Ptosis

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Blepharoptosis, ‘the fallen eyelid’, is a clinical sign that neurologists encounter regularly. Knowledge of the anatomy and the normal physiology of the eyelid makes it easier to understand the various ways in which ptosis may present. The aetiology of ptosis can be divided into structural abnormalities affecting the eyelid muscles and/or surrounding tissues in the orbit, myogenic causes, neurogenic causes, disorders of the neuromuscular junction and central causes. Differentiating between these causes can often be achieved by a carefully directed history and examination. Investigation depends on the clinical assessment and hence the likely underlying cause. Treatment is usually directed at the underlying pathology but occasionally oculoplastic surgery is appropriate. This review summarises these aspects and provides a guide to the clinical assessment of ptosis.

Introduction

Ptosis is a lowering of the eyelid to below its normal position. The word ‘ptosis’ derives from the Greek ‘πτωσις’, which translates as ‘to fall’. It is an abbreviation of ‘blepharoptosis’—a fallen eyelid—but this longer version is now almost never used. The normal palpebral fissure measures 12–15 mm. The distance between the corneal light reflex and the upper eyelid margin is termed the upper marginal reflex distance. These two measurements are used for objective assessment of ptosis (figure 1). The official definition of ptosis is an upper marginal reflex distance below 2 mm or an asymmetry of more than 2 mm between the eyes. Ptosis has many causes and is a presenting symptom in both emergency and outpatient settings. While most ptosis presents

to ophthalmologists, neurologists often see cases in day to day practice. Ptosis may point towards something as dramatic as a leaking aneurysm or something as mundane as a soft tissue injury from rubbing the eye. Careful clinical assessment will prevent unnecessary investigations but may also save a patient’s life. This review focuses on acquired ptosis in adults, starting with an overview of the normal anatomy and function and then a discussion of possible causes. We end with a suggested clinical approach to the patient with ptosis.

Anatomy and function

The eyelid anatomy is shown in figure 2. The levator palpebrae superioris (LPS) is the primary muscle responsible for lid elevation. It arises from the back of the orbit and extends forwards over the cone of eye muscles. It inserts into the eyelid and the tarsal plate, a fibrous semicircular structure which gives the upper eyelid its shape. The LPS is supplied by the superior division of the oculomotor nerve. The way that the LPS attaches to the tarsal plate is modified by the underlying Müller’s muscle. This involuntary muscle, comprising sympathetically innervated smooth muscle, has the capacity to ‘tighten’ the attachment and so raise the lid a few millimetres. Two other muscles affecting the final position of the eyelid are the frontalis muscle and the orbicularis oculi, both supplied by the facial nerve. Frontalis contraction helps to elevate the lid by acting indirectly on the surrounding soft tissues, while orbicularis oculi contraction depresses the eyelid.

The eyelid is held open when awake by the tonic action of LPS, punctuated intermittently by blinks. The upper eyelid normally covers the top

Figure 1 Measurement of eyelid excursion. The top left image shows the margin of the closed eyelid at a level of 20 mm. When the eyelids are fully elevated (bottom left), this increases to 33 mm, giving a normal eyelid excursion of 13 mm. The right-sided image shows a normal eyelid crease (A), upper marginal reflex distance (B) and palpebral fissure (C).

Figure 2 Anatomy of the upper eyelid. (Reprinted from Spalton D et al¹ with permission.)

20% of the cornea but its exact position is determined by several factors. It rises and falls with vertical eye movements but it is also affected by the horizontal position of the eye, being slightly lowered when the eye is either adducted or abducted. The state of arousal influences lid position: fatigue is associated with reduced LPS activity but the overall level of sympathetic activity affects the tone in Müller's muscle so the eyelid sits slightly higher in circumstances associated with increased arousal.

The range of eyelid movement from full elevation to closure ('eyelid excursion') is usually greater than 12 mm.² This range can easily be measured and forms an important part of the assessment of any patient with ptosis (figure 1). LPS function is assessed by measuring the difference in eyelid margin position in upgaze and downgaze (while holding the eyebrow down to prevent frontalis activity). The patient should then be asked to pursue an object slowly from upgaze to downgaze in order to detect lid

retraction or lid lag. Fatigability is assessed by detecting any lowering of the eyelid during sustained upgaze for at least 60 s.

