Disorders of neuro-ophthalmologic significance may affect not only visual sensory, ocular motor, and pupil function but also the function of the eyelids. Indeed, the inability of a patient to fully open or close his or her eyelids may be the first sign of a more extensive process, the early diagnosis and treatment of which may be crucial to the ultimate well-being of the patient. This chapter describes the elements that make up a complete assessment of eyelid function, the anatomy of the structures that provide normal eyelid function, and the various pathologic processes that impair eyelid opening and closure.

EXAMINATION OF EYELID FUNCTION

Detailed observation of eyelid position and movement is an important but often neglected part of the neuro-ophthalmologic examination (Table 24.1). In evaluating the eyelids, one should note the resting position of the upper and lower eyelids, assess the ability of the upper eyelid to open and close, and observe the various settings in which eyelid opening and closing occur, including voluntary, reflex, and spontaneous blinking, and lid movements accompanying eye movements.

Patients with eyelid dysfunction often complain of visual problems. Many of these problems are neurologic in origin. For example, laxity of the eyelids from a facial palsy can cause exposure keratopathy with associated blurring of vision, ocular pain, and tearing. However, the same clinical symptoms can also result from involutional changes in the elastic connective tissue supporting the lids. Ptosis, which also can be neurologic and non-neurologic in origin, can produce visual problems, even when incomplete, if the eyelashes or lid margin cover the pupil. When obtaining a history, it is important to inquire about the onset, duration and progression of the problem, and fluctuations of symptoms at different times of the day, in different seasons, and in different environmental conditions. Systemic diseases often affect eyelid function, but patients may have no reason to associate them with their eye problems. Therefore, a thorough medical history with particular attention to thyroid conditions, diabetes mellitus, systemic hypertension, myopathies, myasthenia gravis, sarcoidosis, and facial paresis should be obtained. In many situations, old photographs are useful in establishing the presence of a pre-existing condition such as ptosis or eyelid retraction which may have only recently become symptomatic or noticed by the patient.

The detailed examination of the eyelids should proceed as outlined in Table 24.1, with the examiner noting any abnormalities or asymmetries. In addition, because abnormalities of eyelid position, function, or both are often associated with other neuro-ophthalmic signs, a complete eyelid evaluation must be accompanied by an assessment of visual sensory function, ocular motility and alignment, pupil function, function of the trigeminal and facial nerves, and the status of the orbit, particularly for signs of orbital disease such as proptosis or enophthalmos.

All lid movements result from the interaction of four simple forces: (a) an active closing force produced by the orbicularis oculi muscle; (b) an active opening force generated by the levator palpebrae superioris muscle (often called the levator muscle or, simply, “the levator”); (c) an active opening force generated by a smooth muscle—Müller’s muscle.

### Table 24.1

<table>
<thead>
<tr>
<th>EXAMINATION OF EYELID FUNCTION</th>
<th>ANATOMY OF THE EYELID SYSTEM</th>
<th>ABNORMALITIES OF EYELID OPENING</th>
</tr>
</thead>
<tbody>
<tr>
<td>Surface Anatomy</td>
<td>Ptosis</td>
<td></td>
</tr>
<tr>
<td>Anatomy of the Muscles of Eyelid Opening and Positioning</td>
<td>Eyelid Retraction</td>
<td></td>
</tr>
<tr>
<td>Anatomy of the Muscles of Eyelid Closure</td>
<td>Insufficiency of Eyelid Closure</td>
<td></td>
</tr>
</tbody>
</table>
Table 24.1
Examination of the Eyelids

