Aesth. Plast. Surg. 27:193–204, 2003
DOI: 10.1007/s00266-003-0127-5

Aesthetic Plastic Surgery

Ptosis: Causes, Presentation, and Management

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Abstract. Drooping of the upper eyelid (upper eyelid ptosis) may be minimal (1–2 mm), moderate (3–4 mm), or severe (>4 mm), covering the pupil entirely. Ptosis can affect one or both eyes. Ptosis can be present at birth (congenital) or develop later in life (acquired). Ptosis may be due to a myogenic, neurogenic, aponeurotic, mechanical or traumatic cause. Usually, ptosis occurs isolated, but may be associated with various other conditions, like immunological, degenerative, or hereditary disorders, tumors, or infections. Besides drooping, patients with ptosis complain about tired appearance, blurred vision, and increased tearing. Patients with significant ptosis may need to tilt their head back into a chin-up position, lift their eyelid with a finger, or raise their eyebrows. Continuous activation of the forehead and scalp muscles may additionally cause tension headache and eyestrain. If congenital ptosis is not corrected, amblyopia, leading to permanently poor vision, may develop. Patients with ptosis should be investigated clinically by an ophthalmologist and neurologist, for blood tests, X-rays, and CT/MRI scans of the brain, orbita, and thorax. Treatment of ptosis depends on age, etiology, whether one or both eyelids are involved, the severity of ptosis, the levator function, and presence of additional ophthalmologic or neurologic abnormalities. Generally, treatment of ptosis comprises a watch-and-wait policy, prosthesis, medication, or surgery. For minimal ptosis, Müller's muscle conjunctival resection or the Fasanella Servat procedure are proposed. For moderate ptosis with a levator function of 5–10 mm, shortening of the levator palpebrae or levator muscle advancement are proposed. For severe ptosis with a levator function <5 mm, a brow/frontalis suspension is indicated. Risks of ptosis surgery infrequently include infection, bleeding, over- or undercorrection, and reduced vision. Immediately after surgery, there may be temporary difficulties in completely closing the eye. Although improvement of the lid height is usually achieved, the eyelids may not appear perfectly symmetrical. In rare cases, full eyelid movement does not return. In some cases, more than one operation is required.

Key words: Ophthalmology—Neuromuscular—Eyelid—Vision—Surgery

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Introduction

Ptosis is the drooping or sagging of a body part [4]. Ptosis of the upper eyelid (upper lid ptosis, blepharoptosis, drooping upper eyelid, droopy-lid-syndrome) is defined as abnormally low-lying upper eyelid margin in primary gaze, resulting in narrowing of the palpebral opening and fissure and covering part of the eye. The normal adult upper lid margin is 0.5–2 mm below the superior corneal limbus and is highest just nasal to the pupil (upper lid height) [7,17]. The deficient function may be due to weakness of the muscle responsible for lid retraction (either the levator palpebrae superioris muscle and its aponeurosis or Müller's muscle), or damage to the nerves that control these muscles. Laxity of the upper eyelid skin leads at best to mechanical ptosis. Ptosis can either be present at birth (congenital ptosis) or develop later in life (acquired ptosis). Ptosis may affect one or both eyes. Sometimes ptosis is an isolated problem that changes the patient's appearance without otherwise affecting health. In other cases, ptosis can be a warning that a more severe condition affecting muscle, nerve, brain, or the eye socket is under way. In particular, ptosis that develops within hours or days may indicate a serious medical problem.

Anatomy

Levator Palpebrae Superioris and its Aponeurosis

The levator palpebrae superioris is a striated muscle that is innervated by the superior division of the oculomotor nerve (cranial nerve III) and controls lid-opening. This muscle originates just above the annulus of Zinn along the lesser wing of the sphenoid and is about 40 mm in length. It continues anteriorly and, at the equator of the globe, a transition from muscle to an aponeurosis occurs, approximately 15–17 mm from the superior tarsal border. At this site, the superior transverse ligament of Whitnall inserts. Whitnall's band, being thick or thin, is a quasi-suspensory ligament of the levator and changes the muscle, or aponeurosis pull to an inferior direction. Whitnall's band extends from the trochlea and its fibers blend into the lacrimal gland capsule and periorbita of the frontal bone. The levator's aponeurosis is 14–20 mm in length and inserts into the anterior aspect of the tarsal plate [17]. Altogether, the levator/aponeurosis complex is 54–60 mm in length. At about the level of the upper border of the tarsus, it also sends attachments to the skin forming the upper eyelid crease (fold of skin). The levator palpebrae is the major elevator of the upper lid. It is found just under the pre-aponeurotic fat [4,17,30].

Müller's Muscle

Directly under the levator aponeurosis and firmly attached to it is Müller's muscle, a 20 × 20-mm, vascularized smooth muscle that originates on the undersurface of the levator and inserts onto the superior tarsal border. Müller's muscle inserts with a 0.5–1-mm-long tendon into the superior tarsal plate. It is sympathetically innervated and regulates the palpebral fissure width. Müller's muscle elevates the lid for approximately 2 mm [7]. Behind Müller's muscle is the conjunctiva with a suspensory check ligament on the fornix [30].

Orbicularis Oculi

The orbicularis oculi muscle is a sheet of thick, richly vascularized muscle fibers. It is divided into an orbital and palpebral component, the latter of which may be separated into a preseptal and pretarsal part. The orbicularis oculi muscle is an antagonist of the eyelid retraction and lowers the upper eyelid [4,30].

Orbital and Palpebral Skin

The eyelid skin is the thinnest of the body. It is affixed firmly to the pretarsal orbicularis of the upper eyelid and more loosely to the preseptal orbicularis.

Orbital Septum

The orbital septum originates from the arcus marginalis of the frontal bone and blends with the levator aponeurosis 5–20 mm above the superior tarsal border [17]. It acts as a barrier in the upper eyelid. The orbital septum varies anatomically and can be thick or thin. A lid may appear extremely full if it has a low-riding orbital septum with fat advancing forward into the lid, or it may appear deep if the orbital septum blending with the levator aponeurosis at a higher level on the lid, thereby keeping the fat from coming forward [4].