Causes of ptosis

The commonest cause of ptosis is disinsertion of the LPS tendon from the tarsal plate (lid dehiscence or aponeurotic ptosis). This causes the whole eyelid to sit low, even though the normal range of movement is preserved. It is important to distinguish this condition from ptosis due to muscle weakness. Ptosis may also result from reduced activity of the muscles that elevate the eyelid. In some situations, the lid may appear lower than normal because of structural abnormalities affecting either the eyelid itself or the surrounding orbit, or because of active contraction of the orbicularis oculi. Table 1 summarises the causes of ptosis but we briefly discuss each in turn.

Structural causes

It is possible for an eyelid with normal muscle function to appear to sit lower than normal. This

Table 1 Causes of ptosis

1. Congenital
 - Isolated congenital ptosis
 - Congenital myasthenic syndromes
 - Transient neonatal myasthenia (myasthenic mother)
 - Anomalous synkineses (eg, jaw winking)
 - Blepharophimosis and branchial arch syndromes
2. Structural
 - Levator dehiscence (age, trauma, contact lenses)
 - Eyelid or orbital tumours
 - Eyelid swelling (allergy, infection)
3. Sympathetic supply (Horner's syndrome)
4. Muscle/neuromuscular junction
 - Myasthenia gravis
 - Myotonic dystrophy
 - Chronic progressive external ophthalmoplegia
 - Other mitochondrial cytopathies (inherited and acquired)
 - Oculopharyngeal muscular dystrophy
 - Graves' disease
5. Oculomotor nerve
 - See table 2
6. Central
 - 'Cortical' ptosis
 - Hemifacial spasm
 - Essential blepharospasm
 - Apraxia of eyelid opening
7. Miscellaneous
 - Fatigue, lethargy, drowsiness, coma
 - Trauma
 - Hysteria, 'functional pseudoptosis'

usually results from structural problems but can be from overactivity of eye closure. Structural problems can include those affecting the globe, the eyelid or the tissues above the eye.

Globe

Any process causing the eye to retract into the orbit may give the appearance of ptosis because the eyelid slips down over the retracted eye. Congenital microphthalmos rarely presents to the adult neurologist but Duane's retraction syndrome may do so. This is a congenital ocular motility disorder which causes restriction of eye movement, usually of adduction. It is characterised by globe retraction during movement in the opposite direction to the restriction, due to co-contraction of extraocular muscles.³ This retraction narrows the palpebral fissure. Of the acquired conditions causing globe retraction, damage to the orbital floor by trauma or malignancy is the most common. Note that extraocular muscle palsy impairing elevation may also give the appearance of ptosis.

Figure 3 Levator dehiscence affecting the left eye. Note the increased distance between the lid margin and the skin crease on that side with preserved range of movement.

Figure 4 Right-sided Horner's syndrome after lateral medullary infarction, demonstrating the subtlety of this abnormality.

Eyelid

Any condition causing the upper eyelid to swell may cause ptosis, as the enlarged lid narrows the palpebral fissure. Inflammation or infection is usually relatively obvious, but tumours, most commonly neurofibromata, may be more subtle. 'Floppy lids syndrome' is a recently described syndrome occurring mainly in overweight men with obstructive sleep apnoea and/or hypertension.⁴ The normal stiffness of the upper eyelids is reduced and they are easily everted. The lids are often lengthened, giving the appearance of ptosis, and there is often accompanying conjunctival irritation due to eyelashes pointing in the wrong direction ('lash ptosis'). Note that any cause of lid elevation in the contralateral eye may give the appearance of ptosis.

Surrounding tissues

In dermatochalasia, the upper orbital skin becomes lax and hangs over the true eyelid, sometimes even impairing vision. This is not uncommon in elderly people and is easily mistaken for ptosis.

Levator dehiscence (aponeurotic ptosis)

This is the most common cause of a lowered eyelid. It typically occurs in middle aged to elderly patients and is caused by a disinsertion of the LPS tendon from the tarsal plate. As a result, the lid sits lower than normal but has a normal range of movement. Lid dehiscence may appear relatively suddenly following trauma or ocular surgery (usually cataract extraction) but its onset is usually subacute in the elderly. It can be caused by wearing hard contact lenses and may even follow rubbing the eye. Clinically, it is characterised by a high skin crease (more than 7 mm from the lid margin) and a rather thinned eyelid but a relatively normal range of lid movement (figure 3). Failure to recognise this diagnosis may result in unnecessary tests. It can be corrected by a simple surgical procedure if functionally disabling.

