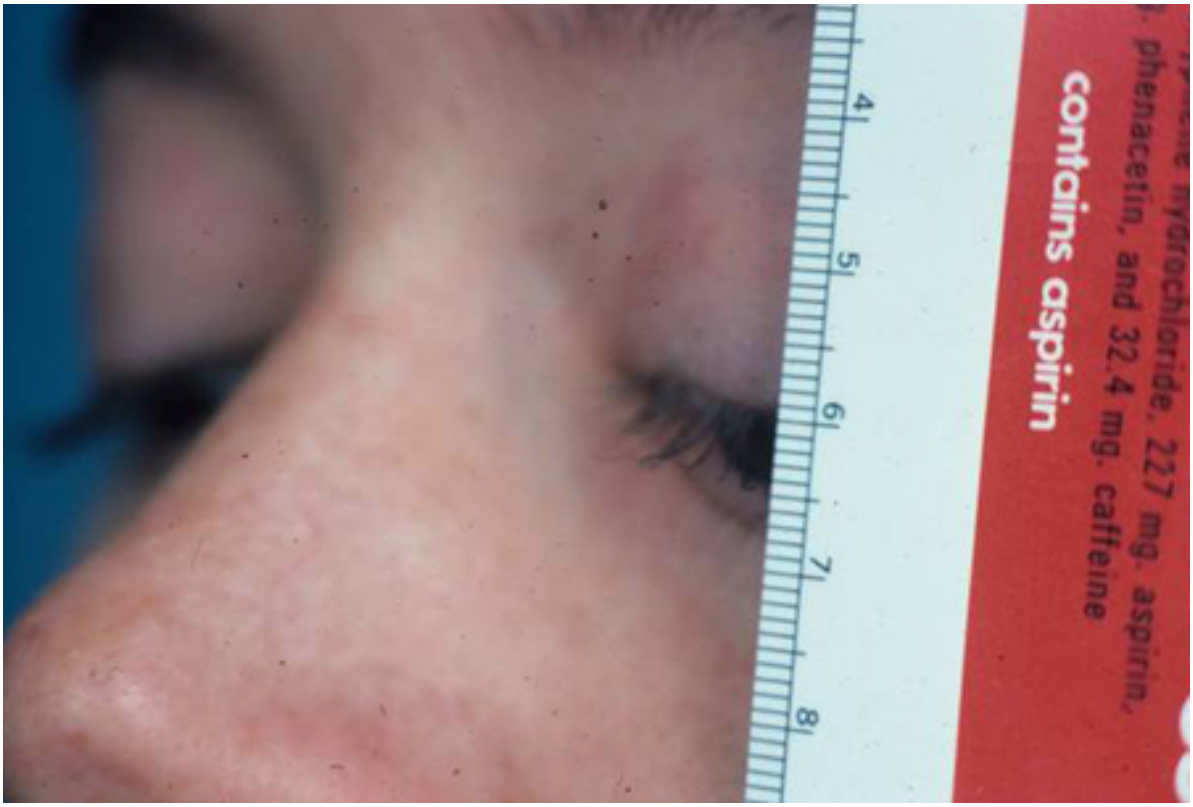

Position of upper eyelid in downgaze

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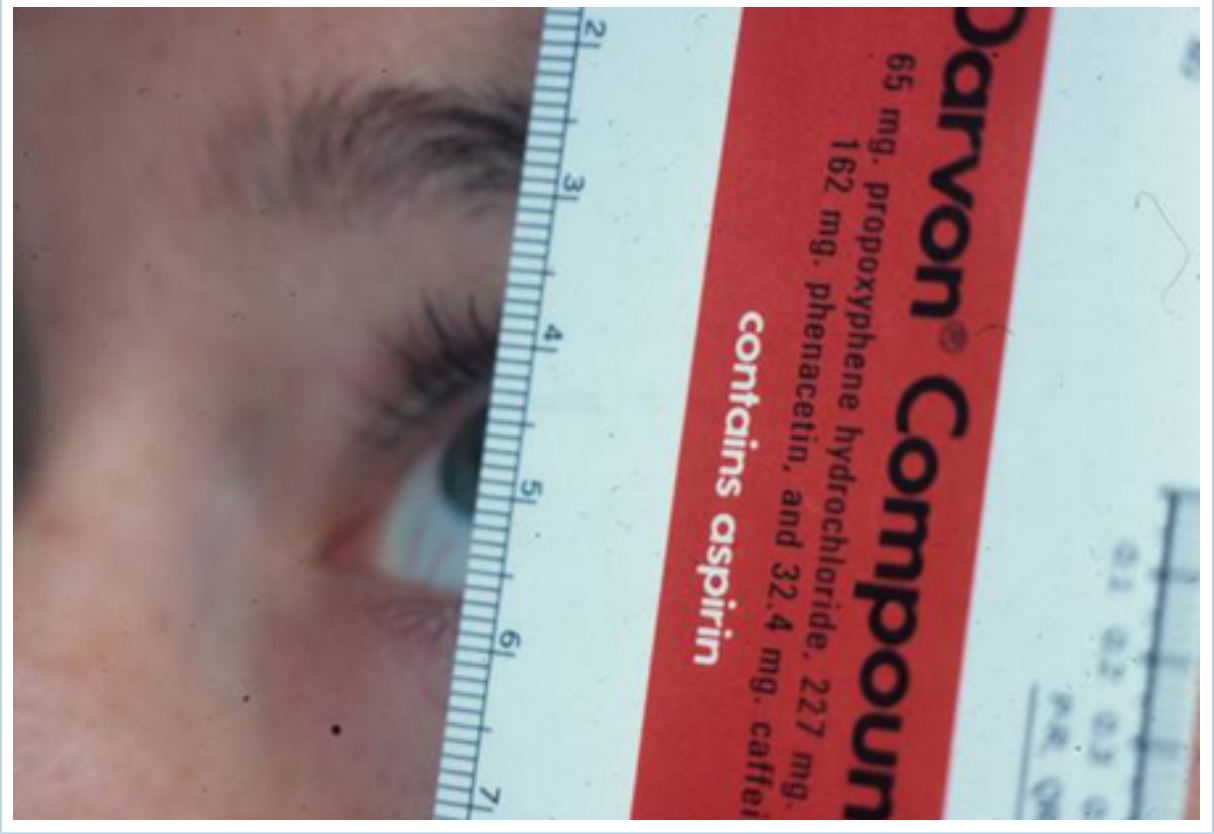

Position of upper eyelid in upgaze

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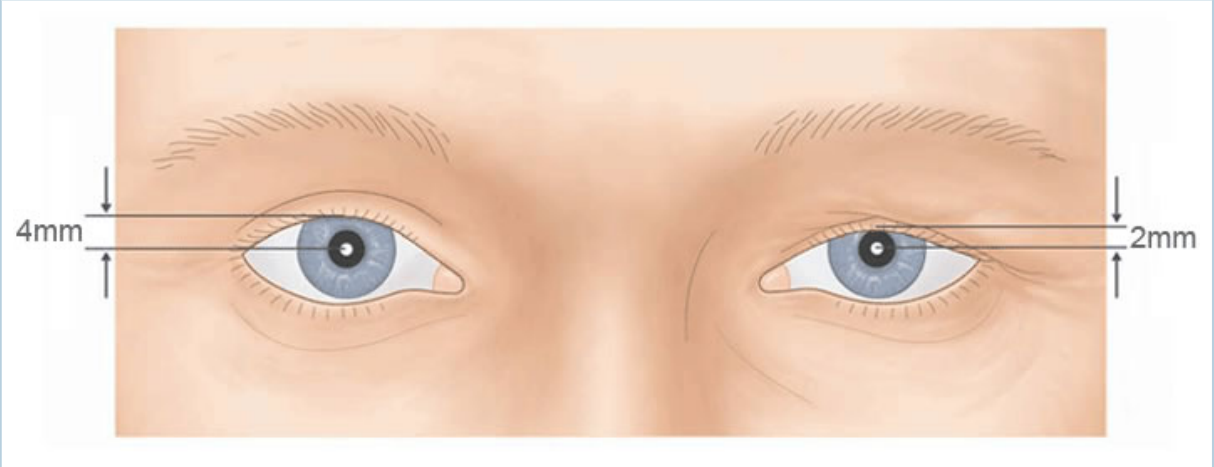

Measurement of margin-reflex distance

From the collection of Dr Allen Putterman

Laboratory and specialized testing

A complete blood count with differential is helpful, but not specific, in the assessment of ptosis. Blood cultures are indicated in the presence of an infectious process, and possibly an eyelid culture. In patients with isolated ocular and orbital findings (such as ptosis, proptosis, and strabismus) in which an infectious etiology is unlikely, thyroid function testing and an autoimmune antibody panel is helpful. Check hemoglobin A1c level in older adults with signs and symptoms of a third nerve palsy.[12]

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Specialized tests are helpful in tailoring appropriate clinical evaluation for patients with ptosis.

- Myasthenia gravis: acetylcholine receptor antibody levels, edrophonium and prostigmine stimulation, electromyographic repetitive nerve stimulation, and the ice-pack test.[28] [29]
- Multiple sclerosis: MRI brain/spinal cord, lumbar puncture with IgG level assay.
- Thyroid eye disease, eyelid or orbital tumors, and globe malposition: Hertel exophthalmometry.[30]
- Eyelid laceration: Berke levator function test for intact musculature.
- Previous eye-related surgery or implant, fracture, and globe malposition: forced duction testing.
- Syphilis: serum fluorescent treponemal antibody absorption (FTA-ABS) and direct fluorescent antibody testing (DFA-TP).
- Benign essential blepharospasm: Schirmer testing for tear production.
- Horner syndrome: cocaine and hydroxyamphetamine/tropicamide ophthalmic challenges.[31]

Imaging

Computed tomography (CT) and magnetic resonance imaging (MRI) of the orbits and brain allow precise localization of compressive lesions that cause oculomotor nerve palsies and impairment of sympathetic input.[12] Mechanical forms of ptosis require imaging if the lesion or foreign body is suspected to have orbital involvement. Eyelid edema, secondary to an inflammatory or infectious process with concomitant ocular signs and symptoms or decreased vision, warrants radiologic investigation with contrast.[26]

Orbital imaging is indicated in traumatic forms of ptosis if the eyelid laceration is associated with a deeply embedded foreign body or frank globe trauma. Orbital fracture is not an uncommon association with eyelid laceration, and is evaluated with CT of the orbits without contrast.[32] Orbital imaging with and without contrast may evaluate for soft-tissue and bony abnormalities in cases of pseudoptosis.