Surgically, the orbital septum is an important structure. In blepharochalasis, where fat may advance forward due to attenuation or a dehiscence of the septum with the levator aponeurosis, it must be opened to reach the pre-aponeurotic fat that covers the levator aponeurosis. Suturing the orbital septum may result in lid lag and lagophthalmos [10,30].

Tarsal Plate and Upper Eyelid Crease

The tarsal plate in the upper lid is 25-mm long and 10-mm high. The levator aponeurosis inserts onto the tarsal plate and extends into the orbicularis and subcutaneous tissue, thereby creating the upper eyelid crease. The orbital septum blends with the levator aponeurosis above the superior tarsal border, providing varying degrees of fullness in the upper lid. In general, the upper eyelid crease is 7–12 mm from the lashes [10].

Eyelid Fat

Usually, the upper eyelid has three pockets, including a nasal and a middle fat pocket. The third, a lateral pocket, is occupied by the lacrimal gland. However, in many cases, a small amount of fat may be adjacent to the lacrimal gland. This pre-aponeurotic fat serves as a glide for the levator muscle and aponeurosis, lying underneath the pre-aponeurotic fat. In the aging face, the loss of support of the suborbital fat pad leads to a trough deformity (prominent nasal jugal fold) and a prominent sulcus in the transition from the lower eyelid in the malar complex.

Classification

Ptosis may be classified according to various criteria, such as age at onset, etiology, severity, and levator function. Based on age at onset, ptosis may be congenital or acquired [2]. Based on etiology, ptosis may have a myogenic, neurogenic, aponeurotic (disinsertion or dehiscence of the levator aponeurosis from its normal position on the anterior surface of the tarsus), mechanical, or traumatic cause [1,2]. According to

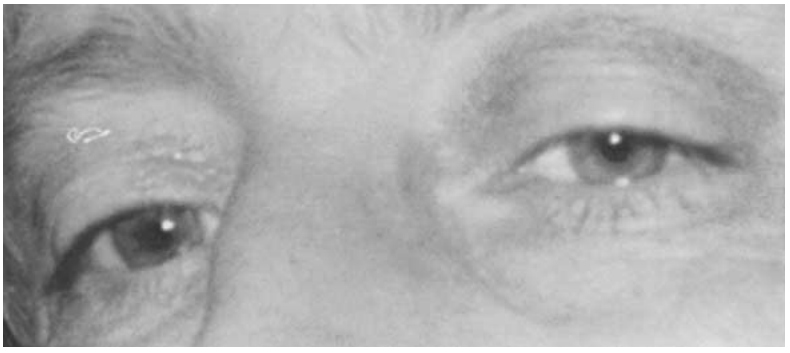

Fig. 1. Mild congenital ptosis of the left eye in a 35-year-old patient.

Table 1. Disorders associated with ptosis, classified according to etiology

Myogenic	Aromatic L-amino acid decarboxylase deficiency, congenital myopathies (central core disease), congenital fibrosis of the extraocular muscles, facio-scapulo-humeral muscular dystrophy, glycogenosis, mitochondriopathy, myositis, myotonic dystrophy, myasthenia gravis, myasthenic syndrome, oculo-pharyngeal muscular dystrophy, orbital rhabdomyosarcoma
Neurogenic	Apraxia of lid opening, blepharospasm (benign, essential), botulinism, botulinum toxin therapy, cerebellar vermis hypoplasia, cerebral tumor, cerebral, ocular, dental, auricular, skeletal anomalies (CODAS) syndrome, crebral vasculitis and venovascular hypertension, chronic rhinocerebral mucormycosis, cluster headache, cortical dysplasia and maldevelopment of the basal ganglia, facial nerve palsy, hemifacial spasm, Horner's syndrome, Marcus Gunn jaw-winking syndrome, multiple sclerosis, mycotic meningitis, migraine ophthalmique, optic glioma, orbital dermoidal cyst, oxiliplatin neuropathy, paraneoplastic syndrome, Raeder Syndrome (acquired Horner's syndrome with ipsilateral headache), Recklinghausen's neurofibromatosis, rheumatoid pachymeningitis, Riley-Day syndrome, Schwartz-Jampel syndrome, sleep apnea syndrome, stroke (mesencephalic, hemispheric), SUNCT syndrome, syringomyelia, third-nerve palsy (carotid aneurysm, cavernous sinus thrombosis, congenital, degenerative CNS diseases, heavy metal intoxication, increased intracerebral pressure, trauma, superior orbital fissure syndrome, tumors (dermoidal cyst, fibrous tumor, neurinoma, non-Hodgkin's orbital lymphoma)), vascular lesions, Wernicke's encephalopathy
Mechanical	Scarring, excessive weight—dermatochalasis, eyelid mass (lid tumors: neurinoma, neurofibroma), orbital mass
Traumatic	Birth trauma, forceps delivery, corneal abrasion, corneal foreign body, eyelid laceration, hard contact lens embedding, orbital fracture (apex or floor), orbital hemorrhage, postcataract ptosis, transorbital penetrating brain injury, trauma to the levator aponeurosis
Miscellaneous	Anophthalmos, atopic dermatitis, blepharochalasis, blepharophimosis-ptosis-epicanthus inversus syndrome (BPES) due to FOXL2 gene mutations, capillary hemangioma, carotid aneurysm, carotid cavernous fistula, chalazion, chromosome 14q terminal deletion syndrome, combined valproate and hydantoin embryopathy, craniofacial syndromes, de-novo duplication dup (Xq22.1–q25), distichiasis with FOXC2 truncating mutations, double partial monosomy (10p13–10pter and Xp11.4–Xpter), Down syndrome, Duane syndrome, exophthalmos, eyelid edema, fetal alcohol syndrome, fibrosis syndrome (CFEOM1 locus on chromosome 12), floppy eyelid syndrome, giant papillary conjunctivitis, glaucoma, iris coloboma, hypertelorism, mental retardation due to deletion on chromosome 2, Joubert's syndrome, King-Denborough syndrome, lacrimal gland hemangiopericytoma, mandibulofacial dysostosis, mucolipidosis type IV, mycotic aneurysm of the internal carotid artery, oculo-facio-cardiac-dental syndrome, orbital artery obstruction, orbital or preseptal cellulitis, orbital fat prolapse, orbital fibromatosis, orbital Langerhans cell granulomatosis, orbital osteoclastoma, orbital phlegmona, Parry-Romberg syndrome, partial trisomy 1q32–qter 'pure,' poorly fitting ocular prosthesis, Rubinstein-Taybi syndrome, Smith-Magenis syndrome, Smith-Lemli-Opitz syndrome, socket contraction, Sturge-Weber syndrome, supernumery chromosome