Reduced sympathetic activity

Any condition interrupting the sympathetic supply to Müller's muscle may cause ptosis. This almost always occurs in the context of a Horner's syndrome (figure 4). The ptosis is only ever partial and is often very subtle, as the maximum excursion of the muscle is 3 mm. It may be accompanied by minor elevation of the lower lid due to involvement of the much smaller sympathetically innervated muscle in the lower eyelid; this can give the eye a 'sunken' appearance. The presence of other features of a Horner's syndrome (ie, a miotic pupil, anhidrosis and facial vasomotor abnormalities) depend on the site of the lesion. With congenital or very chronic lesions, there may be iris depigmentation. Lesions causing Horner's syndrome are traditionally classified according to the three neurons involved in the sympathetic supply (hypothalamus to spinal cord, spinal cord to sympathetic ganglion and sympathetic ganglion to end organ). Lesion localisation

is often determined by the presence or absence of accompanying clinical abnormalities and by radiological investigation but pharmacological testing of the pupil is occasionally useful. Further investigation and treatment (if any) depend on the likely site of the lesion. A comprehensive list and details of pharmacological pupil testing can be found elsewhere.²

Myogenic/neurogenic

The conditions causing myogenic or neurogenic ptosis are most easily considered on the basis of anatomy. We discuss these in terms of lesions affecting muscle, the neuromuscular junction, the oculomotor nerve and the CNS.

Muscle

Several conditions affect the LPS itself. These are often bilateral and may also affect the extraocular muscles, causing disordered eye movements. If the onset is slow, as in many of the congenital syndromes, patients are often unaware of their ptosis and may not experience diplopia, even though their eyes are manifestly malaligned. As the ptosis becomes more severe, patients increasingly have to adopt a characteristic backward head tilt to prevent the ptotic lids from obscuring vision.

Mitochondrial disorders

Because the eyelids and extraocular muscles have a relatively large number of mitochondria due to high levels of metabolic activity, mitochondrial disorders often cause ptosis. The most common of these is chronic progressive external ophthalmoplegia, in particular Kearns–Sayre syndrome, although mitochondrial neurogastrointestinal encephalopathy (MNGIE), mitochondrial encephalomyopathy, lactic acidosis and stroke-like episodes (MERRF) and several other mitochondrial function mutation disorders may cause

Figure 5 Myasthenia gravis: before treatment (left) and after 3 days of intravenous immunoglobulin (right). Note previous blepharoplasty scars.

Figure 6 Ptosis props are rarely effective.

Figure 7 Partial right oculomotor nerve palsy with mild ptosis, mydriasis and a 'down and out' eye.

Table 2 Causes of oculomotor nerve palsy (reprinted from Lueck C¹⁰ with permission)

Common		Uncommon	Rare
Localisation by anatomical site			
Fascicular		Head trauma Brainstem infarction Multiple sclerosis	Brainstem tumour Brainstem haemorrhage Syphilis Poliomyelitis Encephalitis
Subarachnoid	Head trauma Aneurysm (posterior communicating artery) Tentorial herniation	Neurosurgical complication Aneurysm (basilar artery, superior cerebellar artery) Subarachnoid haemorrhage Meningitis (infective, inflammatory or neoplastic)	Arteriovenous malformation Ectatic basilar or posterior communicating artery Oculomotor nerve tumour Post lumbar puncture/ myelography Hypertrophic pachymeningitis Superficial siderosis Idiopathic intracranial hypertension Cerebral venous sinus thrombosis
Cavernous sinus/ superior orbital fissure	Head trauma Tumour, lymphoma	Tumour Carotico-cavernous fistula Internal carotid artery aneurysm, occlusion or dissection Cavernous sinus thrombosis Tolosa–Hunt syndrome Wegener's granulomatosis Herpes zoster	Pituitary apoplexy
Orbit	Head trauma	Tumour, lymphoma	Infections (eg, mucor mycosis) Retrobulbar anaesthesia Paget's disease of skull
Localisation uncertain			
Congenital		'Congenital oculomotor nerve palsy'	Aplasia Congenital fibrosis of the extraocular muscles
Vascular	Nerve infarction (diabetes mellitus, hypertension, etc)	Nerve infarction (giant cell arteritis, vasculitis)	Carotid artery stenosis Protein S deficiency Hyperviscosity syndrome Cocaine, sildenafil
Inflammatory/infective		Sarcoidosis Post-viral Sinusitis	Sjögren's syndrome, Systemic lupus erythematosus
Metabolic/toxic			Ophthalmoplegic migraine Wernicke's encephalopathy Drug/substance toxicity
Miscellaneous	Idiopathic		Dental anaesthesia

