Metadata of the chapter that will be visualized online

ChapterTitle	Periorbital Surgical Rehabilitation After Facial Nerve Paralysis	
Chapter Sub-Title		
Chapter CopyRight - Year	Springer Science+Business Media, LLC 2010 (This will be the copyright line in the final PDF)	
Book Name	Ophthalmic Oncology	
Corresponding Author	Family Name	**Chang**
	Particle	
	Given Name	**Heather**
	Suffix	
	Division	
	Organization	Jules Stein Eye Institute, UCLA Medical Center
	Address	Los Angeles, CA, USA
	Email	hkwon88@yahoo.com
Author	Family Name	**Taban**
	Particle	
	Given Name	**Mehryar**
	Suffix	
	Division	Department of Orbito-Facial Surgery
	Organization	Jules Stein Eye Institute, University of California, Los Angeles
	Address	Los Angeles, CA, TX, USA
	Email	TabanMD@yahoo.com
Author	Family Name	**Nakra**
	Particle	
	Given Name	**Tanuj**
	Suffix	
	Division	Ophthalmic Plastic and Orbital Surgery
	Organization	Texas Oculoplastic Consultants
	Address	Austin, TX, USA
	Email	tnakra@tocaustin.com

Abstract

Facial nerve paralysis can result from a number of causes, including neoplasms, Bell's palsy, infections, trauma, congenital conditions, and idiopathic processes. Both the medical and social consequences of facial nerve paralysis can be distressing for patients. The most significant ophthalmic consequence of facial nerve paralysis is loss of function of the orbicularis oculi muscle. The complete assessment of a patient with facial nerve paralysis includes clinical evaluation of the resting tone and active function of the facial muscles, as well as determination of the extent of dry eye and the function of the lacrimal gland and lacrimal drainage system. The goal of medical therapy is symptomatic relief of dry eye and exposure keratopathy. Botulinum toxin can also be employed to treat other symptoms, such as synkinesis, hypertonicity, and spasms. The goal of surgical therapy is improved protection of the cornea, as well as a more symmetric static and dynamic appearance. Lagophthalmos and exposure keratopathy can be addressed with procedures such as surgical closure of the eyelids, known as tarsorrhaphy, or other alternatives, such as placement of an alloplastic gold weight in the upper eyelid, injection of hyaluronic acid gel into the upper eyelid, or palpebral springs. Ectropion also commonly results from facial nerve paralysis and can be improved with lateral or medial canthal procedures. Reanimation of the midface can be accomplished by any of several surgical techniques; some provide static support for the midface, while others attempt to restore dynamic movement to the paralyzed face.

Chapter 24
Periorbital Surgical Rehabilitation After Facial Nerve Paralysis

Heather Chang, Mehryar Taban, and Tanuj Nakra

Abstract Facial nerve paralysis can result from a number of causes, including neoplasms, Bell's palsy, infections, trauma, congenital conditions, and idiopathic processes. Both the medical and social consequences of facial nerve paralysis can be distressing for patients. The most significant ophthalmic consequence of facial nerve paralysis is loss of function of the orbicularis oculi muscle. The complete assessment of a patient with facial nerve paralysis includes clinical evaluation of the resting tone and active function of the facial muscles, as well as determination of the extent of dry eye and the function of the lacrimal gland and lacrimal drainage system. The goal of medical therapy is symptomatic relief of dry eye and exposure keratopathy. Botulinum toxin can also be employed to treat other symptoms, such as synkinesis, hypertonicity, and spasms. The goal of surgical therapy is improved protection of the cornea, as well as a more symmetric static and dynamic appearance. Lagophthalmos and exposure keratopathy can be addressed with procedures such as surgical closure of the eyelids, known as tarsorrhaphy, or other alternatives, such as placement of an alloplastic gold weight in the upper eyelid, injection of hyaluronic acid gel into the upper eyelid, or palpebral springs. Ectropion also commonly results from facial nerve paralysis and can be improved with lateral or medial canthal procedures. Reanimation of the midface can be accomplished by any of several surgical techniques; some provide static support for the midface, while others attempt to restore dynamic movement to the paralyzed face.

24.1 Introduction

Facial nerve paralysis can result from a number of causes, including neoplastic processes, Bell's palsy, infections, trauma, congenital conditions, and idiopathic processes. Tumors can lead to facial nerve paralysis directly by mass effect or

H. Chang (✉)
Jules Stein Eye Institute, UCLA Medical Center, Los Angeles, CA, USA
e-mail: hkwon88@yahoo.com

B. Esmaeli (ed.), *Ophthalmic Oncology*, M.D. Anderson Solid Tumor Oncology Series 6, DOI 10.1007/978-1-4419-0374-7_24,

nerve infiltration; facial nerve paralysis can also occur following tumor resection [1]. Tumors can impact the facial nerve centrally, at the cerebellopontine angle (e.g., acoustic neuromas and meningiomas), and peripherally (e.g., parotid gland tumors).

Facial nerve paralysis can be distressing for patients and has both medical and social consequences. Periocular sequelae of paralysis, such as exposure keratopathy and paralytic ectropion, can cause significant discomfort and morbidity. Activities of daily living, such as eating and speaking, are often affected, and this can cause the patient emotional distress. Facial nerve paralysis can be accompanied by synkinesis, in which attempted voluntary movements lead to involuntary and undesired movements of other facial muscles, which is another potential source of emotional distress. Patients with facial nerve paralysis often reduce their participation in social activities, which negatively affect their mental health [2].