severity, ptosis may be minimal or mild (1–2 mm), moderate (3–4 mm), or severe (>4 mm) [30]. According to levator function, ptosis may be poor (0–4 mm), moderate (5–10 mm), or good (>10 mm) [17]. Generally, mild ptosis is associated with good levator function (>8 mm), moderate ptosis with fair levator function (5–7 mm), and severe ptosis with poor levator function (1–4 mm) [10,17,23].

Congenital

Congenital ptosis is present at birth or manifests in the first year of life. In three quarters of the cases, congenital ptosis affects only one eye (Fig. 1) [21]. Concerning etiology, congenital ptosis may be idiopathic, myogenic (primary myopathies, congenital myasthenia, muscular dysgenesis), aponeurotic, neu-

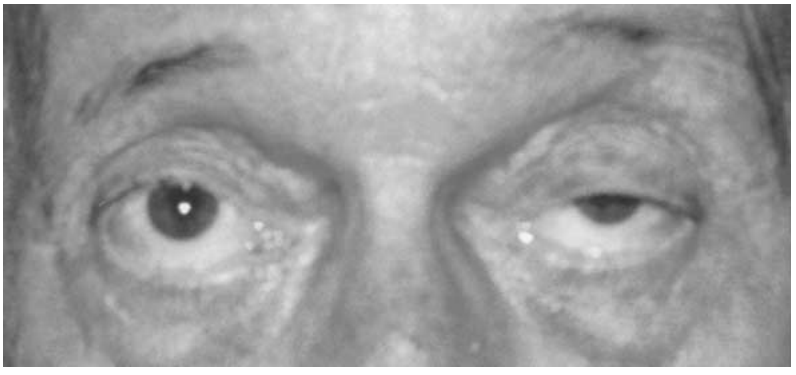

Fig. 2. Left-sided ptosis in a patient with myasthenia gravis before initiation of therapy with cholinergic drugs and immunosuppressants.

Fig. 3. Bilateral ptosis with left-sided predominance in a patient with chronic progressive external ophthalmoplegia prior to surgery. The levator function was less than 5 mm and there was severe ophthalmoparesis in all directions.

rogenic (congenital third cranial nerve palsy, congenital Horner's syndrome (mild ptosis, associated with ipsilateral miosis, areola hypopigmentation, and if the lesion is preganglionic, anhidrosis and vasodilation of the head), Marcus Gunn jaw-winking syndrome (side movements of the mouth during sucking, swallowing, or chewing will cause elevation of the lid on the contralateral side, secondary to innervation of the lid muscle by aberrant fibers of the fifth cranial nerve joining the third cranial nerve; when the masticatory muscles are at rest, the upper eyelid will show ptosis), due to mechanical reasons (periorbital tumor, neuroma, neurofibroma, cicatricial skin changes), or due to trauma (birth trauma, blow trauma, trauma to the aponeurosis) (Table 1) [23]. Contrary to acquired ptosis, congenital ptosis is not the same at up- and down-gaze and the lid crease is often absent [17].

The majority of congenital ptosis results from a localized myogenic dysgenesis (improper or faulty development of the levator muscle) or innervation abnormalities [17]. The eyelid muscles are either scarred or do not work. Instead of normal muscle fibers, fibrous and adipose tissues are present within the muscle belly, diminishing the ability of the levator to contract and relax. The infant may throw the head back to enhance vision and use the frontalis muscle to elevate the lid. Congenital ptosis is bilateral if due to infantile myasthenia gravis or antibodies transferred to the neonate by a mother with myasthenia gravis. In Horner's syndrome, sagging of the eyelid is milder than in oculomotor nerve affection [29]. Congenital ptosis may be also seen with Sturge–Weber syndrome, M. Recklinghausen, or fetal alcohol syndrome (Table 1) [20]. Congenital ptosis must be addressed and treated properly to insure normal maturation of the visual system and to avoid amblyopia (lazy eye) secondary to deprivation or uncorrected astigmatism, strabismus, or decreased visual field.

Acquired

Acquired ptosis can have a myogenic, neurogenic (peripheral or central lesion of the sympathetic nerves, peripheral or central lesion of the oculomotor nerve), aponeurotic, mechanical, or traumatic cause. True acquired ptosis has to be delineated from pseudoptosis.

Myogenic ptosis. Myogenic ptosis is a reflection of a primary or secondary myopathy. Weak muscles cannot pull the eyelid up into position. The most frequent disorders causing myogenic ptosis are myasthenia gravis (Fig. 2) [12], myotonic dystrophy (MD) [26], facio-scapulo-humeral muscular dystrophy, oculopharyngeal-muscular dystrophy (OPMD) [3,28], congenital myopathies [11,13], and mitochondriopathy (Fig. 3) [24] (Table 1). In a retrospective study by Wong et al., the most frequent myogenic

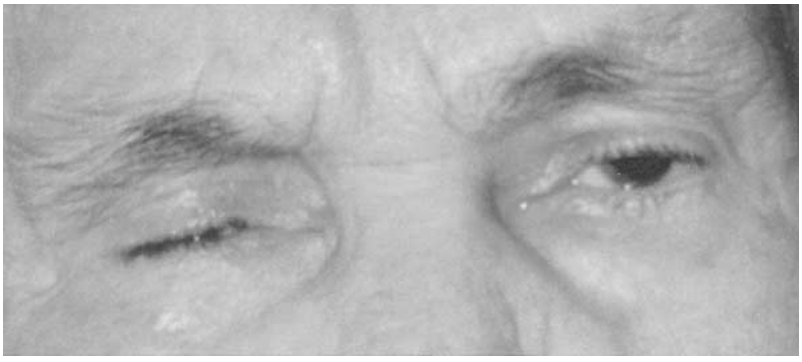

Fig. 4. Initially complete (later incomplete) right-sided ptosis in a patient with acute stroke and seizures.

