

Abstract

Treatment of the ocular and periocular structures in facial nerve palsy has traditionally centered on treating exposure keratopathy and optimizing eyelid malposition. Facial nerve palsy has multiple causes, and it is paramount for the physician to identify the etiology of the palsy. Recent advances in the treatment of the eye in facial nerve palsy include eyelid weight placement, lower lid malposition correction, the recognition of meibomian gland dysfunction as a contributor to ocular surface disease, axial positioning of the globe, and cross-facial nerve grafting. Tarsorrhaphies are punitive and should only be used in rare instances. The purpose of this review is to update the ophthalmologist on recent advances in the treatment of facial nerve palsy.

The facial nerve innervates the muscles of the face, some of which are associated with eyelid closure and eyebrow elevation. Patients with facial nerve palsy often note difficulty closing the eye and elevating the brow. The causes of facial nerve palsy are varied. It can be easy for a physician to be lulled into a sense of security that the cause of the facial nerve palsy is inflammatory, for example, in the case of Bell's palsy. However, it is important to rule out more sinister causes of facial paralysis. Bell's palsy has a very characteristic acute onset over days. When facial nerve palsy is caused by a tumor, the onset is often gradual. When in doubt, the patient should be imaged.

First and foremost, lubrication of the eye should be instituted. This usually consists of artificial tears during the day and ointment at night. Lubrication should be increased as necessary. It is important for the patient to be able to feel when the eye is dry. If there is any indication of a neurotrophic cornea, the patient should be monitored more frequently and may be considered for a scleral contact lens. A tarsorrhaphy should be reserved for patients who are unable to comply with lubrication; the threat of tarsorrhaphy often encourages compliance.

A recently recognized component of ocular surface disease in facial nerve palsy is meibomian gland dysfunction.¹⁻³ The muscle of Riolan, which is closely associated with the orbicularis muscle and is innervated by the facial nerve, wraps around the meibomian glands.⁴ Contraction of the muscle of Riolan results in emptying of the meibomian glands. In the context of facial nerve palsy, the glands are no longer emptied, resulting in meibomian gland dysfunction. Studies using meibography have shown that in patients with unilateral facial nerve palsy, there is significant meibomian gland pathology on the side of the palsy compared to the contralateral side.^{1,3} Patients with facial nerve palsy should be instructed in meibomian gland hygiene (warm compresses at a minimum) to decrease the contribution of meibomian gland dysfunction to their ocular surface disease.

The timing of surgical intervention in patients with facial nerve palsy usually depends upon whether there is any significant exposure keratopathy due to upper and/or lower lid malposition, and whether there is any significant brow ptosis that interferes with the patient's visual field.

For those patients with injury to the facial nerve (iatrogenic or otherwise), surgical intervention may be performed sooner if recovery is not expected. In a typical Bell's palsy, there should be improvement in the condition within 6-12 months. If the patient has any difficulty with exposure keratopathy that does not respond to conservative measures, surgical intervention can be performed with the understanding that there may need for later revision or reversal of the surgery. Hyaluronic acid has been used as a "gel weight" to temporarily aid in the closure of the upper eyelid.⁵ Aberrant regeneration of the facial nerve often occurs after Bell's palsy, which can result in facial synkinesis. In these patients, botulinum toxin can be used to decrease the hypertonicity of the aberrantly innervated muscles.6

Upper eyelid closure is affected by facial nerve palsy. The addition of an upper eyelid weight is a commonly performed procedure to improve eyelid closure. Traditionally, the weight is placed in a pretarsal position. This results in the improvement of eyelid closure using gravity. Common weight materials include gold and platinum. However, approximately 5% of the population suffers



Figure 1. Visible gold weight in a patient who has pretarsal placement of the weight. Published with the patient's permission.

from gold allergy (type IV hypersensitivity),⁷ which can lead to the removal of an implanted gold weight. Platinum, meanwhile, has a very rare incidence of allergy; for this reason, platinum is the preferred material for implanted weights.

A pretarsal placement of the weight has distinct disadvantages. The weight is often visible (Figure 1), and with long-term facial nerve palsy, thinning of the orbicularis muscle may result in exposure and extrusion of the weight. Placement of the weight in a supratarsal position (Video 1) has advantages over the lower pretarsal position: the weight is covered by more tissue planes, resulting in a lower chance of exposure; a higher-positioned weight induces less astigmatism; the weight is less visible; and the levator aponeurosis can be recessed and incorporated in the eyelid crease incision, preventing anterior lamellar slide and lash ptosis.8,9 There have been no reports in the literature of levator injury in the use of this procedure.