CT and MRI of the orbits may be considered in atypical presentations of myogenic, aponeurotic, and involutional forms of ptosis. Patients with a clear etiology of ptosis (without ocular and systemic symptoms) rarely require neuroimaging. The American College of Radiology has produced guidelines on the most appropriate diagnostic imaging tests depending on the clinical presentation.[17]

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Differentials overview

Common

- Involutional changes
- Prolapsed orbital fat
- Dermatochalasis
- Congenital myogenic ptosis
- Thyroid eye disease
- Previous eye-related surgery or implant
- Chalazion
- Stye (hordeolum)
- Uveitis
- Orbital cellulitis
- Orbital inflammatory syndrome
- Eyelid tumors
- Orbital tumors
- Chronic progressive external ophthalmoplegia (CPEO)
- Stroke
- Eyelid foreign body
- Eyelid laceration

Uncommon

- Blepharophimosis
- Myasthenia gravis
- Multiple sclerosis

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Uncommon
Blepharochalasis
Giant cell arteritis
Preseptal cellulitis
Globe malposition
Benign essential blepharospasm (BEB)
Third nerve palsy
Horner syndrome
Transection of levator muscle or aponeurosis
Orbital and facial fracture

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Differentials

Common

◊ Involutional changes

History	Exam	1st Test	Other tests
use of forehead muscles to see clearly and improve peripheral vision; worsening of peripheral vision over the course of the day; brow ache and/or headache common later in the day	facial soft-tissue laxity with brow position below superior orbital rim; horizontal forehead furrows common; suspect sequelae of peripheral facial nerve (cranial nerve VII) palsy if unilateral	»**none:** clinical diagnosis	

◊ Prolapsed orbital fat

History	Exam	1st Test	Other tests
progressive, nonerythematous, nontender upper and lower eyelid prominence	diffuse and focal prominence of the upper and lower eyelids, may vary with extraocular movement; smooth yellow tissue visible beneath tenon capsule on the lateral aspect of the globe; smooth, nontender, pale-colored masses on temporal aspect of the eye; bilateral common	»**no initial test:** clinical diagnosis	

◊ Dermatochalasis

History	Exam	1st Test	Other tests
heavy feeling around the eyes, brow ache, eyelashes visible especially on upgaze; progressively diminished superior visual field	redundancy of upper eyelid skin; skin often thin with visible subcutaneous telangiectasias, orbital fat prolapse frequent	»**no initial test:** clinical diagnosis	

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Common

◊ Congenital myogenic ptosis

History	Exam	1st Test	Other tests
ptosis, unilateral or bilateral	chin-up head position, reduced levator function, decreased supraduction (upward rotation of the eye), poor eyelid crease, poor eyelid closure, and poor eyelid excursion in downgaze	»**none:** clinical diagnosis	

Thyroid eye disease

History	Exam	1st Test	Other tests
progressive pain and discomfort around the eye; dry eye or foreign body sensation, double vision, production of tears or light sensitivity; decreased vision requires urgent ophthalmologic consultation	eyelid retraction and swelling, exophthalmos, extraocular muscle restriction, globe malposition, decreased vision and/or relative afferent pupillary defect, unilateral or bilateral	»**serum#thyroid stimulating hormone:** low, normal, or elevated Hyperthyroidism is not necessary for diagnosis; subset of patients may be hypothyroid or euthyroid. »**serum free T4:** elevated, normal, or low Values should be rechecked if no evidence of abnormal thyroid function or regulation. »**Hertel exophthalmometry:** elevated (>20 mm) or normal Excessive globe proptosis may cause corneal exposure secondary to poor and inadequate eyelid closure (lagophthalmos).	»**visual field assessment:** constriction of peripheral isopters, scotomas variable Progressive visual field abnormalities suggest optic nerve dysfunction »**MRI or CT of orbits:** extraocular muscle enlargement sparing tendons, increased volume of retro-orbital fat, globe proptosis Extraocular muscle enlargement at the orbital apex may cause compressive optic neuropathy with resultant permanent visual loss.

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Common

◊ Previous eye-related surgery or implant

History	Exam	1st Test	Other tests
eyelid malposition following orbital fracture repair, previous gold weight eyelid implants for paralytic lagophthalmos or a scleral buckle for retinal detachment repair, extraocular muscle surgery for strabismus, filtering and shunting surgery for glaucoma; progressive symptoms	eyelid ptosis or retraction, unilateral or bilateral; depending on the nature of the implant, variable measurements of eyelid position and motility; surgical anatomic changes with associated implanted hardware	»**forced duction testing:** restriction and limitation of ocular movement May require modification or removal of implanted hardware.	»**CT of orbits:** malpositioned or migrated implantable device May require modification, replacement, or removal of implanted hardware.

◊ Chalazion

History	Exam	1st Test	Other tests
blepharitis and ocular rosacea are frequently associated; dry eye and foreign body sensation are common; production of tears and pain may be variable	local or diffuse erythema; moderately firm nodule palpable over eyelid; circumscribed nodular swelling with meibomian gland inspissation (mucus plug of duct); fluorescein staining may show rapid tear break-up time; recurrent, chronic, or multiple lesions may require ophthalmologic consultation	»**eyelid biopsy:** chronic granulomatous inflammation Chronic or recurrent lesions may be suspicious for underlying malignancy, such as sebaceous cell carcinoma.	

◊ Stye (hordeolum)

History	Exam	1st Test	Other tests
acute and subacute swelling of the eyelid margin; variable pain; occasionally frank purulent discharge	focal or diffuse eyelid swelling with overlying erythema, typically tender to palpation; frequently peaked to skin surface with purulent drainage; progressive, worsening periorbital edema and erythema with	»**eyelid culture:** positive for normal skin flora in majority of cases Large or atypical lesions may reveal aggressive and	»**CT of orbits:** orbital abscess formation with adjacent soft-tissue stranding may represent cellulitis Orbital cellulitis should be treated aggressively, especially

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Common

◊ Stye (hordeolum)

History	Exam	1st Test	Other tests
	ocular symptomatology requires urgent ophthalmologic evaluation	resistant bacterial strains.	in immunocompromised or at-risk patients.

Uveitis

History	Exam	1st Test	Other tests
ocular pain and discomfort with redness and photophobia; pain with extraocular movement and flashes are not uncommon; variable onset of symptoms; correlate with constitutional symptoms in possible systemic disease	decreased vision; conjunctival injection; anterior chamber cell and flare; hypopyon; vitreous haze and retinal and optic nerve edema are common with posterior disease; presence of profoundly decreased vision or a relative afferent pupillary defect requires urgent ophthalmologic evaluation	»**CBC with differential:** leukocytosis Reflects immune activation; does not differentiate inflammatory or infectious etiologies.	»**fluorescent treponemal antibody absorption (FTA-ABS):** current or previous syphilis infection Curable etiology for uveitis; may require intravenous penicillin therapy for neurosyphilis. »**serum direct fluorescent antibody testing (DFA-TP):** current or previous syphilis infection Curable etiology for uveitis; may require intravenous penicillin therapy for neurosyphilis. »**serum Treponema pallidum antibody:** current or previous syphilis infection Curable etiology for uveitis; may require intravenous penicillin therapy for neurosyphilis.

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Common

Orbital cellulitis

History	Exam	1st Test	Other tests
acute and chronic sinusitis (>90%), recent dental procedure, systemic and intracranial infection, immunocompromised, recent eye and orbital surgery, trauma with and without foreign body	decreased vision; strabismus and diplopia variable; pain with extraocular movement, proptosis, chemosis, fever; relative afferent pupillary defect requires urgent ophthalmologic consultation	»**CBC with differential:** leukocytosis Nonspecific indicator of disease activity. »**blood cultures:** identification of pathogen Initiation of antibiotics should not be delayed while waiting for results; broad-spectrum coverage should be started. »**CT of orbits with contrast:** sinusitis, abscess, foreign body Presence of sinusitis requires otolaryngologic consultation.	

◊ Orbital inflammatory syndrome

History	Exam	1st Test	Other tests
acute or insidious onset of eye pain, eyelid swelling and eye redness; double vision; previous episodes; systemic symptoms (headache, lethargy, nausea, vomiting)	eyelid edema and erythema; ptosis variable; pain with extraocular movements; chemosis, proptosis, strabismus and diplopia; decreased visual acuity, visual field defects, relative afferent pupillary defect; must differentiate from orbital cellulitis	»**CT of orbits with contrast or MRI:** lacrimal gland enlargement (most common), extraocular muscle thickening involving tendons (contrast to thyroid eye disease), fat-stranding Classic signs on imaging with typical clinical presentation may obviate surgical biopsy.	»**Hertel exophthalmometry:** elevated (>20 mm) or normal Reflect orbital congestion; often improves to normal with treatment. »**visual field assessment:** visual field constriction, scotomas Often improves to normal with treatment. »**B-scan ultrasound:** posterior sclera and tendon capsule edema

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Common

◊ Orbital inflammatory syndrome

History	Exam	1st Test	Other tests
			Performed by ophthalmologist; may reveal exudative retinal detachment. »**CBC with differential:** leukocytosis, eosinophilia Not specific for etiology of inflammatory process. »**serum erythrocyte sedimentation rate:** elevated Not specific for etiology of inflammatory process; atypical cases of giant cell arteritis may present similarly. »**serum antinuclear antibodies:** abnormal May reveal autoimmune etiology.