1. General observation of contour, shape, and symmetry of eyelids.
2. Abnormal movements: Involuntary twitches, fasiculations, and synkinesis with other facial muscles.
3. The palpebral fissure (opening) is measured in primary position, and if an abnormality is suspected, in upgaze and downgaze, for each eye. If there is a strabismus or apparent retraction of the eyelid, these measurements are made with the contralateral eye occluded.
4. Upper eyelid position can also be documented and evaluated by measuring the distance between the upper eyelid margin and the corneal light reflex (marginal-reflex distance, or MRD).
5. Levator function is assessed by measuring difference in position of the eyelid margin between downgaze to upgaze while preventing contraction of the frontalis muscle by holding down the eyebrow.
6. Eyelid movement during slow pursuit of a target from upgaze to downgaze is observed for lid retraction or lid lag.
7. Fatigability of the levator is assessed by having the patient maintain maximal upgaze on a target for at least 1 minute. A progressive drooping of the eyelid during this test is a sign of ocular myasthenia gravis.
8. Cogan’s lid twitch, which is an upward overshoot of the eyelid on reflexion from downgaze to primary position, is also a sign of myasthenia.
9. Synkinesis between the levator and other muscles (especially the ocular motor muscles, but, also occasionally with muscles innervated by cranial nerves V, VII, IX, and XI) is noted.
10. Abnormal spontaneous contractions of the orbicularis oculi, such as twitches, tics, fasciculation, blepharospasm, and synkinesis with other facial muscles and may also be signs of disease.
11. The strength and function of the orbicularis oculi is evaluated by observing normal blinking for frequency (rate) and completeness, and by testing the strength of closure by attempting to pry open the forcibly closed eyes.

(Also called the superior tarsal muscle); and (d) passive lid-closing forces produced by stretching of ligaments and tendons of the eyelid. For example, normal blinks result from a cessation of the tonically active levator followed by a transient burst of the normally quiescent orbicularis oculi. The active orbicularis oculi force combined with passive lid-closing forces rapidly lower the lid. When the orbicularis oculi activity terminates, the tonic levator activity resumes. This action slowly raises the eyelid until the passive closing forces match the active opening forces generated by the levator. Moving the lid in conjunction with vertical eye movements involves changes only in the activity of the levator. This muscle receives an eye movement input qualitatively identical with that of its developmental progenitor, the superior rectus muscle. As the eye elevates, the tonic activity on the levator increases and raises the eyelid. With decreases in levator activity, the passive downward forces pull the lid down until the passive closing and active opening forces again match. By understanding these forces, it is possible to determine which element or elements of the lid system that are affected by a local, systemic, or neurologic disorder.

The eyelids contain three main muscles innervated by three different neural networks. The oculomotor nerve (see Chapter 17) provides innervation to the levator palpebrae superioris muscle that keeps the eyelids open, a function assisted somewhat by Müller’s muscle which is innervated by the sympathetic nervous system (see Chapter 14). Eyelid closure is achieved by contraction of the orbicularis oculi muscle, innervated by the facial nerve (see below).

SURFACE ANATOMY

The palpebral fissure, the opening between the upper and lower eyelids, is the entrance into the conjunctival sac bounded by the margins of the lids (Fig. 24.1). When the lids are open, the palpebral fissure is an asymmetric ellipse 22–30 mm long and 12–15 mm high. In newborn infants, the upper eyelid rises well above the cornea, and the lower eyelid crosses the inferior margin of the cornea. By adulthood, the upper eyelid covers the upper cornea, and the lower lid lies at or slightly below its inferior margin. The lateral extent of the palpebral fissure is at the same level or slightly higher than its medial extent when the eyelids are open, but when they are closed, the outer angle of the fissure is 2–3 mm below the level of the median angle. The eyelids are firmly fixed at the inner angle, and only the position of the outer canthus is changed on opening and closing the lids.

The width of the palpebral fissure often influences judgment regarding the size and prominence of the eye. When one fissure is abnormally wide, as may occur in patients with thyroid orbitopathy or upgaze paresis, the eye may appear

Figure 24.1. Superficial anatomy of the orbit and eyelids.
proptotic or enlarged when, in fact, it is of normal size and is in the same anteroposterior position in the orbit as the opposite eye. When the palpebral fissure is narrowed, as in Horner syndrome, the eye may appear enophthalmic.

The eyelids have several characteristic folds. A well-delineated superior palpebral sulcus, also called the upper eyelid crease, lies 3–4 mm above the lid margin and is the most conspicuous of the eyelid folds (Fig. 24.1). The crease represents the interface where the orbicularis oculi attaches to the tarsus and where the levator aponeurosis attaches to the pre-tarsal skin. It divides each eyelid into an orbital and a tarsal portion. The orbital portion lies between the margins of the orbit and the globe, whereas the tarsal portion lies in direct relation to the globe.

The upper tarsus extends about 29 mm from medial to lateral and is 10 mm high at the center of the lid. The lower tarsus is 3.5–5 mm high at the center of the lower lid but is the same length as the upper tarsus. The tarsi contain sebaceous glands of the eyelid, called the Meibomian glands. Ffirmly attached to the posterior aspect of the tarsus is the tarsal conjunctiva.