The treatment of facial nerve paralysis includes both medical and surgical management. The goal of medical therapy is symptomatic relief of dry eye and exposure keratopathy. Botulinum toxin can also be employed to treat synkinesis, hypertonicity, and spasms [3]. The goal of surgical therapy is a more symmetric static and dynamic appearance, as well as protection of the cornea. Surgical therapy can address lagophthalmos, ectropion, brow ptosis, and facial droop.

24.2 Relevant Anatomy

The facial nerve, the seventh cranial nerve, has multiple functions. It provides motor innervation to the muscles of facial expression, the throat muscles, the posterior belly of the digastric muscle, and the auricular, stylohyoid, and stapedius muscles. It also provides parasympathetic innervation to the lacrimal and salivary glands, along with sensory innervation to the external ear and the anterior two-thirds of the tongue. The course of the facial nerve is complex (Fig. 24.1). Four nuclei within the brainstem supply the facial nerve, which exits the brainstem laterally at the cerebellopontine angle and travels with the eighth cranial nerve to enter the internal auditory canal. The nerve then travels within the temporal bone and gives off the greater and lesser superficial petrosal nerves, as well as the nerve to the stapedius muscle and the chorda tympani. The facial nerve courses through the stylomastoid foramen, exits the temporal bone, passes through the parotid gland, and then finally divides into the temporal, zygomatic, buccal, mandibular, and cervical branches, which, in turn, innervate the corresponding facial muscles (Fig. 24.1).

The most significant ophthalmic consequence of facial nerve paralysis is loss of function of the orbicularis oculi muscle. This muscle's major function is to close the palpebral aperture, opposing the action of the levator palpebrae superioris muscle. In addition, the orbicularis oculi powers the "lacrimal pump": the simultaneous

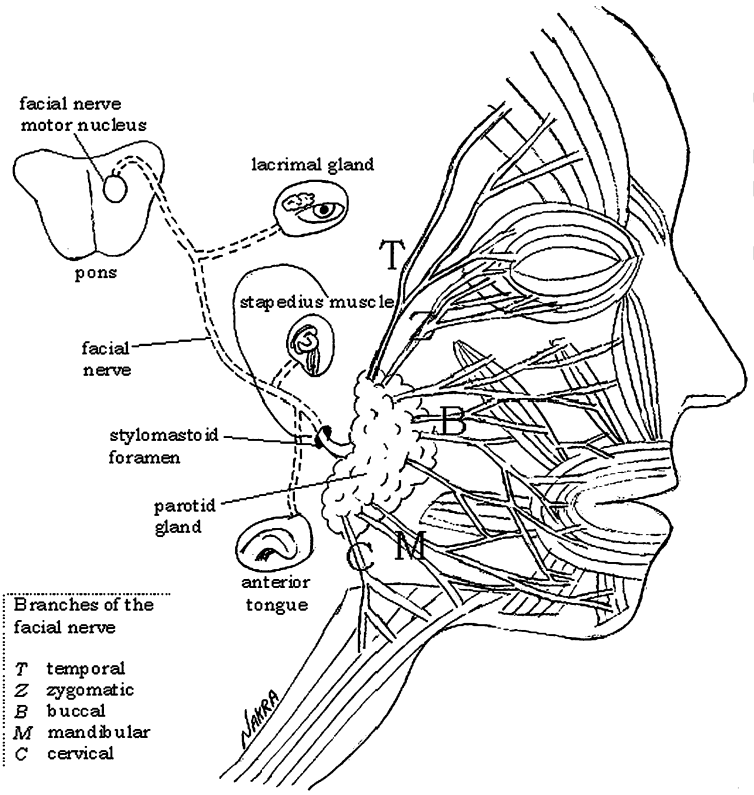

Fig. 24.1 Anatomy of the facial nerve. The facial nerve begins in the pons and provides parasympathetic innervation to the lacrimal and salivary glands. The nerve branches to innervate the stapedius muscle and anterior tongue and then exits the temporal bone through the stylomastoid foramen to innervate the parotid gland. Within the gland, the facial nerve divides into the temporal, zygomatic, buccal, mandibular, and cervical branches that provide motor innervation to the muscles of facial expression

contraction of the deep pre-tarsal head of the orbicularis oculi (Horner's muscle), which pulls the eyelid nasally and posteriorly, and the preseptal orbicularis oculi, which pulls the lacrimal sac laterally. This coordinated contraction compresses the canaliculi, pumping tears into the lacrimal sac. Subsequent relaxation of the orbicularis oculi then creates negative pressure in the canaliculi, thereby drawing tears in for the next pump cycle. Paralysis of the orbicularis oculi can result in lagophthalmos and paralytic ectropion, which places the ocular surface at risk of exposure and breakdown and can also result in epiphora.