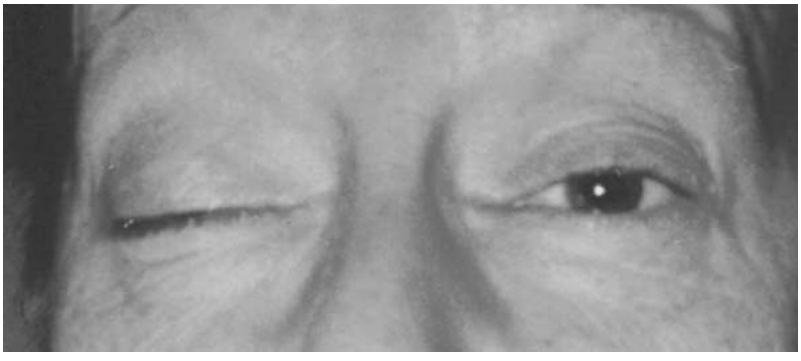

Fig. 5. Severe right-sided ptosis due to a carotid aneurysm in the right cavernous sinus.

causes of ptosis were chronic progressive external ophthalmoplegia (43%), OPMD (18%), and MD (18%) [33]. People with myasthenia gravis will often have crossed eyes in addition to ptosis due to imbalance of the muscles that control ocular motility. A localized toxic myopathy has been thought to be responsible for ptosis in association with ingestion of lead or exposure to other heavy metals. Myogenic ptosis is generally progressive and has a high incidence of recurrence, despite repeated surgery (Table 1) [16,17].

Neurogenic ptosis. Neurogenic ptosis is rare and proper diagnosis is important in order to avoid unnecessary surgery. When a neurological disorder is present, symptoms typically include visual complaints independent of the ptosis. Neurogenic ptosis is due to malfunction or damage of the oculomotor or sympathetic nerve(s) or due to CNS abnormalities (Fig. 4). Frequent conditions that affect the oculomotor nerve are diabetes, tumors, aneurysms (Fig. 5), vasculopathy, multiple sclerosis, intoxication with heavy metals, and injuries (Table 1). If sympathetic nerves are damaged, Horner's syndrome develops. Horner's syndrome may be hereditary or acquired. Acquired Horner's syndrome can be secondary to trauma, neoplasm, stroke, or vascular disease of the sympathetic pathway. Because sympathetic nerves travel at the top of the chest cavity before going up the neck to the head, abnormalities in the upper areas of the lungs should be considered. Because eye muscles are controlled by nerves that originate in the brain, conditions that injure the cerebrum can also cause ptosis. These conditions include strokes, tumors, multiple sclerosis, aneurysms, and diabetes (Table 1) [18,31].

Aponeurotic ptosis. Aponeurotic ptosis, also termed "senile" or age-related ptosis, is the most common cause of acquired ptosis [7,17]. In aponeurotic ptosis due to advanced age, the long-term effects of gravity and aging cause involuntary changes like stretching of the levator muscle and its aponeurosis. The muscle becomes thinned, resulting in a loss of muscle tone and the inability to hold the upper lid in the proper position above the eye. Additionally, ptosis may be due to disinsertion or a dehiscence of the aponeurosis [7]. Chronic inflammation or intraocular surgery can also incite weakness of the levator aponeurosis from the anterior surface of the tarsal plate. Patients who wear hard contact lenses or have a history of severe eye infection, cataract surgery, or blunt trauma to the eye may have an increased risk for age-related ptosis. Although both eyes are usually affected, drooping may be worse in one eye [22].

Mechanical ptosis. Mechanical ptosis occurs when the eyelid is too heavy for the muscles to lift. Excess skin (blepharochalasis), fat, or lid tumors (neurofibroma, hemangioma) can weigh down the eyelid (brow ptosis). Over time, many people develop excess eyelid skin. Because eyelid skin is the thinnest of the body, it is more likely to stretch. In the upper eyelid, the stretched skin may limit the field of vision and

produce a feeling of heaviness and tired appearance. In this condition, common in men, the entire brow area droops down. If folds of skin then block vision, surgical correction is proposed. Mechanical ptosis can also stem from scarring due to cicatrisation secondary to inflammation, surgery, Stevens-Johnson syndrome, or ocular pemphigoid [7,22].

Traumatic ptosis. According to the severity of trauma, traumatic ptosis may be mild, moderate, or severe. Mild degrees of trauma, associated with edema or hemorrhage, may produce a levator disinsertion that can be readily repaired. Moderate trauma may result in lacerations of the lid, which sever the levator tendon and lead to scarring and secondary mechanical ptosis [17]. Severe trauma involves damage to the nerve supply of the levator muscle. Since the levator and the superior rectus muscle are commonly innervated, such injuries may affect elevation of the eye, including Bell's phenomenon. In case of blow trauma to the eye, like in an automobile accident, structures around and in the eye may be damaged, but nerve and muscle may work properly. Trauma may cause lid laceration with transsection of the upper lid elevators or disruption of the neural input. However, if the muscle is no longer fully attached to the solid structures inside the eyelid, the muscle contracts but the eyelid does not come up. Posttraumatic ptosis may also be associated with synkinesia (when patient moves the jaw, the upper lid contracts) [7].

Pseudoptosis

Causes of pseudoptosis are dermatochalasis, lacking support behind the upper lid, acquired hypotropia, superior sulcus deformity, contralateral vertical eyelid retraction, anophthalmos, enophthalmos, microphthalmos, phthisis bulbi, blepharospasm, hemifacial spasm, and hypermetropia [2,4,7,21].

Occurrence in the Population

Sufficient data concerning the incidence of ptosis either internationally or for a particular country are not available. Ptosis may present within a broad range of a human's lifetime. There is equal frequency among different races and equal frequency between the sexes. Risk factors for developing ptosis include aging, diabetes, myasthenia gravis, brain tumor, or cancer, which can affect nerve or muscle response.