Figure 8 Mild right-sided ptosis in Miller Fisher syndrome. Note the mid-dilated pupils and the left upper lid retraction as attempted compensation for the ptosis. (B) The ptosis worsens when the retracted lid is elevated manually, so-called enhanced ptosis.

ptosis. Because some mitochondrial proteins are coded for by mitochondrial DNA while others are coded for by nuclear DNA, some of these conditions are inherited maternally while others are inherited autosomally. In those that are maternally inherited, there may be a variable degree of penetrance due to heteroplasmy. There is often a family history but the absence of affected relatives does not exclude mitochondrial disease. Patients typically present in childhood or early adult life with progressive bilateral ptosis and ophthalmoparesis. The abnormalities may be restricted to the eyes and eyelids, or there may be more generalised abnormalities such as myopathy, scoliosis, pigmentary retinopathy and cardiac conduction defects (as in Kearns–Sayre syndrome). It is now possible to detect many genetic causes of mitochondrial dysfunction on blood tests. If these are negative but mitochondrial disease is still suspected, further investigation in the form of muscle biopsy may be appropriate because histology may show abnormalities of mitochondria—for example, ragged red fibres—and further genetic testing may be possible on the muscle tissue itself. Importantly, mitochondrial dysfunction may be acquired, particularly from drug treatment. A recent report highlighted ptosis occurring in patients treated with highly active antiretroviral treatment for HIV.⁵

Muscular dystrophies

The eyelids are usually spared in the dystrophinopathies and the limb girdle group of muscular dystrophies. However, some other muscular dystrophies are associated with ptosis. People with myotonic dystrophy, particularly DM1, commonly have ptosis. It is usually mild but can become severe enough to impair vision as the disease progresses. If ptosis occurs in facioscapulo-humeral dystrophy, it is a late finding despite facial weakness being characteristic. Oculopharyngeal muscular dystrophy is a rare condition due to abnormalities of the polyadenine binding protein

nuclear 1 gene on chromosome 14q11. It is characterised by progressive ptosis, dysphagia and proximal limb weakness.⁶ Symptoms usually start in mid-life and are commonly initially mistaken for ocular myasthenia gravis (MG, see below). Several infantile onset muscular dystrophies, including muscle–eye–brain disease, can cause ptosis; these conditions are associated with other severe neurological abnormalities and are rarely encountered by adult neurologists.

Myopathies

Myopathies rarely affect the eyelid. Most drug induced myopathies affect large muscles and ptosis would be highly atypical. Inflammatory myopathies, including polymyositis, dermatomyositis and inclusion body myositis almost never cause ptosis. Isolated ocular myositis is rare but may result in ptosis if the LPS/superior rectus muscle complex is affected. Rarely, amyloidosis may affect the LPS, causing a (usually unilateral) ptosis, often preceded by an orbital mass.⁷

Dysthyroid eye disease

This is typically associated with lid retraction. However, it can cause ptosis, usually through mechanical disruption of the LPS as it is stretched by the proptosed globe and hypertrophied muscles. The diagnosis is usually fairly obvious in a patient with disease severe enough to cause ptosis. However, also consider MG if a patient with hyperthyroidism develops ptosis.

Neuromuscular junction

It is well known that patients with MG often have ptosis (figure 5), which may be demonstrably fatigable. Several signs in MG relate to eyelid function, including Cogan's lid twitch sign, curtaining and enhanced ptosis. Cogan's lid twitch is seen after a patient looks down. It appears as a brief overshoot of the upper eyelid as the patient attempts to look back up to the primary position (straight ahead). This means that the sclera is transiently visible above the cornea. 'Curtaining'

and 'enhanced ptosis' refer to the phenomenon of contralateral eyelid lowering when an eyelid is manually elevated. 'Curtaining' occurs when the ptotic eyelid is elevated resulting in a ptosis of the normal eyelid. 'Enhanced ptosis' occurs when the normal eyelid is lifted resulting in further drooping of the ptotic eyelid. Both are explained by Hering's law of equal innervation to paired yoke muscles (ie, the eyelids). Note that these signs are not specific to myasthenia and may be seen in other conditions, including Miller Fisher syndrome (figure 6) and botulism (although in both Miller Fisher syndrome and botulism the pupils are often involved, and typically dilated). Lambert-Eaton myasthenic syndrome is frequently associated with ptosis but almost never in isolation or as the presenting feature.