Paralysis of other facial muscles can affect both voluntary and involuntary movements along with facial symmetry (Fig. 24.2). Decreased function of the frontalis muscle can lead to brow ptosis. The function of the dilator nasi muscle can be

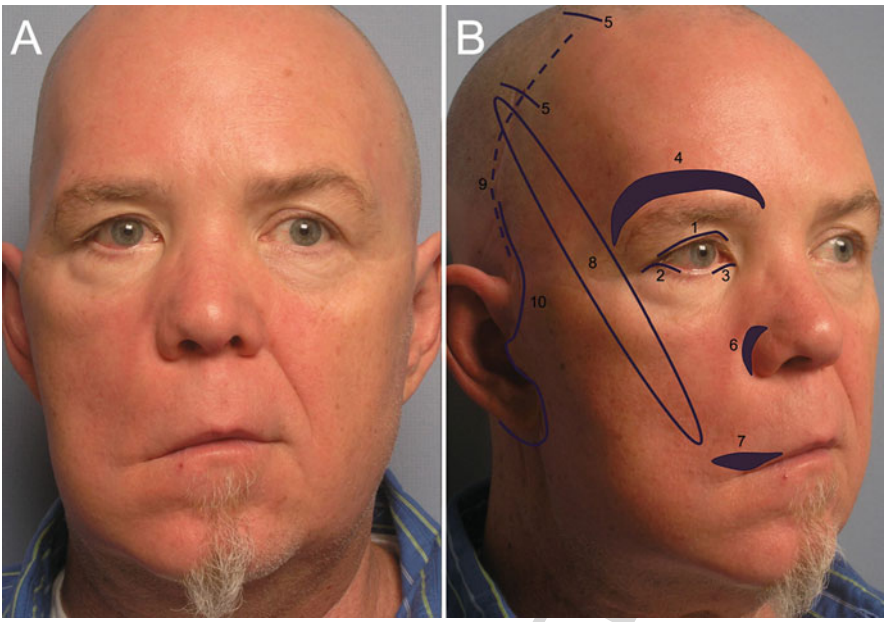

Fig. 24.2 Typical findings of facial nerve palsy and surgical approaches. (**a**) A 50-year-old man with right facial nerve paralysis after excision of a right acoustic neuroma. Note the paralytic right brow ptosis, mechanical dermatochalasis, lower eyelid ectropion, medial canthal laxity, nasolabial fold attenuation, alar collapse, lateral oral commissure droop, lower facial droop, and midfacial droop. (**b**) Composite photograph of the same patient illustrating the locations of various surgical approaches for comprehensive rehabilitation, including (1) insertion of an upper eyelid weight, (2) lateral canthoplasty and/or creation of a static lower eyelid sling, (3) medial canthopexy, (4) direct brow lift, (5) endoscopic forehead lift, (6) direct alar lift, (7) oral commissure lift, (8) midface lift, (9) temporalis muscle transfer or temporalis fascia sling, and (10) deep plane rhytidectomy and neck lift

affected, leading to nasal obstruction. Dysfunction of the orbicularis oris, risorius, or depressor anguli oris muscles contributes to asymmetry and can lead to problems with speech along with drooling and biting of the oral mucosa. Loss of function of the zygomaticus major and minor muscles leads to flattening of the nasolabial fold, increasing facial asymmetry and potentially an increase in the patient's emotional distress.

24.3 Clinical Evaluation

The complete assessment of a patient with facial nerve paralysis includes clinical evaluation of the resting tone and the active function of muscles as well as determination of the extent of dry eye and the function of the lacrimal gland and lacrimal drainage system.

24.3.1 Evaluation of Muscle Function

The evaluation begins with the patient at rest with careful observation of involuntary movements and resting facial symmetry. Asking the patient to voluntarily contract the various muscle groups can further elucidate the pattern of loss of innervation. Electrophysiology testing—electromyography, electroneurography, maximal stimulation test, and nerve excitability test—can be used to further clarify the amount of degeneration of the facial nerve. Grading scales to grade the severity of the impairment of facial nerve function have been developed and emphasize the muscles of facial expression [2, 4].

As previously mentioned, loss of orbicularis oculi function is the most significant ophthalmic consequence of facial nerve paralysis and can result in lagophthalmos and paralytic ectropion, placing the ocular surface at risk for exposure and breakdown. The strength of the orbicularis oculi can be evaluated by observing the degree of lagophthalmos and the presence of Bell's phenomenon at rest and with gentle and then forceful blinking. Orbicularis oculi tone can be further evaluated by asking the patient to close his/her eyes while the clinician tries to manually force open the patient's eyelids. The cornea should be evaluated for both sensation and signs of exposure. The eyelid position should be assessed for the presence of laxity and for the presence of paralytic ectropion.

Evaluating frontalis muscle function can be especially helpful in determining whether facial nerve palsy is central or peripheral. Central facial nerve palsy spares frontalis muscle function as the forehead has bilateral upper motor neuron innervation. In contrast, peripheral facial nerve palsy affects ipsilateral frontalis muscle innervation, and the patient is unable to wrinkle the ipsilateral forehead, resulting in brow ptosis.

Evaluation of midfacial muscle function is also an important part of the clinical examination. The nasolabial fold is created by the zygomaticus major and minor, levator labii superioris, and levator labii superioris alaeque nasi muscles, and this fold may be effaced or absent with facial nerve palsy. The nasal ala may appear collapsed because of loss of levator labii superioris alaeque nasi function and support of the internal nasal valve. The zygomaticus major, which draws the angle of the mouth laterally, and the zygomaticus minor, which elevates and everts the upper lip, can be assessed by asking the patient to attempt to smile. The orbicularis oris muscle narrows the orifice of the mouth, purses the lips, and plays an important role in speech and oral competence. Difficulty in speech, along with the presence of drooling, lip laxity, and biting of the oral mucosa, can indicate involvement of the facial nerve branches innervating the zygomaticus and orbicularis oris muscles.