Investigations

History

Symptoms patients with ptosis complain about are drooping of the eyelid(s), a "bedroom-eye" (sleepy or tired) appearance, blurred vision, or increased tearing. If ptosis is severe, there may be constriction of the upper visual field [7]. Patients with significant ptosis may need to tilt their head back into a chin-up position, lift their eyelid with a finger, or raise their eyebrows in an effort to see from under their drooping eyelid(s). The decreased visual field can affect one's ability to perform activities of daily living. Driving, reading, and navigating a flight of steps can be particularly difficult. In addition, drooping of the eyelid may result in an undesired, uneven facial appearance with which patients are dissatisfied. Raising the entire brow with the muscles of the forehead and scalp may additionally cause frontal tension headache, eyestrain, and an odd "surprised" appearance. An abnormal lid position can have negative psychosocial impacts, particularly in young children and teens. Ostracism can lead to poor academic performance, loss of self-esteem, and alienation [5].

When taking the patient's history, it is important to ask the patient for the family history, previous stroke, diabetes, circulatory problems, time of onset, if there are alleviating or aggravating factors, reaction to jaw-winking, contact lens wear, if there has been any trauma or surgery to head, eye, or neck, if there has been diplopia, and if there are any symptoms associated with myasthenia gravis or other systemic disease, like fatigue, weakness, wasting, muscle cramps, muscle stiffness, fasciculations, dysarthria, or dysphagia.

Clinical Neurologic Examination

On clinical neurologic examination there may be drooping of the eyelid of one or both eyes, leading to reduced palpebral fissure height, weak or absent crease position, reduced levator function, and eyelid lag or eyelid droop on downgaze. Ptosis may be mild (1–2 mm), moderate (3–4 mm), or severe (>4 mm) [6]. It is important to conduct a complete neurologic examination because there may be additional signs if ptosis is due to a systemic neurological disease. Gradual onset of extraocular muscle weakness, particularly on upgaze, in addition to ptosis and slowed saccadic velocity may be an early sign of chronic progressive external ophthalmoplegia. These patients should also be examined for pigmentary retinopathy and frequently associated central nervous system (CNS), endocrine, cardiac, or otologic involvement.

Ophthalmologic Examination

Ophthalmologic examination should include simple observation (abnormal head posture, strabismus, proptosis, synkinesia, lagophthalmos, lower lid laxity), palpitation of lid and orbit, examination of pupils for size, symmetry, and reactivity, versions and duc-

tions, slit lamp examination, ocular motility, testing for Simpson's and Cogan's sign, cover test, status of ocular protective mechanisms (Bell's phenomenon, orbicularis muscle function, corneal sensation, tear film adequacy—tear break-up time, Schirmer's test), notation regarding amount of dermatochalasis, orbital fat prolapse, lid contour, lashes, skin, status of the tarsus and superior fornix, and measurement of refraction (particularly refractive errors like astigmatism may result in amblyopia without visual axis obstruction [7]), visual acuity, visual field, and the following variables: (a) Upper lid height: distance between upper limbus and lid margin (normal: 0.5–2 mm). The cornea is about 11 mm in height [17]. (b) Palpebral fissure width: distance between upper and lower lid in vertical alignment with the center of the pupil. (c) Margin-to-reflex distance: distance between upper eyelid margin and the mid-pupillary reflex in primary position (normal: >2.5 mm) and on downgaze. Acquired ptosis may increase on downgaze; if there is congenital ptosis the upper lid may be relatively "held up." If the lid margin is 1.5 mm above the corneal light reflex, there is 2–3 mm of ptosis [17,22]. (d) Levator function: expressed as distance between excursion of upper lid margin from full downgaze to full upgaze without brow movement. Levator function may be excellent (>10 mm), good (8–10 mm), fair (5–7 mm), or poor (1–4 mm) [17]. As a rule of thumb, mild ptosis is usually associated with good levator function, moderate ptosis with fair levator function, and severe ptosis with poor levator function [17,22]. (e) Upper-lid margin to lid crease distance (normal 10–11 mm in women and 8–9 mm in men) [4]. If the lid crease is higher than normal and if there is a deeper upper lid sulcus, these may be signs of an apparent aponeurotic disinsertion. When the patient is asked to look up, both the sulcus and the lid crease move superiorily. Presence of a crease suggests that there is some levator function, even if the lid moves poorly. An absent lid crease is usually accompanied by poor levator function [17,22]. (f) Iliff's sign: positive if the lid everted by the examiner does not flip back to its normal position when looking up.

Instrumental Investigations

Blood chemical studies should look for serum glucose, creatine-kinase values, antibodies against acetyl-cholin receptor antibodies, thyroid function, and signs of inflammation or malignancy. CSF investigations can aid in the diagnosis of mitochondriopathy, multiple sclerosis, and CNS tumor. Work-up of myopathy additionally requires electromyography and muscle biopsy with morphological, biochemical, and genetic investigations. If neurogenic ptosis is suspected, if the patient has other neurological findings like abnormal pupil, strabismus consistent with third cranial nerve palsy, if there is acute ptosis, or if there is any suspicion of an orbital process or sulcus filling, an MRI or CT scan of the head and orbita should be carried out to rule out mass [17]. Immediate MRI scan, however, is not required in the diagnostic work-up of pupil-sparing third nerve palsy at age >50 years. MRI of the brain with contrast medium is the imaging modality of choice for suspected multiple sclerosis. For the diagnosis of Horner's syndrome MRI/CT scans of the brain, spine, and thorax are helpful. Sweat tests may demonstrate the extension of the sympathetic lesion. If there is Horner's syndrome with impaired homolateral sweat secretion, the lesion is between the radix and the ganglion. Following administration of hydroxy-amphetamine, pupillary dilation occurs in healthy subjects and those with central Horner's syndrome, but is absent in postganglionic Horner's syndrome. Visually evoked potentials are helpful to assess function of the visual pathways in mitochondriopathy, multiple sclerosis, and other CNS lesions. The described instrumental investigations are helpful in almost all cases of acquired ptosis, but hardly in congenital ptosis.