Lower lid malposition secondary to facial nerve palsy has traditionally been described as an ectropion. However, loss of protractor (orbicularis) function results in unopposed lower lid retractor action. This results in a lower lid retraction more than an ectropion. In treating lower lid malposition in the setting of facial nerve palsy, it is necessary to include recession of the lower lid retractors.¹⁰ This is performed by placing a lower lid spacer with a lateral canthoplasty. There are many potential spacer types, including autogenous and synthetic. The author prefers the use of an acellular dermal matrix in primary repairs (**Video 2**).

Brow ptosis in facial nerve palsy is due to loss of the function of the frontalis muscle. There is a myriad of brow elevation techniques, including browpexy, direct, mid-forehead, pretrichial, endoscopic, and coronal. With a paralyzed frontalis muscle, a strong technique is needed to allow adequate, long-term elevation of the brow. A direct browplasty is very effective in facial



Figure 2. Well-healed direct browplasty scar on the left side. Published with the patient's permission.

There has been recent recognition of changes in the axial position of the globe in patients with facial nerve palsy. A studypending publication-has shown that in patients with unilateral facial nerve palsy, there is approximately 1 mm of proptosis on the side of the palsy compared to the contralateral side (manuscript submitted). This axial displacement may be related to the loss of orbicularis tone, posteriorly displacing the globe. This can result in worsening exposure keratopathy and upper and lower eyelid malposition. In patients with recalcitrant exposure keratopathy and eyelid malposition, a number of surgeons (including the author) have performed orbital decompression on the side of the facial nerve palsy to improve eyelid position and ocular surface disease (Figure 3). Controlled studies will need to be performed to determine if this is a safe and effective long-term procedure.

Lastly, cross-facial nerve grafting is a technique in which a nerve graft is connected to the contralateral facial nerve

to provide innervation to the paralyzed muscles on the side of the facial nerve palsy. This is a surgery primarily performed by plastic or head and neck surgeons. A number of studies have shown promising results with this procedure, including improved eye closure, but it is still considered to be under development.¹² It seems to be more effective early after a facial nerve injury, and in younger patients.

In summary, the treatment of periocular changes and ocular surface disease in patients with facial nerve palsy can be challenging. However, there have been newly recognized factors to consider in treating these patients. Meibomian gland disease is often present in these patients, and meibomian gland hygiene should be instituted. The axial position of the globe may exacerbate eyelid malposition and ocular surface disease; the surgeon may want to consider moving the globe relative to the eyelids rather than the eyelids relative to the globe. Brow ptosis can be effectively treated with a direct browplasty, and the scar usually heals favorably due to it being placed in a field of paralysis. Lastly, upper and lower lid lagophthalmos can be better treated by placing weights in a supratarsal position and recognizing the contribution of retraction to the lower lid malposition. Although these patients can be challenging, recognizing these contributing factors can give satisfactory results and a healthy ocular surface.



Figure 3. Patient before (left) and after (right) orbital decompression for lower lid retraction and lagophthalmos which is improved after the procedure. Published with the patient's permission.

Conflict of interest

Key points:

- Patients with facial nerve palsy often have a component of meibomian gland dysfunction.
- Supratarsal placement of a platinum weight has distinct advantages over pretarsal placement and the use of a gold
- Lower lid malposition in facial nerve palsy has a component of retraction, and this must be addressed for satisfactory
- There is relative exophthalmos on the side of a facial nerve palsy which may exacerbate upper and lower lid malposition.
- Cross-facial nerve grafting is a promising technique, but it is still under development.

Videos

Scrub in and watch the procedures on YouTube:





Video 2. Lower eyelid retraction repair with acellular dermal matrix.

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Video 1. Supratarsal platinum weight

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Is orbicularis weakness an under-recognized component of meibomian gland dysfunction?

Hypothesis:

Dry eye disease is one of the most common diseases, not only in ophthalmology but medicine in general.¹ Meibomian gland dysfunction (MGD) is the leading cause of evaporative dry eye disease, the most common form.² Multiple contributing factors have been implicated in the etiology of MGD. Changes in the consistency of the oil produced by the glands and associated inflammation are commonly cited components.³ Causes include hormonal changes, dysbiosis, Demodex infestation, and inflammation.³ Identifying causes and risk factors is