24.3.2 Evaluation of Lacrimal Gland and Lacrimal Drainage System Function

Both lacrimal gland function and the lacrimal drainage system can be compromised in patients with facial nerve palsy. The parasympathetic innervation for the

secretomotor function of the lacrimal gland travels with the proximal part of the facial nerve. Thus, facial nerve paralysis can lead to decreased lacrimal gland function and decreased tear production, which can exacerbate exposure keratopathy. Decreased function of the lacrimal gland is particularly common with facial nerve paralysis resulting from acoustic neuromas or other central nervous system tumors. Lacrimal gland function is evaluated using the Schirmer test without anesthesia: less than 10 mm of wetting on a filter paper strip in 5 min can indicate that the proximal facial nerve is damaged. Patients with facial nerve paralysis can also be affected by lower eyelid ectropion and decreased function of the lacrimal pump, leading to epiphora and worsening of corneal exposure.

24.4 Medical Management

The first step in managing facial nerve palsy is supportive treatment to stabilize and protect the cornea. Maintaining the health of the cornea is crucial, as exposure keratopathy and corneal abrasions can result in serious corneal infections, corneal perforation, and even blindness. Artificial tears and lubricating ointments are mainstays of treatment. Preservative-free artificial tear preparations can be employed when frequent administration is required. In more severe cases, moisture chambers that are designed to slow the evaporation of tears from the surface of the eye can be used during sleep. These range from chambers created with a cellophane cover taped over the eyes to customized moisture goggles. Even the simple application of tape can be used to force the palpebral fissure closed during sleeping; however, this needs to be done cautiously as the tape itself can be a source of corneal abrasion if it loosens during sleep. In patients with some remaining lacrimal pump function, punctal plugs can be useful in the treatment of dry eye. When corneal abrasions do occur, bandage contact lenses and pressure patching with ointment can help promote corneal healing.

Botulinum toxin can be useful in treating both exposure keratopathy and facial spasms. If the patient has a good Bell's phenomenon, injection of botulinum toxin into the levator palpebrae superioris at the upper border of the tarsus can produce ptosis and protect the cornea, although the patient's vision is obviously affected and some patients experience diplopia [4]. However, this procedure is a minimally invasive and temporary measure that still allows the cornea to be easily examined.

Another application of botulinum toxin in facial nerve paralysis is the treatment of facial muscle spasms and synkinesis. Hypertonicity of facial muscles often occurs during recovery after facial nerve trauma, and aberrant regeneration of the facial nerve branches can lead to involuntary spasms and involuntary muscle movements. Botulinum toxin can be selectively injected to target the hypertonic muscles and prevent involuntary muscle movements [3, 4].

In cancer patients, facial nerve paralysis can be caused by involvement of the facial nerve by leptomeningeal disease from hematologic malignancies or invasion of the facial nerve from metastases from solid tumors. In these cases, systemic or intrathecal chemotherapy may lead to resolution of facial nerve paralysis.

24.5 Surgical Management

When medical therapy insufficiently addresses the medical and social/emotional consequences of facial nerve paralysis, surgical therapy may be indicated. Occasionally, urgent surgical intervention is indicated for cases of impending ocular surface damage. The soft tissue changes that occur in facial nerve paralysis, such as lower eyelid ectropion, brow ptosis, and facial droop, can be ameliorated with surgical interventions (Fig. 24.2).

24.5.1 Treatment of Lagophthalmos and Exposure Keratopathy

Lagophthalmos and exposure keratopathy can be addressed with surgical closure of the eyelids, known as tarsorrhaphy. As a first step, application of cyanoacrylate glue to the eyelashes or placement of a nonabsorbable suture can create a temporary partial tarsorrhaphy. A temporary tarsorrhaphy may be preferable in cases of recent paralysis that may improve spontaneously over time. A partial tarsorrhaphy preserves some visual function and allows access to the cornea for examination. However, for patients without adequate Bell's phenomenon, a permanent tarsorrhaphy (either partial or complete) is a more effective treatment. A permanent tarsorrhaphy can be performed for established cases of severe palsy. A permanent tarsorrhaphy is accomplished by de-epithelializing the upper and lower eyelid margins and then approximating the upper tarsus and lower tarsus with mattress sutures.

Another option to address lagophthalmos is the placement of an alloplastic weight in the upper eyelid (Fig. 24.3). Gold and platinum are commonly used and aid passively in eyelid closure by exerting a gravitational effect while the levator palpebrae superioris muscle relaxes. The weight is especially useful in patients with exposure keratopathy accompanied by decreased tear production and poor Bell's phenomenon. Preoperative application of tester weights can facilitate selection of the weight that allows the greatest eyelid closure while also allowing adequate eyelid opening in primary gaze. Once the proper weight is selected, it is then secured to the upper border of the tarsus deep to the orbicularis oculi through an eyelid crease incision. Some surgeons choose to wrap the weight in a Dacron (terephthalate fiber) mesh to allow fibrosis around the weight to stabilize its position. Potential complications of alloplastic weight placement are persistent inflammation, extrusion, and eyelid distortion. For temporary facial nerve paralysis, personalized external weights that are secured to the skin of the upper eyelid with adhesive tape are commercially available and can lead to patient satisfaction.