◊ Eyelid tumors

History	Exam	1st Test	Other tests
middle-aged to older adult, fair complexion, history of prolonged sun exposure, Northern European descent; cigarette smoking, previous skin cancers, immunosuppressed patients (particularly post-transplant patients)	upper eyelid lesions with ptosis; firm, raised, pearly nodule; adjacent telangiectasias; central ulceration, lash loss, disruption of normal eyelid architecture; entropion and ectropion variable, strabismus and diplopia variable; prominent keratinization suggests squamous cell carcinoma, multicentric lesions with yellow appearance suggest sebaceous	»**Hertel exophthalmometry:** unilateral proptosis Malignant lesions of the medial and lateral canthus may extend posteriorly into the orbit.	»**nasolacrimal irrigation:** proximal and distal obstruction Performed by an ophthalmologist; medial canthal lesions may constrict or involve the lacrimal drainage apparatus. »**CT of orbits:** extent of eyelid and orbital involvement, sinus lesions

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Common

◊ Eyelid tumors

History	Exam	1st Test	Other tests
	cell carcinoma, darkly pigmented lesions suggest melanoma; must check preauricular and submandibular lymph nodes		Consider in lesions involving the medial and lateral canthal regions and lesions associated with strabismus. »**serum alanine aminotransferase, aspartate aminotransferase, gamma-GT:** abnormal Metastasis of melanoma and sebaceous cell carcinoma often hematogenous.

◊ Orbital tumors

History	Exam	1st Test	Other tests
acute and insidious proptosis; eyelid asymmetry, recent changes in refractive error, decreased vision, variable double vision; previous malignancies suggest metastatic spread	eyelid ptosis and retraction, decreased visual acuity; color vision and visual field defects variable; proptosis, strabismus and diplopia, conjunctival vascular dilatation, relative afferent pupillary defect	»**CT of head, orbits with and without contrast:** tumor (diffuse and encapsulated), bony changes (remodeling and hyperostosis), soft-tissue involvement, optic nerve impingement Contrast images useful in vascular lesions.	»**Hertel exophthalmometry:** unilateral proptosis Difference more important than absolute values; may follow over time to gauge progression. »**visual field assessment:** visual field constriction, scotomas Presence and extent of visual field loss important in treatment algorithm. »**MRI of orbits with and without contrast:** posterior tumors, optic nerve involvement

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Common

◊ Orbital tumors

History	Exam	1st Test	Other tests
			Excellent in defining soft-tissue composition of lesions; limited utility for surgical planning. »**forced duction and generation testing:** extraocular muscle restriction and paresis Performed by ophthalmologist; diffuse lesions may present with muscle restriction. »**retinoscopy:** hyperopic shift Performed by ophthalmologist; retrobulbar lesions may compress the globe in the anteroposterior dimension.

◊ Chronic progressive external ophthalmoplegia (CPEO)

History	Exam	1st Test	Other tests
slowly progressive limitation of eye movements with drooping of eyelids; cardiac arrhythmias and maternal inheritance suggest Kearns-Sayre syndrome; later onset with difficulty eating and swallowing with French-Canadian ancestry suggests oculopharyngeal dystrophy	bilaterally symmetric limitation of extraocular movement, ptosis, generalized weakness; diplopia uncommon	»**ECG:** conduction abnormalities Triad of CPEO, pigmentary retinopathy, and cardiac conduction abnormalities is highly suggestive of Kearns-Sayre syndrome; mitochondrial analysis warranted.	

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Common

Stroke

History	Exam	1st Test	Other tests
typically middle-aged or older adult, acute onset of vision loss (central and peripheral), facial and peripheral weakness, slurred speech, ataxia, numbness and tingling; incomplete resolution of symptoms, extent of ophthalmologic symptoms depends on carotid versus vertebrobasilar circulation	visual acuity decreased, visual field defects variable, ptosis secondary to generalized facial weakness or involvement of cranial nerve nuclei; strabismus and diplopia variable	»**non-contrast CT of head:** territory of infarction or hemorrhage Rapid and easily attained; not useful early in course of ischemic events.	»**MRI of brain:** territory of infarction or hemorrhage Diffusion-weighted imaging is most sensitive in detecting acute ischemic events. »**carotid duplex scan:** atherosclerotic carotid occlusive disease »**transesophageal echocardiogram:** source of thrombi, patent foramen ovale, atrial myxoma »**CBC:** abnormal Supportive in hemorrhagic events; useful in atypical presentations. »**serum prothrombin time, PTT:** abnormal Supportive in hemorrhagic events; useful in atypical presentations.

Eyelid foreign body

History	Exam	1st Test	Other tests
unilateral, focal eyelid swelling following trauma or surgery; redness, tenderness, and discharge depending on nature of foreign body	focal eyelid lesion with possible erosion through skin; edema, erythema, tenderness, and variable discharge, common with vegetable matter, less common with surgical materials	»**CT of orbits:** location and size of foreign body Concurrent globe trauma and rupture should be evaluated for; metallic foreign bodies are clearly visible; nonmetallic and vegetable foreign bodies can be	

DIAGNOSIS

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Common

Eyelid foreign body

History	Exam	1st Test	Other tests
		discerned by relative radiolucency and adjacent soft-tissue changes.	

Eyelid laceration

History	Exam	1st Test	Other tests
men more than women; decreased vision, difficulty and/or inability in moving eyelids; diplopia variable	decreased visual acuity, mild to severe ptosis, strabismus, variable diplopia; orbital fat prolapsed from wound; decreased vision or relative afferent pupillary defect requires urgent ophthalmologic consultation	»**Berke levator function:** decreased levator function (<10 mm), significant asymmetry Orbital fat prolapse from the wound with decreased levator function suggests traumatic laceration and dehiscence of the levator aponeurosis.	»**CT of orbits:** fracture, foreign body, extraocular muscle entrapment Contrast imaging not required in acute setting; direct axial and coronal and sagittal thin cuts preferable for evaluation of foreign body or intracanalicular optic nerve impingement. »**forced duction and generation testing:** extraocular muscle restriction and paresis Bradycardia induced by extraocular movement may require urgent surgical reduction and repair. »**B-scan ultrasound:** vitreous hemorrhage, retinal detachment, globe rupture Performed by ophthalmologist for evaluation of posterior segment if view inadequate.

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Uncommon

◊ Blepharophimosis

History	Exam	1st Test	Other tests
facial anomalies present from birth, family members with similar facial features; eyes appear disproportionately small, difficulty opening eyes	significant upper eyelid ptosis, wide intercanthal separation (telecanthus); medial eyelid skin fold extending from lower to upper eyelid (epicanthus inversus)	»**no initial test:** clinical diagnosis	

Myasthenia gravis

History	Exam	1st Test	Other tests
fluctuating eyelid position and diplopia over time, symptoms worse in evening and after exertion; systemic symptoms (e.g., difficulty eating, breathing, speaking, movement)	variable measurements of eyelid position and motility; variable field of diplopia; fatigability with prolonged upgaze; dyspnea, dysphagia are potentially life-threatening and require prompt evaluation	»**acetylcholine receptor antibody test:** presence of binding, blocking, or modulating antibodies; presence of antimuscle-specific kinase (MuSK) antibodies Binding and modulating antibodies commonly present in cases of generalized myasthenia gravis. »**serum thyroid function tests:** abnormal function and regulation High comorbidity of thyroid dysfunction with secondary thyroid eye disease. »**serum#antinuclear antibodies:** autoimmune activation High comorbidity of other autoimmune disease, such as systemic lupus erythematosus.	»**edrophonium and prostigmine test:** improvement of symptoms after administration Potential major adverse effects, including bradycardia and respiratory arrest; monitoring of vital signs with resuscitation equipment available is necessary. »**ice-pack test:** improvement of symptoms after placement of ice pack for several minutes Patient must manifest eyelid malposition in order to gauge improvement. »**electromyographic#repetitive nerve stimulation:** decremental response Single-fiber electromyography is most sensitive.

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Uncommon

Myasthenia gravis

History	Exam	1st Test	Other tests
		»**CT of chest:** anterior mediastinal mass indicating thymoma Thymomas present in 10% or less of patients.	

◊ Multiple sclerosis

History	Exam	1st Test	Other tests
women more than men; young and middle-aged; eye symptoms (pain with movement, blurred vision, decreased peripheral vision); prior and coincident neurologic symptoms	decreased vision, diplopia, visual field defects, variable optic disk edema, relative afferent pupillary defect; must evaluate for ataxia, bowel, and bladder dysfunction, and peripheral weakness	»**MRI of brain:** disseminated white matter plaques Neurologic consultation mandatory.	»**lumbar puncture:** elevated IgG with oligoclonal bands Not specific for multiple sclerosis.

◊ Blepharochalasis

History	Exam	1st Test	Other tests
adolescent or young adult with repeated episodes of painless eyelid swelling; unilateral or bilateral; may have history of preceding physical and emotional stress or allergy	nonerythematous eyelid edema with thinning and redundancy of the skin, superficial telangiectasias, blepharoptosis, orbital fat and lacrimal gland prolapse	»**CT of orbits:** anterior orbital soft-tissue swelling without abscess formation Imaging may be used to differentiate from thyroid orbitopathy, idiopathic orbital inflammation, or other processes.	