The orbito-septum is a mesodermal layer extending from the bony margin of the orbit toward the upper tarsus. The septum attaches to the aponeurosis about 3.4 mm above the upper border of the tarsus. It acts as a barrier for anterior migration of fat pads and as an attachment of the aponeurosis to the overlying skin.

**ANATOMY OF THE MUSCLES OF EYELID OPENING AND POSITIONING**

The principal muscle involved in opening the upper eyelid and in maintaining normal lid posture is the levator palpebrae superioris. Two accessory muscles of lid opening, Müller’s muscle and the frontalis muscle, play only minor roles.

**Levator Palpebrae Superioris**

The levator palpebrae superioris muscle originates at the annulus of Zinn and courses anteriorly along the superior aspect of the orbit, passing through a suspensory structure, Whitnall’s ligament (also called the superior transverse ligament), to which it is attached by bands of connective tissue (Fig. 24.2). This suspensory ligament alters the pulling direction of the levator palpebrae from horizontal to nearly vertical. The attachment of the levator to Whitnall’s ligament is an important component of the passive lid-closing forces acting upon the upper eyelid and described above (Fig. 24.3). The levator does not extend all the way from its origin to the superior tarsus. Instead, it begins to gradually transition from muscle to tendon just posterior to Whitnall’s ligament, such that anterior to Whitnall’s ligament, it exists only as a tendinous aponeurosis. It is this aponeurosis that fuses with the orbital septum and attaches to the superior tarsus.

The levator contains four types of muscles fibers (1). Three are singly innervated fibers similar to those found in other extraocular muscles: red fast-twitch fibers, intermediate fast-twitch fibers, and pale fast-twitch fibers. Fast-twitch fibers, particularly the red type, are highly resistant to fatigue and thus help sustain contraction against the passive downward force produced by various ligaments and gravity. Fast-twitch fibers also probably play an important role in the generation of lid saccades. The fourth type of muscle fiber is found only in the levator: the levator slow-twitch fiber. The slow-twitch fibers of the levator may assist the red fast-twitch fibers in resisting the passive downward forces described above. Thus, the fiber composition of the human levator palpebrae is consistent with its two main roles: sustained contraction against the passive downward forces acting on the upper lid and active upward lid movements accompanying saccadic eye movements (2).

The levator palpebrae superioris is innervated by branches of the superior division of the oculomotor nerve. A detailed description of the anatomy of the nerve is presented in Chapter 17 of this text and is not repeated here.

**Müller’s Muscle**

Müller’s muscle is a thin band of smooth muscle about 10 mm in width that inserts on the superior border of the upper tarsus (Figs. 24.2 and 24.4). The muscle originates 10–12 mm above the tarsus from tendons inserted near the origin of the levator aponeurosis. Müller’s muscle is innervated by fibers of the oculosympathetic pathway. These are described in detail in Chapter 14 of this text and are not repeated here. The mean total excursion of Müller’s muscle is 3 mm, which raises the upper eyelid 1.5 mm (3,4).

Although Müller’s muscle is located in the upper eyelid, there is a similar, much smaller, sympathetically innervated muscle located in the lower eyelid. Hypofunction of this

![Figure 24.2.](image-url)
Figure 24.3. Schematic of the elastic structures responsible for the passive forces acting on the upper and lower eyelid. Elevating the lid to the open position or in upward gaze, increases the tension along the orbicularis oculi (not shown), Whitnall’s ligament, the canthal-tarsal complex, and Lockwood’s ligament. Relaxation of the levator palpebrae releases this stored elastic energy causing the eyelid to close or descend on downgaze. (From Sibony PA, Manning KA, Evinger C. Eyelid movements in facial paralysis. Arch Ophthalmol 1991;109:1555–1561.)