Another alternative for the treatment of lagophthalmos resulting from temporary facial nerve weakness is the injection of hyaluronic acid gel into the upper eyelid [5]. A 30-G needle is used to perform multiple injections of small amounts of hyaluronic acid gel in sites across the length of the upper eyelid into the pre-tarsal and pre-levator aponeurosis regions (Fig. 24.4). The hyaluronic acid gel is layered using

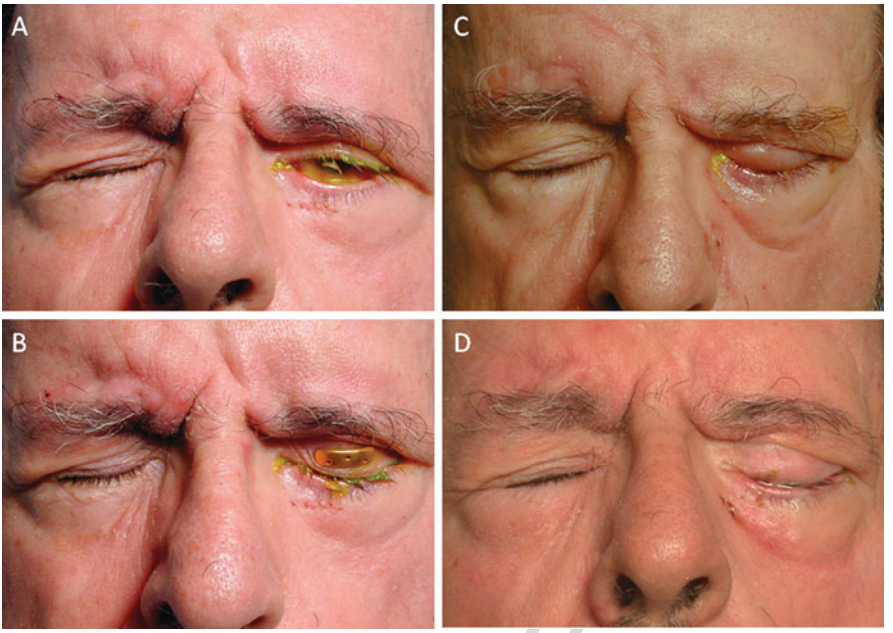

Fig. 24.3 Upper eyelid gold weight in a 60-year-old man with left paralytic lagophthalmos. (**a**) Degree of lagophthalmos before weight placement. (**b**) Fitting the patient with a trial weight preoperatively and having the patient test it by attempting to close the eyelid, which facilitates appropriate weight selection to avoid undercorrection or overcorrection. (**c**) Photograph taken 1 week after insertion of a 1.4-g gold weight into the left upper eyelid and medial canthopexy. (**d**) Photograph taken 1 year after placement of the gold weight, showing stable resolution of lagophthalmos

multiple thread-like injections that are placed deep to the orbicularis oculi muscle. Among the advantages of this technique is that it is temporary and that its effect can be reduced if necessary with hyaluronidase. Hyaluronic acid gel injections can also be used as an adjunct to previous gold weight placement when lagophthalmos persists, and these injections offer an alternative for patients who are poor surgical candidates.

Palpebral springs made of stainless steel can also be used in the treatment of lagophthalmos. These springs can restore a natural-appearing blink with full closure of the eyelid and do not rely on gravity. However, the process of adjusting the spring shape and tension for the individual patient is complex and requires frequent adjustments after the initial surgery. The size and shape of the spring are modified before implantation to follow the eyelid curvature. After exposure of the tarsus via an eyelid crease incision, the tarsal limb of the spring is wrapped in Dacron mesh and then secured to the tarsus, and the fulcrum of the spring is secured to the lateral orbital periosteum. The remaining limb of the spring is then sutured to the periosteum of the superior orbit. Potential complications include extrusion, spring