Giant cell arteritis

History	Exam	1st Test	Other tests
older adult with history of a temporal headache, vision loss, and variable ocular or orbital discomfort;	jaw claudication, ptosis, palpable tenderness along the course of the superficial temporal artery and lack of	»**erythrocyte sedimentation rate (ESR):** elevated Take blood for measuring ESR	»**high-resolution MRI:** mural inflammation or luminal changes of cranial or extracranial arteries

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Giant cell arteritis

History	Exam	1st Test	Other tests
fatigue, lethargy, loss of appetite, and weight loss	pulsation of this artery; vision loss may be significant, symptoms may develop in the contralateral eye; urgent ophthalmologic evaluation is required	before starting a high-dose glucocorticoid, as inflammatory markers decrease once glucocorticoid therapy is started.[33] [34] [35] »**C-reactive protein (CRP):** elevated Take blood for measuring CRP before starting a high-dose glucocorticoid, as inflammatory markers decrease once glucocorticoid therapy is started.[33] [34] [35] »**CBC:** patients with giant cell arteritis may have a normochromic, normocytic anemia with a normal WBC count and elevated platelet count; mild leukocytosis may occur Take blood for CBC before starting a high-dose glucocorticoid.[34] [35] »**vascular ultrasonography:** mural inflammatory changes If available, use rapid-access vascular ultrasonography of the temporal and axillary artery first line to diagnose suspected giant cell arteritis.[33] [34] [36] [37]	High-resolution MRI can be used as an alternative to ultrasonography for the assessment of cranial and extracranial arteries in suspected giant cell arteritis.[36]

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Uncommon

Giant cell arteritis

History	Exam	1st Test	Other tests
		»**temporal artery biopsy:** histopathology typically shows granulomatous inflammation Recommended by the ACR/EULAR as the definitive test for the diagnosis of giant cell arteritis (GCA).[38] However, imaging is being increasingly used for diagnosis and ultrasound is the preferred early imaging test.[36] [37] For patients with suspected GCA, initially, a unilateral biopsy is recommended.[37] In addition, consider a temporal artery biopsy if expertize for ultrasonography is not available, or if the patient's pre-test probability is high (typical features of GCA with raised inflammatory markers) but the ultrasonography is normal.[34] Do not delay treatment while waiting for the biopsy to be performed.[34]	

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Preseptal cellulitis

History	Exam	1st Test	Other tests
recent penetrating trauma to the skin; cutaneous source of infection, sinusitis, infected chalazion; production of tears or mucopurulent discharge, insect bite	eyelid edema and warmth, cutaneous infection; production of tears, decreased vision or any ocular symptoms suggest orbital involvement	»**CBC with differential:** leukocytosis Low threshold for starting intravenous antibiotics in children or immunocompromised patients. »**CT of orbits with contrast:** sinusitis, superficial foreign body Low threshold to order if no direct site of inoculation, sinusitis, or poor response to oral antibiotics.	

Globe malposition

History	Exam	1st Test	Other tests
previous ocular and eye muscle surgery and/or congenital anomaly; diplopia or decreased visual acuity variable; trauma variable; if progressive, correlate with systemic symptoms	hypertropia, enophthalmos, anophthalmos (congenital absence of ocular tissue), phthisis bulbi (atrophy, degeneration of a blind eye), or a superior sulcus deformity	»**forced duction testing:** restriction and limitation of ocular movement May be related to previous surgical intervention or congenital maldevelopment.	»**Hertel exophthalmometry:** asymmetry in globe position Deviation may reflect size of globe, atrophy of orbital tissues, or changes in orbital volume. »**CT of orbits:** fracture; eyelid and orbital lesion; soft-tissue enlargement, especially extraocular muscles Eyelid and orbital lesions may represent metastatic disease; inflammatory and infectious etiologies

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Globe malposition

History	Exam	1st Test	Other tests
			may cause soft-tissue changes.

◊ Benign essential blepharospasm (BEB)

History	Exam	1st Test	Other tests
women more than men; middle-aged, increased involuntary blinking, must differentiate from severe dry eye syndrome	involuntary, episodic contractions involving the orbicularis oculi, procerus, and corrugator muscles; visual acuity variable	»**Schirmer testing:** <10 mm in 5 minutes Supportive data for diagnosis of BEB and dry eye syndrome. Measures amount of tear moisture accumulated on filter paper over 5-minute intervals.	

Third nerve palsy

History	Exam	1st Test	Other tests
history of hypertension, diabetes mellitus, arteriosclerotic disease; older adult, significant eyelid droop, double vision, pain variable; younger patient, pupillary dysfunction, multiple cranial nerve palsies; requires urgent evaluation	significant ptosis, strabismus, diplopia, mild to severe orbital pain; relative afferent pupillary defect; requires urgent neurologic consultation	»**HbA1c:** elevated Complete ptosis and third nerve palsy with pupil-sparing highly suggestive of microvascular disease. »**CBC with differential:** leukocytosis Useful in atypical presentations (e.g., infection, leukemia, and lymphoma).	»**CT angiography:** aneurysm of the internal carotid or posterior communicating artery Pupillary dysfunction with cranial nerve palsy; CT angiography better for evaluation of subarachnoid hemorrhage. »**magnetic resonance angiography:** aneurysm of the internal carotid or posterior communicating artery Pupillary dysfunction with cranial nerve palsy.

DIAGNOSIS

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Third nerve palsy

History	Exam	1st Test	Other tests
			»**carotid angiography:** aneurysm of the internal carotid or posterior communicating artery Test of choice for detection of intracranial aneurysms. »**CT of orbits and brain:** tumor, infection, trauma, inflammation Should be performed if pupil-sparing third nerve palsy does not fully resolve in 3 months and in all atypical presentations. »**MRI of orbits and brain:** tumor, infection, trauma, inflammation Should be performed if pupil-sparing third nerve palsy does not fully resolve in 3 months and in all atypical presentations. »**serum angiotensin-converting enzyme and lysozyme:** elevated in neurosarcoidosis Useful in atypical presentations.

Horner syndrome

History	Exam	1st Test	Other tests
mild unilateral eyelid droop; eye appears smaller, pupil asymmetry;	mild to moderate unilateral ptosis, ipsilateral pupillary miosis; facial	»**cocaine test:** minimal to no improvement of miosis	»**hydroxyamphetamine/tropicamide ophthalmic test:**

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Horner syndrome

History	Exam	1st Test	Other tests
inability to sweat variable, headache may be associated with intermittent associated neurologic symptoms, requires neurologic consultation; cough, hemoptysis, and neck swelling suggest oncologic etiology	anhidrosis variable, iris heterochromia in congenital cases; decreased visual acuity and/or ocular dysmotility with strabismus demands further investigation	Administration of topical cocaine eye drops blocks the reuptake of norepinephrine at the neuromuscular junction. Patients with Horner syndrome have minimal to no release of norepinephrine, therefore cocaine administration will have no effect.	minimal to complete resolution of miosis Administration of topical hydroxyamphetamine/ tropicamide ophthalmic eye drops stimulates the release of norepinephrine from the presynaptic terminal. A postganglionic lesion (third-order neuron) will dilate poorly. A preganglionic lesion (first- and second-order neuron) will dilate normally. »**CT of the chest:** mass lesion in apex of lung, previous chest surgery, thoracic aortic aneurysms Associated with second-order neuron lesions. »**MRI of brain and neck:** lesions of the brainstem and upper cervical spinal cord, tumors, cervical disk disease, changes of the internal carotid artery or cavernous sinus Associated with first- and third-order neuron lesions. »**carotid angiography:** aneurysm of the internal carotid or posterior communicating artery

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Horner syndrome

History	Exam	1st Test	Other tests
			Diagnostic standard for investigation of aneurysm or dissection.

Transection of levator muscle or aponeurosis

History	Exam	1st Test	Other tests
lacerations or contusive trauma to the eyelid; history of eyelid surgery	herniation of orbital fat from an eyelid laceration with or without decreased vision, inability to elevate the upper eyelid	»**Berke levator function:** decreased levator function (<10 mm), significant asymmetry	

Orbital and facial fracture

History	Exam	1st Test	Other tests
men more than women; trauma to orbital and intracranial structures and adjacent paranasal sinuses, decreased visual acuity, pain and discomfort on attempted eye movement; diplopia variable; systemic symptoms (e.g., nausea, vomiting, headache, loss of consciousness)	drooped and retracted eyelid, decreased vision, incomitant strabismus, ptosis, subconjunctival hemorrhage, hyphema; relative afferent pupillary defect requires evaluation for globe rupture, retinal detachment, and traumatic optic neuropathy	»**CT of orbits:** fracture, foreign body, extraocular muscle entrapment Contrast imaging not required in acute setting; direct axial, coronal, and sagittal thin cuts with bone windows preferable for evaluation of foreign body or intracanalicular optic nerve impingement.	»**forced duction and generation testing:** extraocular muscle restriction and paresis Bradycardia induced by extraocular movement may require urgent surgical reduction and repair. »**B-scan ultrasound:** vitreous hemorrhage, retinal detachment, globe rupture Performed by ophthalmologist for evaluation of posterior segment if view inadequate.