Congenital myasthenic syndromes

This is a heterogeneous group of rare disorders of the neuromuscular junction, usually inherited in an autosomal recessive pattern. Fatigable ptosis is very common. Multiple identified genetic defects can result in acetylcholine receptor deficiency, acetylcholinesterase deficiency or kinetic abnormalities of the acetylcholine receptor ion channels. The clinical phenotype depends on the genotype, and each type may also have characteristic neurophysiological features. These syndromes usually present in childhood but occasionally in adulthood when they may mimic MG or a muscular dystrophy. Congenital myasthenia is worth considering in seronegative myasthenic patients or those with an unexplained muscle disease.⁸

Neurogenic

Oculomotor nerve palsy is a common presentation to neurologists. The classical signs are a ptosis (ranging from subtle to complete), mydriasis and ophthalmoparesis with the eye resting 'down and out'. However, partial lesions are common (figure 7). Oculomotor nerve lesions may arise anywhere along its course, including the nucleus, the fascicles in the midbrain tegmentum and the nerve itself as it passes through the subarachnoid space, the cavernous sinus, the superior orbital fissure and the orbit (table 2).

The nuclear supply of the LPS arises from the central caudal subnucleus of the oculomotor nucleus. This is a single midline structure supplying the LPS on both sides. Hence ptosis resulting from lesions of the oculomotor nucleus is typically bilateral. Because there may be damage to adjacent neural pathways such as the corticospinal tract or red nucleus, fascicular lesions of the oculomotor

Figure 9 Left functional ptosis.

nerve are often associated with contralateral hemiparesis or contralateral limb ataxia.

The oculomotor nerve divides into superior and inferior divisions and these can be damaged individually. The LPS is supplied by the superior division. Superior divisional palsies therefore cause ptosis and elevation failure (due to superior rectus involvement), while inferior divisional palsies cause ophthalmoparesis, frequently with pupil involvement but spare the LPS.

There are many possible causes of oculomotor nerve damage. These include giant cell arteritis, or trauma and compression by an aneurysm (usually of the posterior communicating artery). Infection, other causes of inflammation, thrombosis or tumour can affect the oculomotor nerve in the cavernous sinus, superior orbital fissure or orbit. Similarly, the it may be affected by several metabolic or inflammatory disorders such as diabetes mellitus or the Miller Fisher syndrome, associated with antibodies to ganglioside GQ_{1b} (figure 8). In Miller Fisher syndrome, extraocular muscle involvement is the rule but there are reports of isolated ptosis.⁹ Space does not permit a more detailed discussion of oculomotor nerve palsies here and the interested reader is referred elsewhere.¹⁰

Central causes

Cerebral cortex

Cortical (or supranuclear) ptosis is unusual but well recognised. Exactly how this happens is not clear, granted that the central caudal nucleus innervates the LPS on both sides. When it occurs, the ptosis is usually contralateral to the lesion, but ipsilateral and bilateral cases have been reported. It occurs most frequently in large non-dominant hemisphere lesions.¹¹

Basal ganglia

Apraxia of eyelid opening may occur in several neurological conditions, most commonly progressive supranuclear palsy. It manifests as a

(transient) inability to open the eyes although the eyelids can be held open normally once they are open. Patients often have to open their eyes with their fingers to overcome the apraxia. As eyelid function is normal, this is not a true ptosis.

Excessive orbicularis activity

This may be mistaken for ptosis. The excessive activity may be involuntary, as in hemifacial spasm, aberrant regeneration following Bell's palsy, or essential blepharospasm. Alternatively, it may be voluntary as in functional pseudoptosis.¹² Functional ptosis is recognisable by normal levator function with apparent weakness of the frontalis and overcontraction of the orbicularis oculi (figure 9).