Figure 24.4. Anatomy of Müller’s muscle. Anterior view of transected and partially dissected upper and lower eyelids. The lateral portion of the orbital septum and the levator have been excised to demonstrate adjacent relationships. Note that Müller’s muscle (mm) is located just underneath the levator aponeurosis (la) and inserts directly into the upper aspect of the superior tarsus (st), whereas the levator aponeurosis inserts into the anterior aspect of the tarsus (la-in), it, inferior tarsus; la-lh, lateral horn of levator aponeurosis; lct, lateral canthal tendon; lg-ol, orbital lobe of lacrimal gland; lg-pl, palpebral lobe of lacrimal gland; llr, lower lid retractors; lof, lateral orbital fat; os, orbital septum (cut edge); paf, preaponeurotic fat; wl, Whitnall’s ligament. (From Rootman J, Stewart B, Goldberg RA. Orbital Surgery: A Conceptual Approach. Philadelphia: Lippincott-Raven, 1995.)

Figure 24.5. Anatomy of the orbicularis oculi muscle and associated muscles. Note that the orbicularis has three separate divisions: pretarsal, preseptal, and palpebral.
muscle (such as occurs in Horner syndrome [discussion following]) results in slight elevation of the lower eyelid (up-side-down ptosis).

Frontalis and Associated Muscles

The muscles controlling the eyebrow contribute to the eyelid appearance and, to a lesser extent, its elevation. Control of the eyebrow involves three sets of muscles: frontalis, procerus, and corrugator superciliaris (Fig. 24.5). The frontalis has no bony insertions. It originates as an extension of the anterior belly of the epicranius with connections to the occipital muscle via the galea aponeurotica, and it has cutaneous insertions at the level of the eyebrow. Frontalis fibers also intermingle with peripheral orbicularis oculi muscle fibers. Contraction of the frontalis raises the entire eyebrow and the upper eyelid through its cutaneous and orbicularis oculi connections.

The procerus muscle originates from the medial portion of the lower region of the frontalis muscle and inserts on the nasal bone. Contraction of the procerus pulls the medial portion of the eyebrow downward. The corrugator superciliaris lies beneath the frontalis and orbicularis oculi originating from the medial origin of the frontal bone. The corrugator superciliaris extends 2–3 cm laterally, where it blends with the frontalis and orbicularis oculi fibers. Contraction of the corrugator superciliaris pulls the eyebrow medially and downward.

ABNORMALITIES OF EYELID OPENING

Abnormalities of eyelid opening include ptosis—insufficient opening of the eyelid—which can be caused by a variety of congenital and acquired disorders, many of which are of neuro-ophthalmologic importance, and retraction, a very sign of thyroid eye disease and an occasional sign of other neuropathic, neuromuscular, myopathic, and mechanical disorders.

PTOSIS

A deficiency of levator tonus produces the clinical sign called blepharoptosis or ptosis. Ptosis may be produced by damage to the motor system controlling eyelid elevation and position at any point along the pathway, from the cerebral cortex to the levator muscle itself. Topical diagnosis of ptosis depends on the character of the deficiency and on evidence of neuropathic, neuromuscular, aponeurotic, developmental, mechanical, or myopathic disease (8–10).

The degree of ptosis can be quantified clinically by measuring the vertical length of the palpebral fissure—about 9 mm in normal subjects—assuming the lower eyelid is normally positioned. A more useful measure is the distance between the upper lid margin and the midcorneal reflex when the globe is in primary position. This is called the uMRD. Ptosis can be defined as an uMRD less than 2 mm or an asymmetry of more than 2 mm between eyes (11). Using this definition, most patients with ptosis exhibit a contraction of the superior visual field to 30° or less (12).

Neurogenic Ptosis

Neurogenic ptosis may be caused by supranuclear, nuclear, or infranuclear dysfunction. In some cases, associated symptoms and signs make the distinctions among these causes obvious; in other cases, however, neither the location nor the nature of the lesion is clear.

Supranuclear Ptosis

Unilateral ptosis is a rare manifestation of hemisphere dysfunction. This manifestation, called cortical ptosis, usually, although not invariably, is contralateral to the lesion. Unilateral ptosis has been described contralateral to lesions of the angular gyrus (13), to seizure foci in the temporal lobe (14), to hemispheric stroke (15,16), and to frontal lobe arteriovenous malformations (AVMs) (17). It also has been described ipsilateral to ischemic hemispheric strokes (15,16) (Fig. 24.6).

Bilateral cortical ptosis can also occur (16,17). It is associated most frequently with extensive nondominant hemisphere lesions. In most instances, the ptosis is accompanied by midline shift, gaze deviation to the right, and other signs of right hemisphere dysfunction, including left hemiparesis.