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Online resources

1. Benign Essential Blepharospasm Research Foundation *(external link) (http://www.blepharospasm.org)*

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Key articles

- Finsterer J. Ptosis: causes, presentation, and management. Aesthetic Plast Surg. 2003;27:193-204. Abstract
- Yanovitch T, Buckley E. Diagnosis and management of third nerve palsy. Curr Opin Ophthalmol. 2007 Sep;18(5):373-8. Abstract
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Images

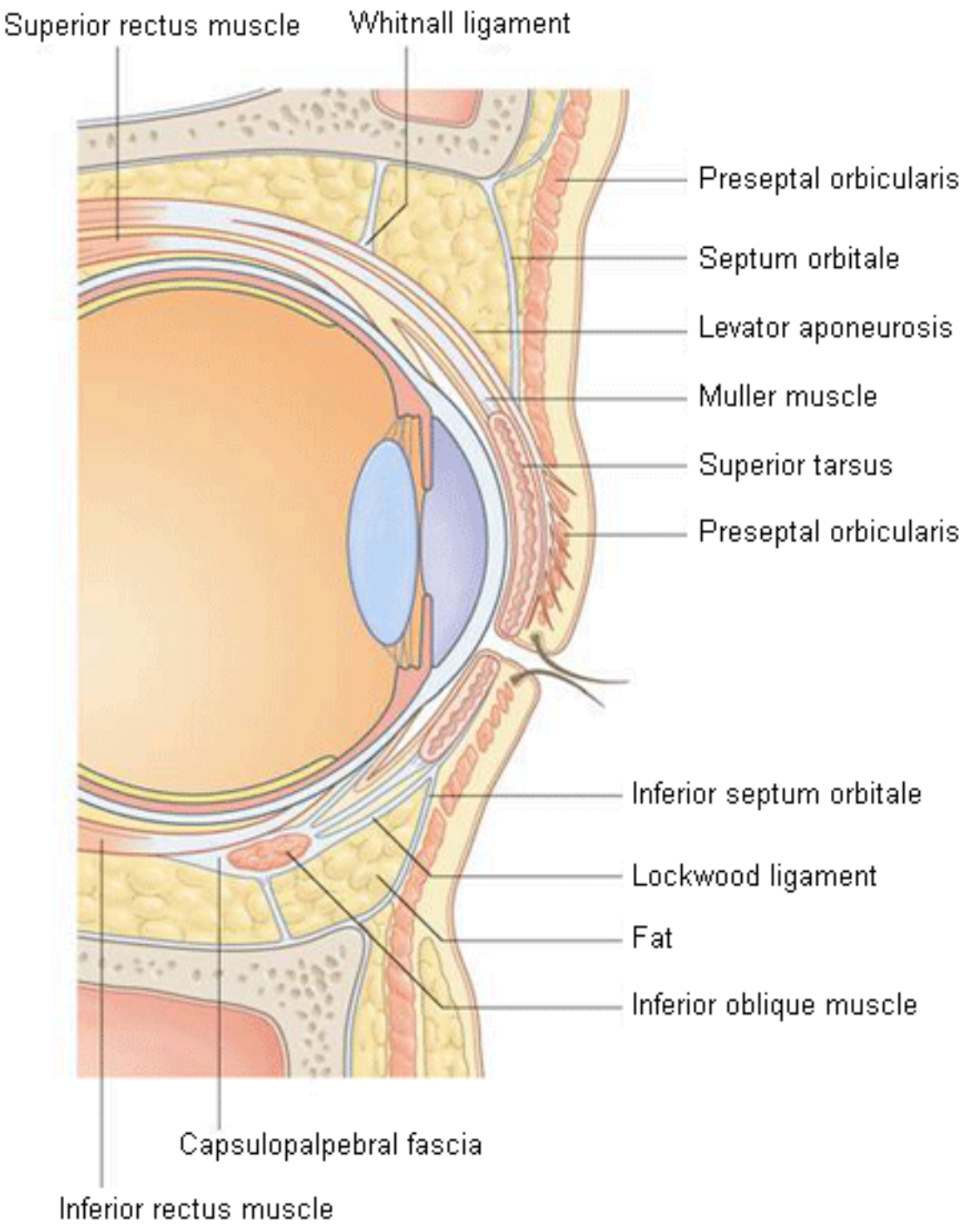

Figure 1: Sagittal view of eyelid anatomy

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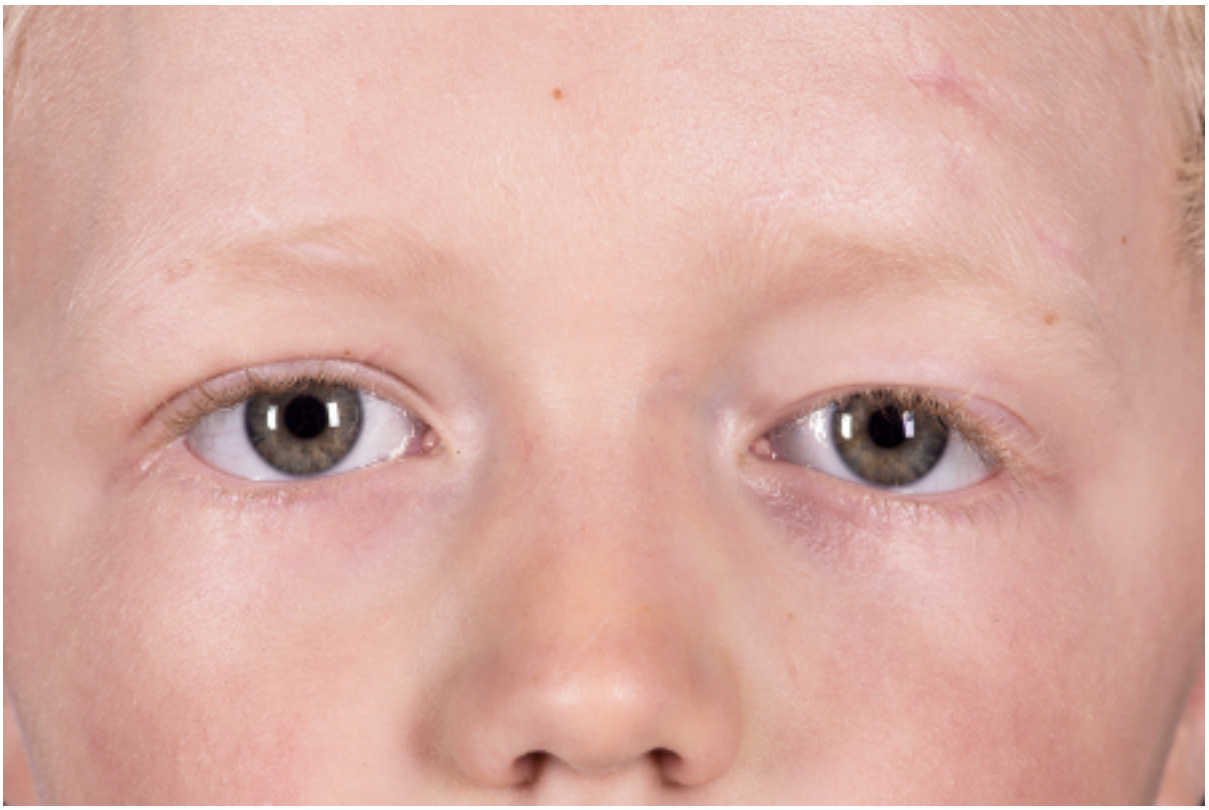

Figure 2: Bilateral, asymmetric, congenital myogenic ptosis

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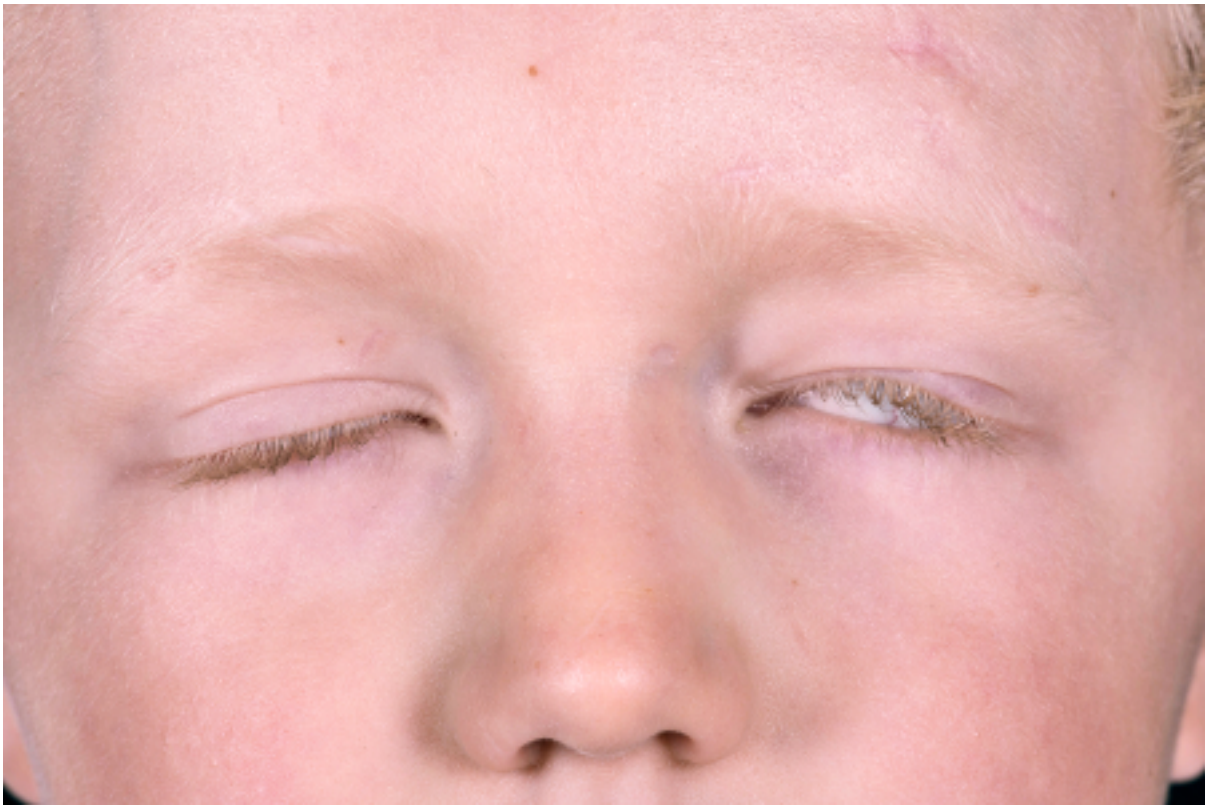