Treatment

Treatment and management of ptosis require an experienced neurologist, ophthalmologist, or plastic surgeon. Usually, ptosis can be corrected if the underlying cause is treated. Aponeurotic ptosis is not preventable. If an underlying disease is found, the treatment may be specific to that disease.

Conservative

Mild to moderate congenital ptosis may improve over time without any ocular complication [8]. Some types of neurogenic ptosis also get better by themselves. Ptosis in myotonic dystrophy has been treated by a special make-up "eye-putti" which is glue, fixing the upper eyelid to the supraorbital structures [26]. Glasses can be made with a crutch attachment that can hold up the lid. Some forms of myogenic ptosis, particular in myasthenia gravis and myasthenic syndrome, respond to medication. Patients with ptosis do not profit from any diet. Physical activity can be carried out as tolerated, if there is no limiting systemic neuromuscular disorder.

Surgery

In most cases, ptosis is corrected by surgery (ptosis surgery, blepharoplasty). In nearly all cases of ptosis in adults, surgery is performed by the ophthalmologist or plastic surgeon on an outpatient basis with a local anesthetic and a mild sedative. For children, general anesthesia is necessary. Preoperative examinations should include those recommended for the

Table 2. Therapy of upper eyelid ptosis

Conservative	Surgery
Watch-and-wait policy in neurogenic ptosis	*Müller's muscle procedures* (*ptosis 1–2 mm*, *levator function >10 mm*)
Glasses with a crutch attachment	Müller's muscle conjunctival resection
Eye putty	Fasanella-Servat procedure
Cholinesterase inhibitors, corticosteroids, azathioprin in myasthenia	*Levator muscle procedures* (*levator function >4 mm*)
Diaminopyridine in myasthenic syndrome	Internal or external levator muscle advancement or orbicularis myectomy with levator advancement
	Levator muscle resection (shortening)
	Brow/frontalis suspension procedures (*levator function <5 mm*)
	Autoplastic material (Crawford method): frontalis suspension sling with autogenous fascia lata or brow suspension with stored fascia lata
	Alloplastic material (Fox pentagon): frontalis suspension or brow suspension by the use of catgut, collagen, prolene, silicone, stainless steel, silk, skin, supramid, sclera, tantalum, tarsus, or mersilene
	Other techniques: endoscopic forehead lift for ptosis of the brow,Whitnall's sling, anchoring correction of eyebrow ptosis, semilunar segment of skin flap

neurologic and ophthalmologic examination. Additionally, a preoperative photograph should be made [17].

Techniques. Though several procedures have been established, ptosis surgery remains a highly individualized art form. The type of surgical procedure will be determined by the etiology of ptosis and the severity of ptosis or levator function, respectively. The two main ways to achieve elevation of the upper lid are to shorten the levator palpebrae superioris or Müller's muscle, or to carry out a brow/frontalis suspension procedure. By shortening the levator or its aponeurosis, the pulling force of the muscle is strengthened. The muscles can also be reattached to the eyelid structures. If there is less than 5 mm levator function, shortening the muscle will not achieve the desired effect. In this case the lid is suspended with tissue attached to the eyebrow muscles so that raising the brow lifts the lid [4]. For determining the optimal lid margin level during surgery the following guidelines are recommended: in bilateral surgery lid margins are placed at or 1 mm below the superior limbus. In unilateral surgery the margin of the ptotic lid is placed 1–2 mm above that of the contralateral lid.

Müller's muscle procedures (ptosis 1–2 mm, levator function >10 mm). (1) Müller's muscle conjunctival resection: Müller's muscle conjunctival resection (resectioning of Müller's muscle and of the conjunctiva) via a posterior approach is a frequently applied procedure in blepharoplasty [10]. This is because of its ease, precision, predictability, and ability to grade the correction (Table 2).

(2) Fasanella-Servat procedure: The Fasanella-Servat procedure involves the excision of conjunctiva, Müller's muscle toward the fornix, and some accessory lacrimal glands (tarsomüllerectomy). The success rate of the Fasanella-Servat procedure is 70% [17]. Resection is carried out according to the following algorithm: 1 mm ptosis: 4 mm resection, 1.5 mm ptosis: 6 mm resection, and 3 mm ptosis: 11–12 mm resection. In patients with dry-eye syndrome, symptoms may worsen after surgery, because they have decreased tarsal stability and fewer accessory lacrimal glands postoperatively, thus less basal tear secretion and the tear film will evaporate faster after elevation of the lids. The procedure should also be avoided in patients with significant corneal disease or filtering blebs. Some surgeons do not use the procedure at all, because promotion of a poor lid crease and partial removal of the tarsal plate may cause secondary problems in some patients.

Levator muscle procedures (levator function 5–10 mm). (1) Levator muscle advancement (levator aponeurotic repair): With this technique the levator and its aponeurosis are reattached to the eyelid structures [27]. A maximal levator aponeurosis advancement is also possible by performing Whitnall's sling procedure. In particular, patients with unilateral congenital ptosis and levator excursions of 5–6 mm often profit from this technique (Table 2) [10].

(2) Levator muscle resection (shortening): Levator resection is the procedure of choice for patients with congenital ptosis when reasonable levator function is present (Table 3). The amount of resection can be small (10–13 mm), medium (14–20 mm), or large (21–26 mm), and can be tailored to be smaller or larger depending on the levator function (i.e., a patient with 7 mm of levator function and 3 mm of ptosis requires a smaller resection than a patient with 3 mm of ptosis and 5 mm of levator function) [17]. Levator resection

Table 3. Amount of levator muscle resection in relation to amount of ptosis and levator function [17,21]

Amount of ptosis	Levator function	Levator resection
Mild (1–2 mm)	Excellent (>10 mm)	Levator resection or Müller's muscle procedure
Mild (1–2 mm)	Good (8–10 mm)	10–13 mm
Moderate (3–4 mm)	Fair (5–7 mm)	14–20 mm
Severe (>4 mm)	Poor (1–4 mm)	21–26 mm
Severe (>4 mm)	None	Sling

may be carried out either via an anterior or posterior approach (Table 2) [15,17].