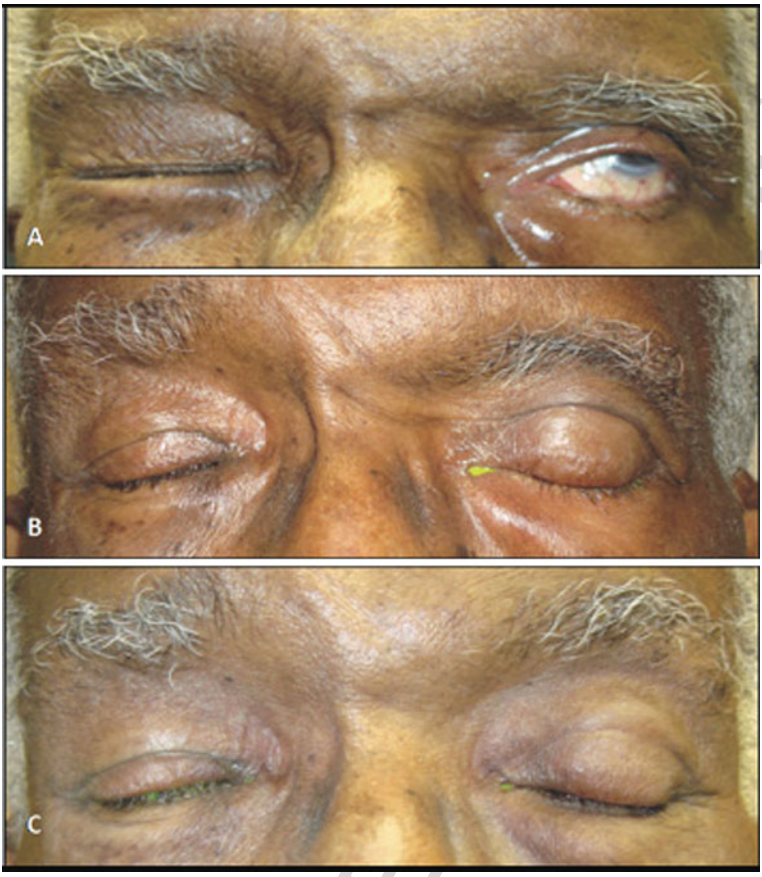

Fig. 24.4 Upper eyelid hyaluronic acid gel weight in a 90-year-old man with left paralytic lagophthalmos due to Bell's palsy. (**a**) Degree of lagophthalmos before injection. (**b**) Attempted closure immediately after injection of hyaluronic acid gel (Juvederm Ultra; Allergan, Inc., Irvine, CA) into the left upper eyelid. (**c**) Attempted closure at 5-month follow-up. Note the resolution of lagophthalmos

malfunction, and the need for frequent readjustment. Because of these problems, very few centers currently use palpebral springs as first-line treatment for surgical rehabilitation of eyelids in facial nerve paralysis.

24.5.2 Treatment of Lower Eyelid Laxity and Ectropion

The midface extends from the lower eyelid margin to the oral commissure. The management of facial nerve palsy in this region is complex owing to the complex interplay of soft tissue gravitational effects and the loss of multiple vectors of muscular pull on the soft tissue. As with surgical options for treatment of lagophthalmos

and exposure keratopathy, surgical therapy for lower eyelid laxity and ectropion begins with procedures designed to protect the ocular surface. Ectropion can be addressed with lateral or medial canthal procedures depending on whether the eyelid laxity is most apparent laterally or medially.

The lateral tarsal strip procedure can be useful in cases of severe laxity or ectropion and involves horizontal eyelid shortening to improve lower eyelid tone and position. The lower eyelid is horizontally shortened and is then reattached more tightly to the periosteum of the lateral orbital rim. However, this procedure is associated with drawbacks, including medial canthal distortion and limited efficacy in patients with prominent globes.

To address medial ectropion or punctal eversion, a medial canthopexy can be performed. The procedure may include excision of skin and/or conjunctiva to advance the lower eyelid superiorly and medially while paying close attention to the integrity of the canalicular system.

When there is laxity of the medial canthal tendon, a static eyelid sling can be created using autologous material, such as temporalis fascia, to further support the lower eyelid. In this procedure, the temporalis fascia is harvested via a posttrichial incision, and the lower eyelid tarsus is exposed along with the lateral and medial canthal tendons via an infralash incision. The fascia is secured to the medial canthal tendon and the tarsus and is then attached to the superficial periosteum of the lateral orbit [4, 6]. This procedure can be combined with the lateral canthal shortening procedures to correct ectropion.

24.5.3 Reanimation of the Midface

Reanimation of the midface in facial nerve paralysis can be addressed by any of several surgical techniques, some of which provide static support for the midface and others of which attempt to restore dynamic movement to the paralyzed face.

24.5.3.1 Static Reanimation

Static midface lifting can correct drooping caused by palsy of the midface muscles. Several different techniques can be utilized, but the goal is to restore the ptotic cheek to a more normal position and also to recruit tissue volume that can support the lower eyelid and ameliorate ectropion.

The classic midface lift is a subperiosteal lift via a lateral canthotomy and transconjunctival lower eyelid incision [7–9]. Another midface repositioning technique is creation of a temporalis fascia sling. In this technique, via a hemicoronal incision, the fascia overlying the temporalis muscle is released and hinged on its inferior–medial border and then brought into the midface as a static sling suspension [6, 10, 11]. A less invasive option is the subperiosteal midface dissection via a temporal and oral incision, along with fixation using a suture, harvested fascia lata, or commercially available midface suspension kit. Another, even less invasive,