Figure 3: Ptosis in a 6-year-old boy. Ptosis is normally due to weakness of the levator muscle of the upper eyelid, here of the left eye (at right). This patient has had this condition since birth, and has had three operations aimed at correcting the condition

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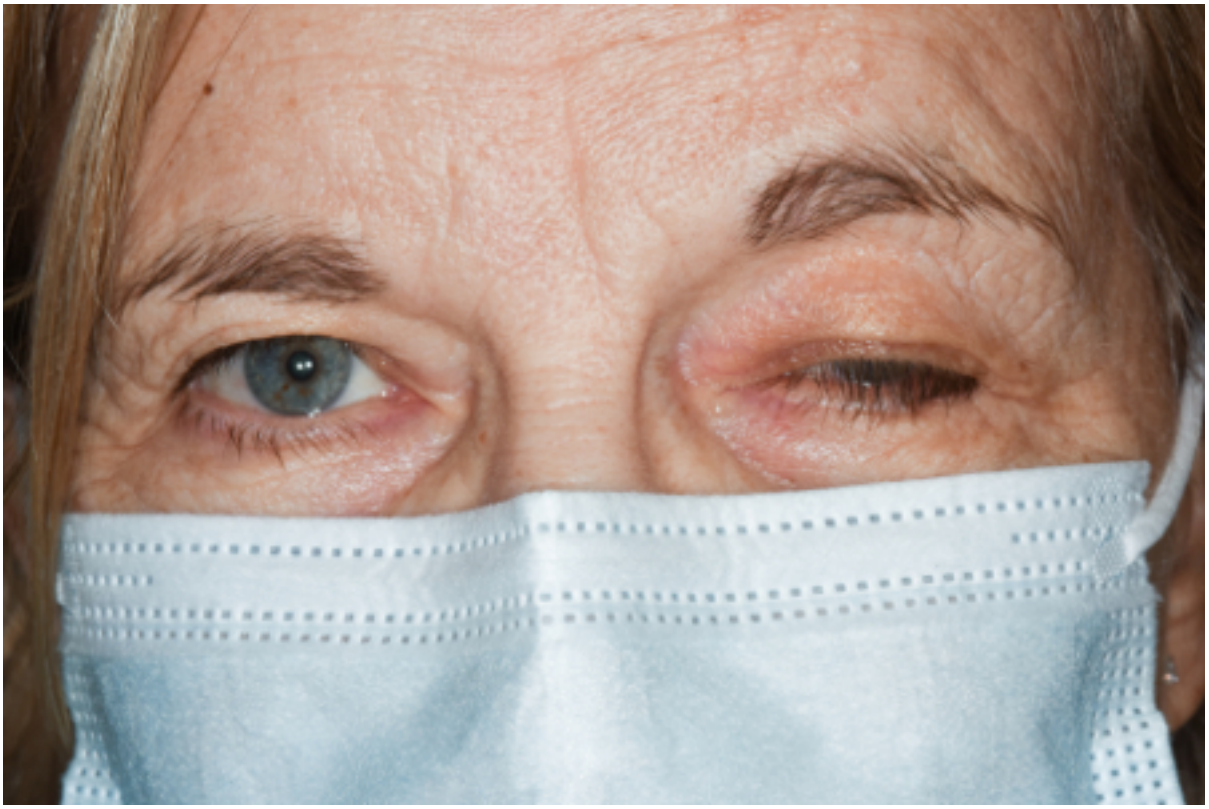

Figure 4: Drooping eyelid (ptosis) in 69 year old female patient due to myasthenia gravis (MG). MG is a rare autoimmune neuromuscular disorder that weakens and fatigues the body's voluntary muscles, which include the muscles that control movement of the eyes and eyelids

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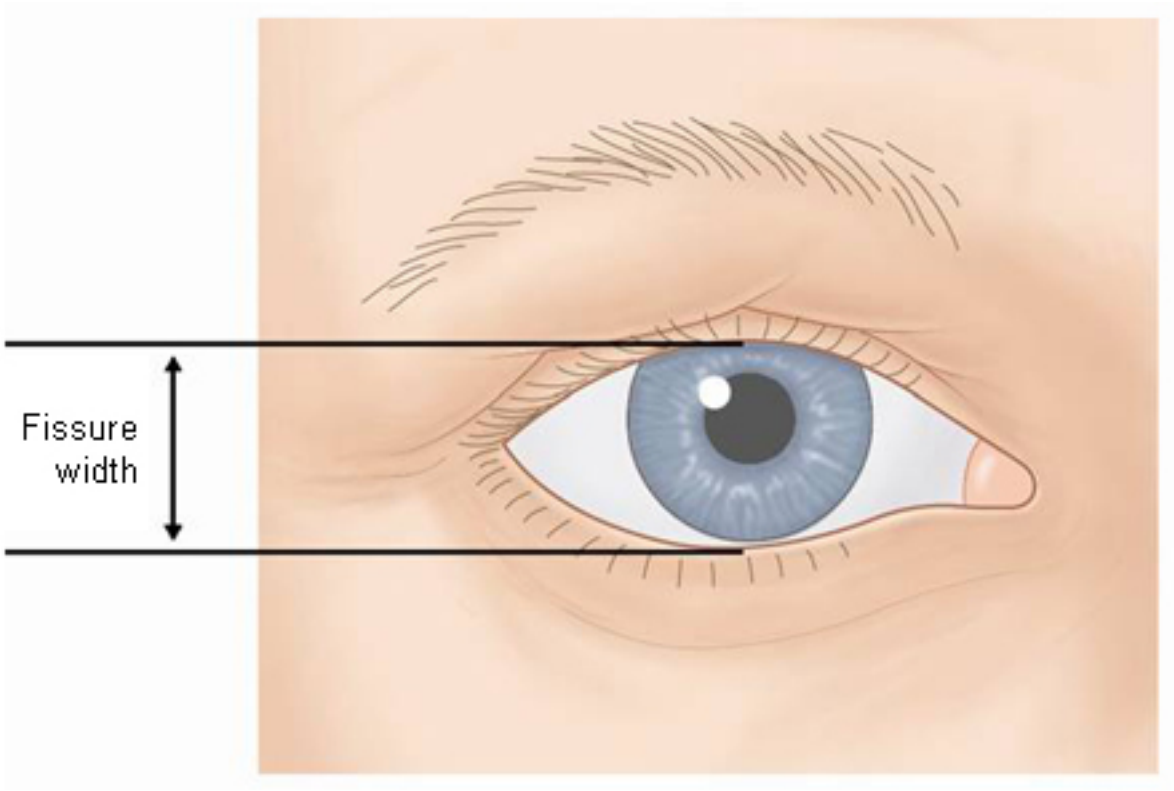

Figure 5: Measurement of vertical interpalpebral fissure

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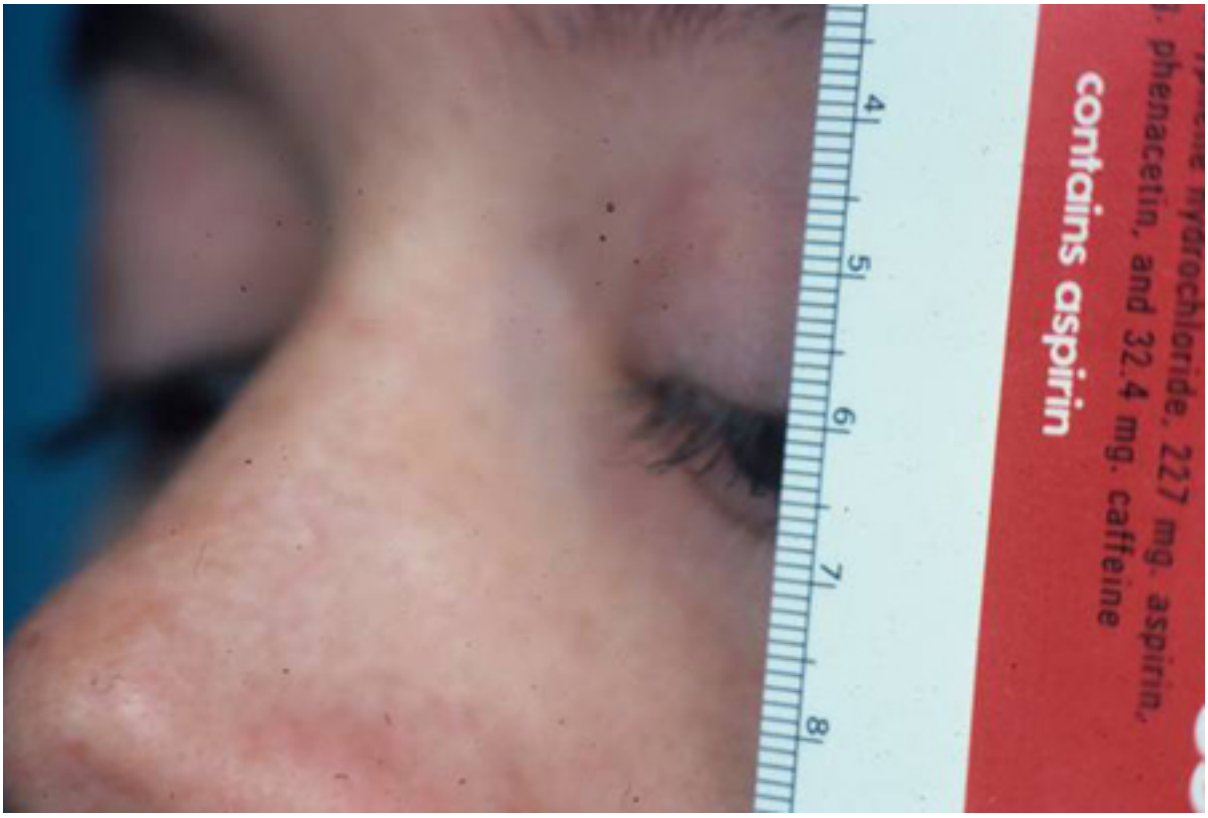