Brow/frontalis suspension procedures (levator function <5 mm). Various brow/frontalis suspension techniques either with autoplastic (autogenous or stored sling fascia lata), or alloplastic material (mersilene mash, silicone rod, silicone slings [17,19,25,32]) are available (Table 2). Though many other autoplastic and alloplastic materials have been proposed, it is generally agreed that fascia lata gives the best results [17]. Autogenous fascia lata is the material of choice for any brow suspension and gives superior results to alloplastic materials. There are few donor site complications and the material is well tolerated. Material that is foreign to the body is likely to become infected or to be extruded at a later date. Alloplastic material may be used only if autogenous fascia is not available or when fascia lata is not necessary, like in temporary lid elevation. If fascia lata is used, the Crawford technique is recommended.

Silicone slings are recommended in all patients with third nerve palsy or myogenic ptosis and a levator function of <8 mm and in all patients in whom harvesting fascia lata is impracticable [10,33]. The treatment of choice for blepharophimosis/ptosis syndrome at an early age are Moustarde's or Whitnall's double Z-plasty (Table 2). In these patients, canthoplasty is also recommended [17]. If alloplastic material is used, it is commonly inserted as a Fox pentagon. To achieve symmetry, it may be necessary to carry out a bilateral brow suspension even if there is only unilateral ptosis (Table 2).

Recently, techniques for carrying out adjustable ptosis surgery have been described. Such techniques allow for a further adjustment of lid height when the effects of local anesthesia have worn off. However, it becomes progressively more difficult to adjust the sutures as more time elapses and adjustment is probably only practical within 24 hours after surgery. It is easier to advance the levator muscle than to achieve recession with the adjustable sutures: therefore, if they are used, it is desirable not to set the level in an overcorrected position at the end of surgery. Adjustable sutures may be used in either anterior approach surgery or where a posterior approach has been chosen. Any skin sutures are normally removed after seven days, depending on the type of surgery [17].

Indication According to Severity

Minimal ptosis (1–2 mm). For minimal ptosis there are three viable options: Müller's muscle conjunctival resection, Fasanella-Servat procedure, or levator aponeurotic repair. Since the decision of whether Müller's muscle procedure or Fasanella-Servat procedure is carried out depends on the result of the phenylephrine test, this test is obligatory in all patients with minimal ptosis prior to surgery. The phenylephrine test is carried out by instilling two drops of 2.5% phenylephrine (sympathomimetic agent) in the ptotic eye. Ten minutes later, both lid heights are recorded [22]. The test is considered positive if the margin-to-reflex distance increases >1.5 mm [10]. Such increase indicates that Müller's muscle is viable and that Müller's muscle conjunctival resection procedure is indicated [10] according to the following algorithm: 1 mm ptosis: 4 mm resection, 1.5 mm ptosis: 6 mm resection, 2 mm ptosis: 10 mm resection, >3 mm ptosis: 11–12 mm resection. A negative phenylephrine test precludes Müller's muscle conjunctival resection procedure [10]. If the response of Müller's muscle to phenylephrine or apraclonidine is poor, the Fasanella-Servat procedure or a levator muscle resection or levator muscle advancement is needed. Levator aponeurotic repair has the advantage that the eyelid height can be set on the operating table. If the margin-to-reflex distance decreases appreciably to phenylephrine in the contralateral eye, this usually indicates that contralateral ptosis is present [10].

Moderate ptosis (3–4 mm). For moderate ptosis the treatment of choice is levator aponeurotic repair. It is also recommended for Marcus-Gunn syndrome (see below). Patients with unilateral congenital ptosis and levator excursions of only 4–5 mm may profit from Whitnall's slings or maximal levator aponeurotic advancement.

Severe ptosis (>4 mm). Severe ptosis requires some type of frontalis suspension [10]. Severe congenital ptosis with little levator function is best treated with autogenous fascia lata grafts. Nonautogenous material is available, the long-term results however are poorer than with autogenous material [10]. Severe acquired ptosis is best treated by frontalis suspensions using a silicone or silastic rod because of

its adjustability and the possibility of removal if the cornea becomes compromised [10].

Indication According to Etiology

Congenital ptosis. Though not always indicated, the most common treatment of congenital ptosis is surgical, when reasonable levator function is preserved [17]. The goal is to tighten the muscles so that the lid is elevated to match the lid on the other side, with a minimum of scars and side effects. In children, the timing of corrective surgery for congenital ptosis varies, depending on the severity of the ptosis and the strength of the levator muscle. If there is no sign of developing amblyopia, the operation can be delayed until the child is 3–5 years old. At this age, the structures of the eyelid are better developed and it is also possible to harvest autogenous fascia lata if necessary. If surgery is postponed, babies and toddlers should be monitored monthly to look for signs of amblyopia, worsening of ptosis, and development of abnormal head posture. However, when ptosis interferes with the child's vision, surgery is recommended at an early age to allow proper visual development. Even within the first year of life a monofilament nylon suture can be a satisfactory, temporary brow suspension material. Such unilateral surgery may be repeated as necessary before the child reaches a suitable age for bilateral brow suspension with autogenous fascia lata. After age 7–10 years, amblyopia may not be reversable. The later amblyopia is treated, the harder it is to achieve a good final visual acuity. Therefore, depending on the severity of ptosis, appropriate and timely treatment is crucial to preserve the child's vision. If the levator function is completely absent, if congenital ptosis is isolated, or if there is blepharophimosis, a frontalis sling is necessary as a rule (Table 3) [17]. In milder cases also a standard Berke-type levator resection would be appropriate.

In Marcus-Gunn phenomenon, a levator muscle advancement is indicated if ptosis is the major problem and the wink is only minimal [17]. If the wink is the cosmetic blemish, the muscle needs to be denervated or destroyed and a frontalis sling performed [17,33]. Alternatively, suturing the levator aponeurosis to the linea alba at the upper orbital rim or transection of the levator followed by its transposition to the brow have been proposed [17]. In patients who demonstrate synkinesis, either with the muscles of mastication or eye movements as a result of aberrant regeneration following a third nerve palsy, it will be necessary to withdraw the levator muscle to abolish synkinesis [14].