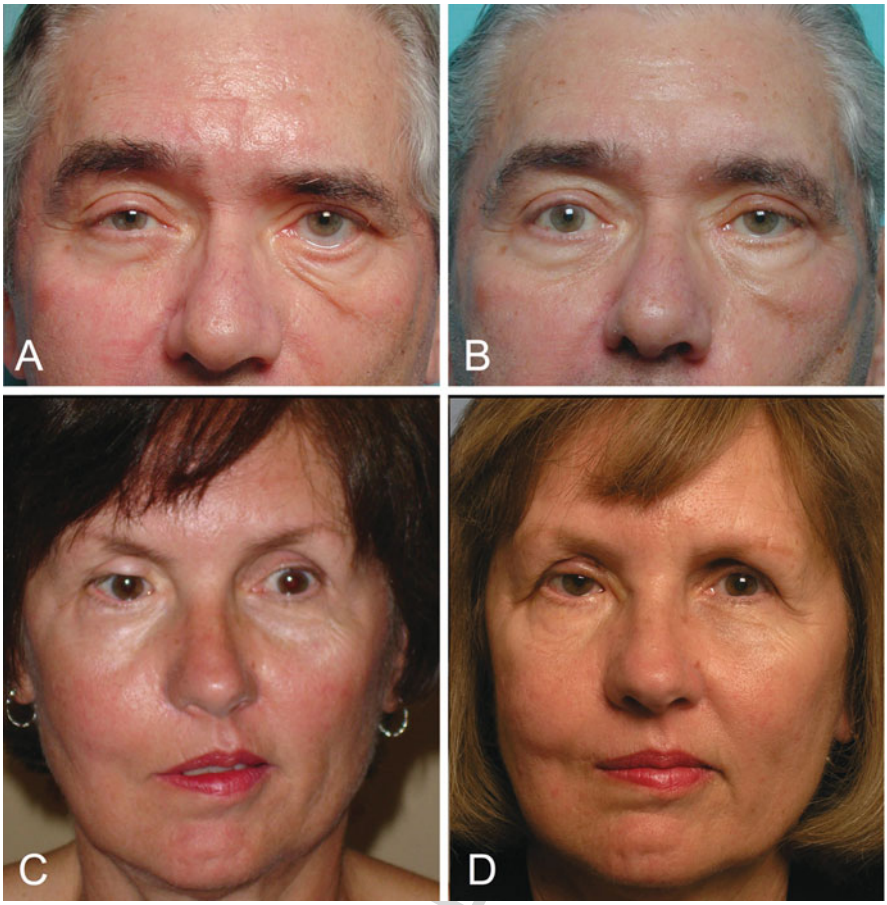

Fig. 24.5 Midface lift. Cable midface resuspension. (**a**) Profound complete left facial nerve palsy with ectropion in a 54-year-old man. (**b**) Photograph taken 1 year after lateral tarsal strip ectropion repair and minimally invasive midface resuspension with multiple 2-0 silk sutures anchored to the deep temporalis fascia. Note resolution of the ectropion. (**c**) Marked right facial nerve palsy in a 46-year-old woman. (**d**) Photograph taken 3 years after minimally invasive midface resuspension with multiple 2-0 silk sutures anchored to the deep temporalis fascia

option is the cable suspension technique, in which long sutures anchored in the midface are suspended to the temporalis fascia [12] (Fig. 24.5).

24.5.3.2 Dynamic Reanimation

Dynamic reanimation of the midface can be performed by muscle transfer and/or nerve grafting. Temporalis muscle transfer can help improve dynamic voluntary facial movement in the paralyzed face and is indicated for patients with chronic facial nerve palsy. Via a hemicoronal incision, the anterior and central portions

of the temporalis muscle and fascia are developed into a muscular flap and dissected inferiorly toward the temporalis insertion [13–18]. The muscle segment is left attached inferiorly, but the flap is divided and sutured to the orbicularis oculi muscle and upper and lower lips, thereby creating a dynamic multiple sling suspension. The patient can then learn to close the eyelid voluntarily by attempting a chewing motion. However, while this procedure is effective in reanimating the lower face, it is also associated with complications including persistent facial droop, facial scarring, and unnatural facial movement.

Several dynamic reanimation techniques involving nerve grafting are available. Following traumatic injury to the facial nerve, such as during tumor resection, direct nerve repair can be attempted. However, during tumor resection, nerve grafting is often required to replace the sacrificed section of the facial nerve. Commonly used donor nerves include the sural nerve and the various cervical nerve branches [19–21]. Cross-face nerve grafting utilizes nerves from the nonparalyzed side of the face to provide innervation to the paralyzed side by incorporating sural nerve grafts [21, 22]. The nerve crossover technique takes advantage of nearby nerves, such as the ipsilateral glossopharyngeal and hypoglossal nerves, to serve as an autograft to the facial nerve [11, 20, 23]. While these procedures can offer some dynamic reanimation to the paralyzed face, their widespread use is prevented by limitations including graft-harvest-induced paralysis, and the complex nerve grafting surgery has the potential to be unsuccessful or result in uncoordinated movements. It also takes at least 6–12 months for the beneficial effect of nerve grafting to become apparent and, in general, nerve grafting is more effective for the lower facial branches of the facial nerve than for the upper facial (periocular) branches.

24.5.4 Options for Correction of Brow Ptosis

Brow ptosis in the paralyzed face can lead to mechanical ptosis, extreme dermatochalasis, and obstruction of the superior visual field. The direct-incision brow lift is the most effective and simplest surgical rehabilitation option. The amount of lift needed is measured with the patient sitting upright with the brows relaxed, and then the eyebrow is elevated to the desired position. The skin and subcutaneous tissue are then dissected to the level of the frontalis muscle and excised using an incision superior to the lateral two-thirds of the eyebrow. While this procedure is very effective, its disadvantages include the occasional unappealing scar and rare sensory nerve damage.

A more cosmetically sensitive approach to the brow lift utilizes endoscopic techniques via an incision hidden in the hairline and is associated with a lower rate of postoperative numbness and paresthesia [24, 25]. The dissection proceeds caudally in either a subperiosteal or preperiosteal plane. The corrugator and procerus muscles may be resected to reduce the action of brow depression. The elevated brow is then fixed posterior to the hairline with any one of various absorbable or nonabsorbable fixation devices. Potential complications of the endoscopic brow lift for elevating a

paralytic brow include malposition of the brow and alopecia at the location of fixation; in addition, the endoscopic brow lift can result in a less robust lift than that achieved with the direct technique. In non-facial nerve palsy cases, the most serious complication of this procedure is damage to the temporal branch of the facial nerve.