Figure 6: Position of upper eyelid in downgaze

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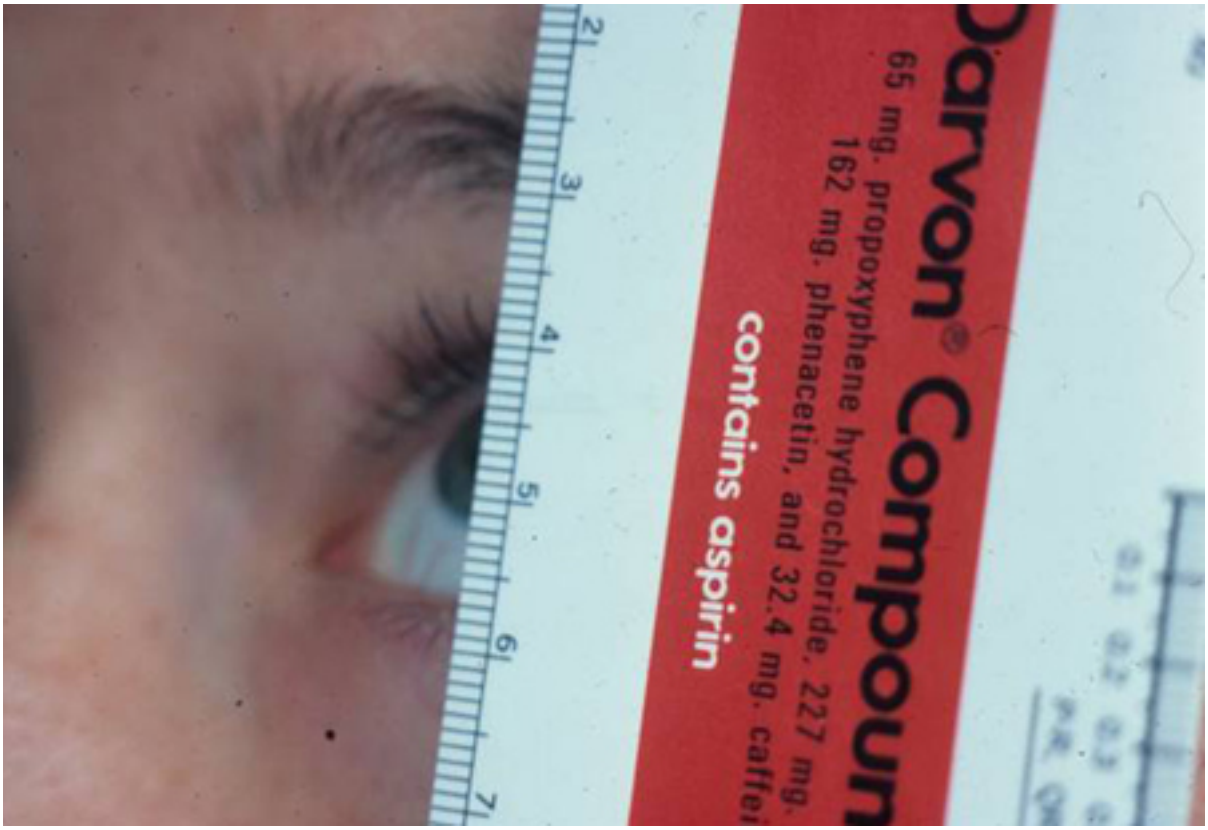

Figure 7: Position of upper eyelid in upgaze

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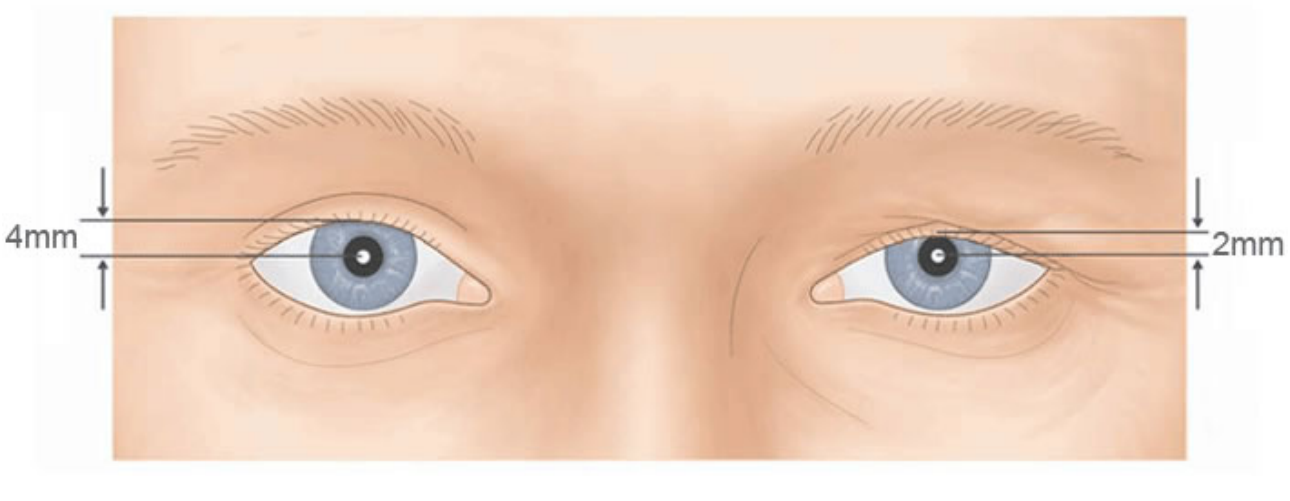

Figure 8: Measurement of margin-reflex distance

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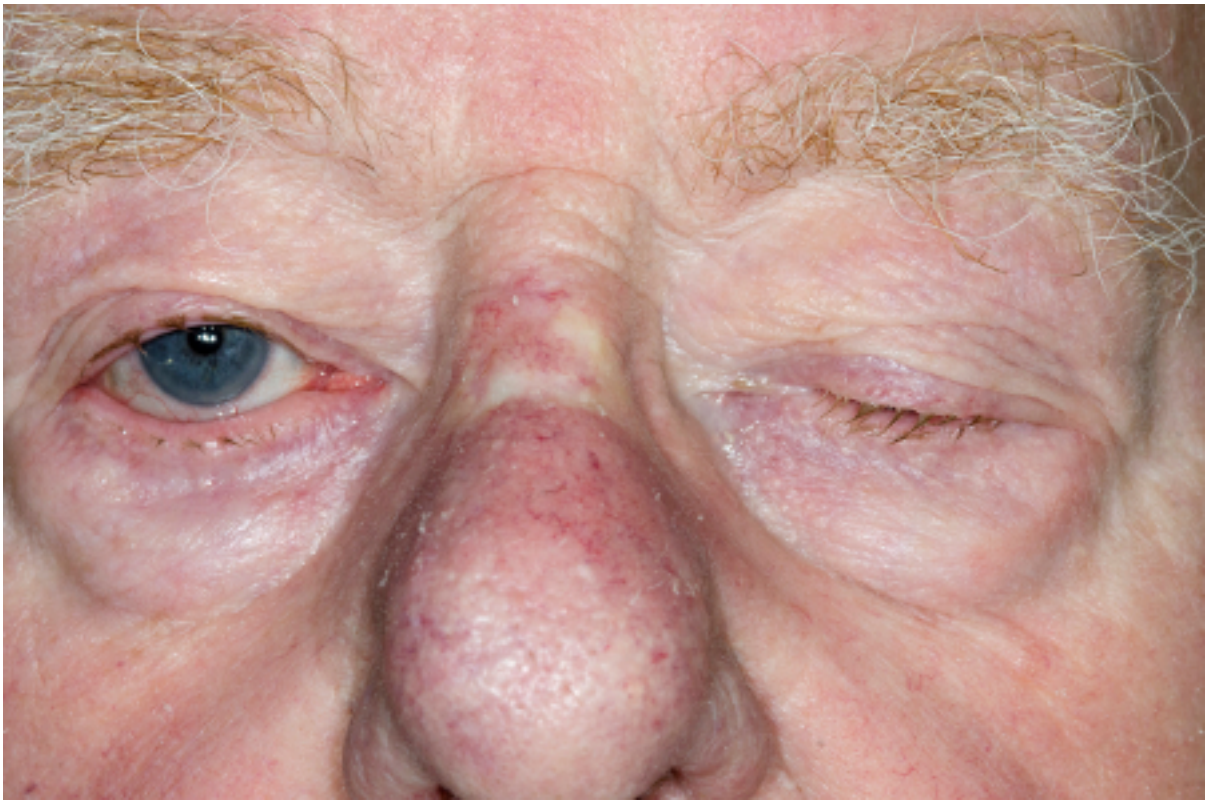

Figure 9: Ptosis in an 89 year old male patient following a botox injection to correct double vision (diplopia)