Acquired ptosis. (1) Myogenic: Myogenic ptosis is generally progressive and has a high incidence of recurrence, despite repeated surgery. If there is adequate tear secretion and orbicularis oculi function, a sling procedure should be considered [17]. For patients with advanced chronic progressive external ophthalmoplegia, surgery may be contraindicated [9]. Ptosis props (possibly in combination with a moist chamber) may then be helpful to the patient and preferable to the risk of potentially sight-threatening exposure keratitis [10].

(2) Neurogenic: In neurogenic ptosis, time should be allowed for any possible recovery of function before planning any surgical intervention. If there is no spontaneous recovery after a period of observation, ptosis is best treated with a frontal suspension using a silicone (silastic) rod (Table 2).

(3) Aponeurotic: When correcting involutional ptosis, the levator aponeurosis should be advanced onto the middle third of the tarsus. If both eyelids are drooping, but only one is low enough to require surgery, the unoperated eyelid will appear lower after a successful repair of the first eye. In such cases, the second eye may also require surgery.

(4) Mechanical: The excess skin in the upper eyelids can be removed surgically to improve the field of vision and other symptoms. Removal of the excess skin in the upper eyelids may improve facial appearance, but it does not elevate the lid. Thus, if there is ptosis in addition to dermatochalasis, shortening of the lid retractors is necessary. If any fatty tissue is present, it may be removed at the same time. If mechanical ptosis is due to lid tumors or scars, their removal is indicated.

(5) Traumatic: If ptosis occurs immediately after trauma and is not neurogenic, it is desirable to carry out an immediate repair of aponeurotic or levator defects and then wait for a period of some months for recovery of function before deciding whether any further surgery is necessary. If the lid is disorganized, operating under local anesthesia may help in locating the position of the levator muscle within the lid tissues [17]. Ptosis due to scarring after moderate trauma with lacerations is best managed by levator aponeurotic repair at the time of primary repair of lid injury. If this is not accomplished, the orbit can be explored at a later time and the levator muscle repaired. After severe trauma, at least six months to one year should be allowed to pass prior to performing any surgery, since some degree of regeneration often occurs. In case of amblyopia, surgery must be performed earlier. After this period, a levator resection or sling procedure can be performed, depending on the severity of ptosis and the degree of levator function recovery [17].

Contraindications to Surgery

Definite contraindications to surgery are: loss of blink reflex, loss of corneal sensitivity, weakness of the orbicularis oculi, or keratitis sicca. Care should be taken in patients with thyroid myopathy, chronic progressive external ophthalmoplegia, or dystrophies

in which a poor Bell's phenomenon, decreased random eye movements during sleep, or poor orbicularis function may exist and produce lagophthalmos and corneal exposure [17].

Complications and Postoperative Course

Initially after surgery, the eyelids are often bruised and swollen. This can take two to three weeks to completely clear up. For that reason, it is normal to leave the pressure dressing in place for 48 hours [22]. This is intended to minimize postoperative swelling and bruising. Additionally, it is important to use ice compresses for the first 48 hours. After the first 48 hours, one should switch to warm compresses. The warm compresses dilate blood vessels and help to reduce swelling and clear bruises.

Complications that may occur after surgery are the following: (a) Undercorrection, the most common complication (10–15% of the cases), may result due to inadequate resection, failure to identify the proper structures, excessive scarring, or misplaced sutures. Significant undercorrection can be adjusted in the early postoperative period. (b) Overcorrection in congenital ptosis is rare and may occur if the lid is sutured to Whitnall's ligament or if the orbital septum is excessively shortened. Overcorrection in acquired ptosis may occur if levator dehiscence is treated with levator resection rather than by repairing the dehiscence. Overcorrection results in the inability to close the eye completely. Such a situation creates a dry eye condition (dry eye syndrome and keratopathy), which is difficult to manage [17]. While it is postoperatively desirable to have a symmetrical height for the upper eyelid with the other side, it is imperative that there is adequate corneal protection. Thus, frequently a Frost suture is made, which keeps the eyelid closed and prevents the eye from becoming exposed. At the first postoperative dressing, an assessment should be made as to whether the patient can achieve adequate lid closure. A judgement is then made whether or not to remove the Frost suture. It is usually removed when the eye pads are removed, 48 hours following surgery, but may remain for a few days longer. Even when there is no immediate threat of corneal exposure, the patient should be instructed to use frequent topical lubricants in the immediate postoperative period and a lubricating ointment at night. In addition to overcorrection, keratitis following ptosis repair may be related to lagophthalmos, inadequate blink, decreased tear production, or poor Bell's phenomenon. (c) Transient diplopia or permanent diplopia may appear in cases of residual third nerve palsy. (d) Reactions to anesthetic agents are possible since ptosis surgery is mostly performed under local anesthesia. Poor response to anesthetic agents may also occur. (e) Bleeding may occur intraoperatively or postoperatively. (f) Infections may develop in the early postoperative period. (g) Mild keratitis and corneal abrasion may result from inadequately placed sutures. (h) Adverse reactions to the implanted alloplastic material or suture abscesses are possible. (i) Eyelid crease abnormalities (absent or low crease) may result from incorrect incision planning or failure to adequately create the crease. (j) Distortion of the eyelid margin contour may result from uneven advancement of the aponeurosis. (k) Lid asymmetry and corneal foreign-body sensation are potential late complications.

Prognosis

In most cases the prognosis is good. Surgical repair is usually very successful and corrects congenital and acquired ptosis, restoring appearance and function. The expected outcome mainly depends upon the cause of ptosis. The outcome may be optimized by operating under local anesthesia and asking the patient to open his eyes from time to time in order to judge both contour and height of the lid and then adjust the sutures between the tarsus and the levator muscle/aponeurosis appropriately. Some estimation has also to be made on the effect of the local anesthesia on the levator and orbicularis oculi muscles. Frequently, there may be a greater anesthetic effect on orbicularis oculi and the surgeon will aim to set the lid at a height that may otherwise be considered a slight overcorrection of the ptosis. In addition, postoperative healing may lift the lid to a variable extent. Although improvement of the lid height is usually achieved, the eyelids may not appear perfectly symmetrical. However, it is not advisable to judge whether surgery was successful or not immediately after surgery. In rare cases, full eyelid movement does not return. In some cases, more than one operation is required [1].

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