Clinical management

History

The history in a patient with ptosis should cover the following points:

- Is one or are both eyes affected?
- Are there associated symptoms, specifically pain, malaise, visual disturbance, diplopia, dysphagia or muscle weakness elsewhere?
- What was the speed of onset, the duration and extent of progression of the ptosis? Does the ptosis fluctuate? Are there any obvious relieving and exacerbating factors?
- Does the patient have any comorbidities? In particular, are there vascular risk factors, a history of injury to the head, neck or chest, a history of HIV or other cause of immunosuppression, features of the metabolic syndrome, cancer or ocular disease? Are there any systemic features of giant cell arteritis?
- Is there a history of trauma, ophthalmic surgery or rubbing of the eyelid? Does the patient wear contact lenses?
- Has the patient had a blepharoplasty in the past?
- Is the patient taking any medications, regular or new?
- Is there a family history of ptosis or of other muscle weakness?

Examination

Patients with ptosis should undergo a general systemic and neurological examination. Of particular importance are the pupils, visual acuity and fields, funduscopy, extraocular and facial movements and other cranial nerve function. Ideally, patients should have an ocular examination, looking specifically for inflammation in the anterior chamber.

The eyelids should be inspected for symmetry, visible lesions, thickening, discolouration and involuntary movement. The position of the skin crease should be noted. The palpebral fissure, upper marginal reflex distance and eyelid

excursion should be measured as in figure 1. The behaviour of the eyelid during eye movements should then be noted.

Evidence of fatigability can be sought by asking the patient to maintain fixation in upgaze for 60 s and remeasuring the palpebral fissure immediately afterwards. An ice or rest test may demonstrate reversibility of the ptosis in myasthenia. Eye closure is frequently weak in ocular myasthenia.

Diagnosis

Most causes of ptosis can be diagnosed clinically. Unequal pupil size suggests a Horner's syndrome or an oculomotor nerve palsy. An elevated skin crease with thinned eyelid but a normal range of movement suggests levator dehiscence. If the ptosis is fatigable, or if there are supportive systemic features, it may be possible to diagnose MG. Dysfunction of extraocular muscles suggests a myopathy or muscular dystrophy if longstanding, and an oculomotor nerve palsy or a neuropathy (such as Miller Fisher syndrome) if of recent onset. Pain at the onset of ptosis suggests the possibility of an aneurysm compressing the oculomotor nerve. Other clinical features such as visual loss or systemic symptoms may suggest giant cell arteritis.

Investigation and management

The causes of ptosis are so numerous that it is impossible to give an exhaustive list of appropriate diagnostic tests. However, imaging may well be important to look for a possible aneurysm or other structural abnormalities. Blood tests (especially erythrocyte sedimentation rate), neurophysiology, lumbar puncture or muscle biopsy may each be appropriate, depending on the likely cause and whether or not there are accompanying symptoms or signs suggesting a specific diagnosis.

Similarly, the management of ptosis depends on its cause. In many cases, ptosis improves with time or with treatment of the underlying condition. When a reasonable amount of time has passed without improvement and further improvement is felt to be unlikely, referral to an oculoplastic surgeon may be considered. The most common surgical techniques used to treat ptosis are shortening of the LPS or insertion of tendon slings. Ptosis props are rarely effective (figure 6).

Conclusion

Ptosis has a myriad of causes but only a handful are seen frequently. The clinical examination is easily understood once the underlying anatomy is known. To avoid unnecessary investigations, it

Learning points

- Ptosis is common, and knowledge of how to examine a ptotic eyelid is useful to neurologists.
- The most common cause of ptosis is levator dehiscence. Other common causes are myasthenia gravis, Horner's syndrome and oculomotor nerve palsies.
- Pseudoptosis refers to the appearance of ptosis unrelated to eyelid dysfunction. Dermatochalasia is the most common cause. Be aware of other conditions that give the appearance of ptosis such as contralateral lid retraction or excessive orbicularis activity.
- Horner's syndrome may cause a very subtle ptosis, and pharmacological testing of the pupil may be required to confirm this diagnosis.
- Oculoplastic surgery should be reserved for cases where the diagnosis is clear and improvement is not expected.

is useful to try to determine whether the cause is likely to be structural, myogenic, neurogenic, neuromuscular or central. A directed history and examination often allows the clinician to localise the cause of the ptosis to a particular anatomical area, if not to reach a specific diagnosis. This in turn permits appropriate investigations and treatment to be instituted. The management of ptosis is very much dependent on the underlying cause but oculoplastic surgery should be considered for patients with static lesions.

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