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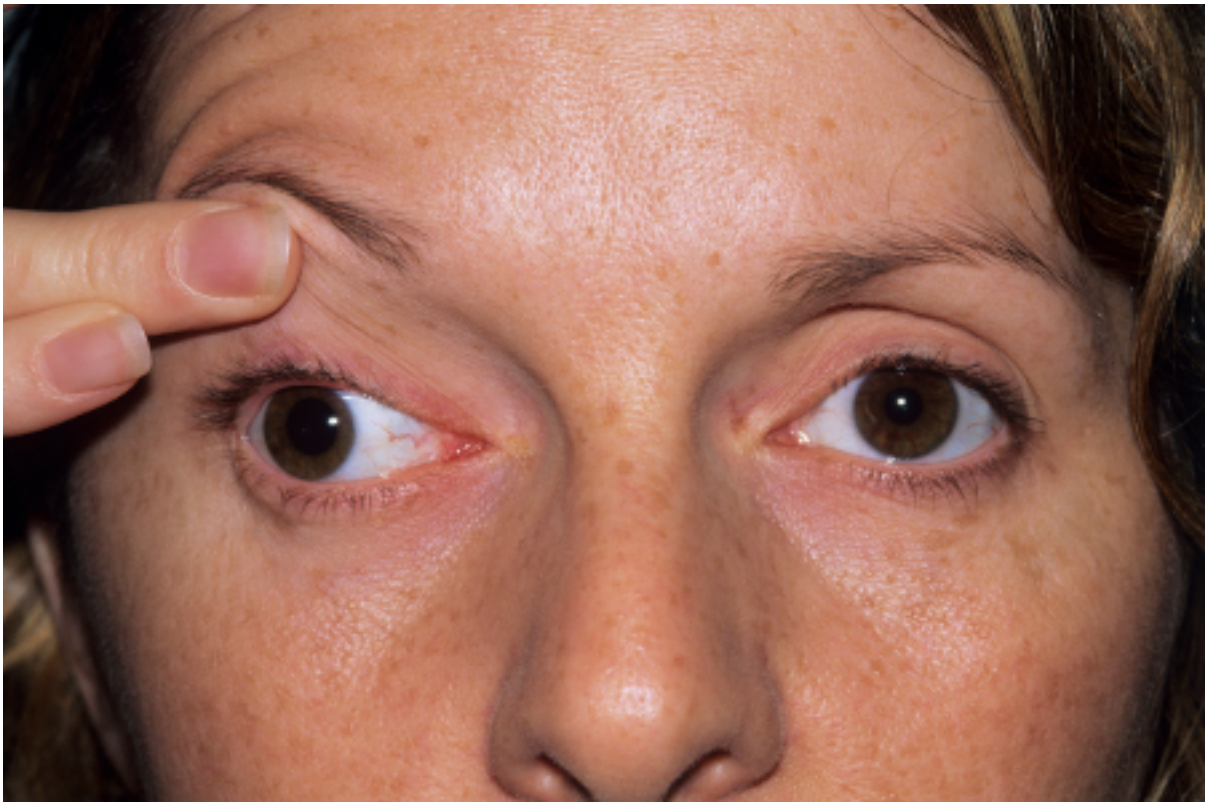

Figure 10: Oculomotor nerve palsy. Face of a 36-year-old woman with third nerve palsy after surgery to treat a subarachnoid haemorrhage. A berry aneurysm, a common localised dilation of an intercranial artery, caused the subarachnoid hemorrhage. Third nerve palsy is a dysfunction of the third cranial nerve, the oculomotor nerve, which controls the movement of the eyes. It leads to an inability to move the eye, double vision, a fixed and non-reactive pupil and eyelid drooping (ptosis, seen here, right eye)

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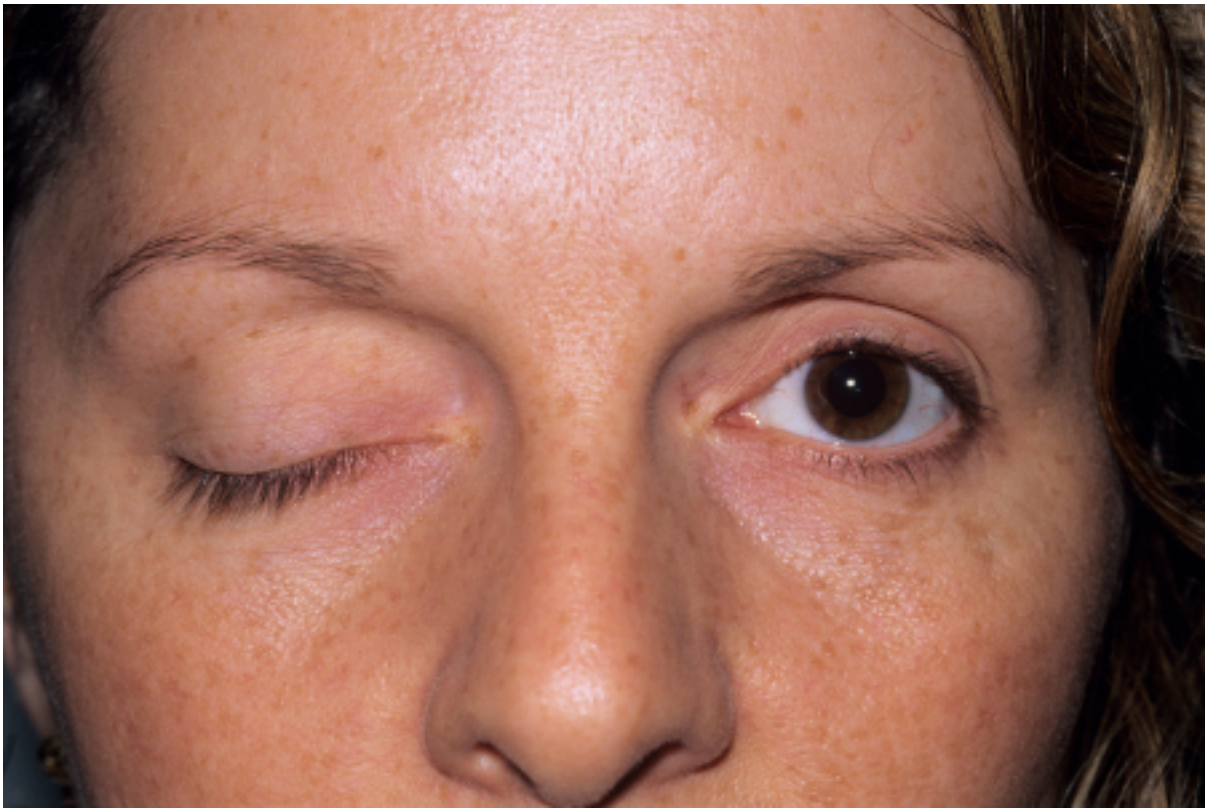

Figure 11: Oculomotor nerve palsy. Face of a 36-year-old woman with third nerve palsy after surgery to treat a subarachnoid haemorrhage. A berry aneurysm, a common localised dilation of an intercranial artery, caused the subarachnoid hemorrhage. Third nerve palsy is a dysfunction of the third cranial nerve, the oculomotor nerve, which controls the movement of the eyes. It leads to an inability to move the eye, double vision, a fixed and non-reactive pupil and eyelid drooping (ptosis, seen here, right eye)

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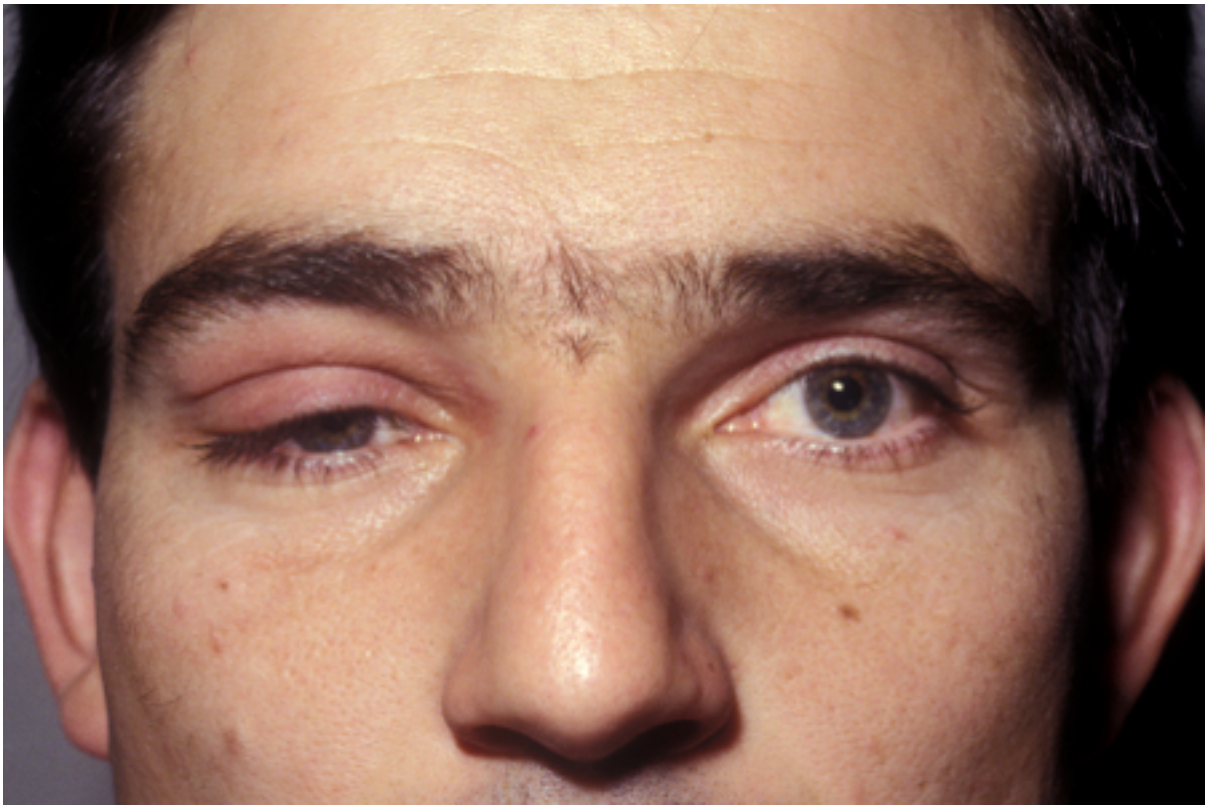

Figure 12: Male patient suffering from post-traumatic acquired ptosis, likely caused by an injury to the eyelid

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Figure 1 – BMJ Best Practice Numeral Style

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numerals < 1: 0.25

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