24.5.5 Additional Procedures for Management of Facial Droop

Additional procedures for management of facial droop include rhytidectomy, lateral oral commissure lift, and lateral alar lift (Fig. 24.2). Rhytidectomy may be performed in conjunction with a midface lift and periocular reconstruction. Options for rhytidectomy via a preauricular incision are the superficial musculo-aponeurotic system lift and the deep plane lift. In patients with facial nerve paralysis, the usual concerns about damage to the facial nerve during the deep plane facelift do not apply, allowing for a more aggressive approach and making the deep plane rhytidectomy the preferred approach for resuspending the midface and lower face in such patients. Drooping of the corner of the mouth can be addressed with a lateral oral commissure lift, in which slings are used to suspend the orbicularis oris to either the orbital rim or the zygomatic arch. A simple variation is to perform a direct excision of skin and subcutaneous tissue along the lateral superior vermillion border; the open approach allows direct plication of the orbicularis oris. A lateral alar lift can correct collapse of the internal nasal valve; a direct curvilinear incision and skin resection elevate the ala and open the nasal vestibule.

24.6 Special Circumstances in Cancer Patients with Facial Nerve Paralysis

Many patients with head and neck cancer who undergo a parotidectomy develop facial nerve paralysis either due to direct mechanical compression of the nerve by tumor or due to the necessary sacrifice of the facial nerve during cancer-ablative surgery. In the majority of these patients, postoperative adjuvant radiation therapy is planned within 4–6 weeks after head and neck surgery. In these patients, we prefer not to perform periocular surgical rehabilitation during the primary ablative procedure as this type of "one-size-fits-all" approach would lead to less than ideal outcomes [1]. Instead, we prefer to evaluate the patient after the ablative head and neck surgery to assess the facial tone, the size of gold weight needed, and the degree of paralytic ectropion and lagophthalmos before we plan surgical rehabilitation of the periocular soft tissues. Periocular surgery can be done either during the week or two after the parotidectomy or a few weeks after radiation therapy is completed. An important consideration in planning the timing of periocular surgery is that although the radiation field in most cases does not include the periocular soft tissues, the mask used during daily head and neck irradiation can be uncomfortable in the immediate postoperative period after periocular surgery.

Another potential special circumstance in cancer patients is chemotherapy-induced pancytopenia due to recent chemotherapy. In patients with such pancytopenia, surgery should be delayed until hematologic parameters have normalized. Prophylactic use of antibiotics in the perioperative period is also appropriate in the majority of cancer patients undergoing surgical rehabilitation for periocular manifestations of facial nerve paralysis.

24.7 Conclusion

Management of facial nerve paralysis in cancer patients poses complex challenges. Spontaneous improvement of facial nerve paralysis in cancer patients is uncommon as the nerve is often sacrificed during tumor resection. However, function may be regained if the nerve is affected only indirectly by compression or is only traumatized during ablative surgery or due to mass effect. The prevention of ocular surface morbidity is of paramount importance in facial nerve paralysis. Medical therapy is the first step, but often surgical intervention is indicated. Surgical procedures can help minimize morbidity to ocular tissues, provide static support for ptotic facial tissues, and sometimes restore dynamic voluntary movements. Furthermore, improvements in both facial function and symmetry from surgery may mitigate the psychological burden to patients living with facial nerve paralysis. Future developments in static and dynamic reanimation will help to further address the challenges of facial nerve paralysis. The extent and timing of rehabilitative surgery in cancer patients with facial nerve paralysis should be individualized and depend on many factors, including age, facial muscle tone, timing of adjuvant radiation therapy and chemotherapy, and long-term prognosis.

References

1. Golio D, De Martelaere S, Anderson J, et al. Outcomes of periocular reconstruction for facial nerve paralysis in cancer patients. Plast Reconstr Surg 2007;119:1233–7.
2. Kahn JB, Gliklich RE, Boyev KP, et al. Validation of a patient-graded instrument for facial nerve paralysis: the FaCE scale. Laryngoscope 2001;111:387–98.
3. Mehta RP, Hadlock TA. Botulinum toxin and quality of life in patients with facial paralysis. Arch Facial Plast Surg 2008;10:84–7.
4. Lee V, Currie Z, Collin JR. Ophthalmic management of facial nerve palsy. Eye 2004;18:1225–34.
5. Mancini R, Taban M, Lowinger A, et al. The use of hyaluronic acid gel in the management of paralytic lagophthalmos: the hyaluronic acid gel "gold weight." Ophthal Plast Reconstr Surg. In press.
6. Rose EH. Autogenous fascia lata grafts: clinical applications in reanimation of the totally or partially paralyzed face. Plast Reconstr Surg 2005;116:20–32.
7. Edelstein C, Balch K, Shorr N, et al. The transeyelid subperiosteal midface-lift in the unhappy postblepharoplasty patient. Semin Ophthalmol 1998;13:107–14.
8. Paul MD, Calvert JW, Evans GR. The evolution of the midface lift in aesthetic plastic surgery. Plast Reconstr Surg 2006;117:1809–27.

AQ2

9. Baylis HI, Goldberg RA, Shorr N. The deep plane facelift: a 20-year evolution of technique. Ophthalmology 2000;107:490–5.
10. Neuhaus RW, Shorr N. Use of temporal fascia and muscle as an autograft. Arch Ophthalmol 1983;101:262–4.
11. Rosson GD, Redett RJ. Facial palsy: anatomy, etiology, grading, and surgical treatment. J Reconstr Microsurg 2008;24:379–89.
12. Ugurbas SH, Goldberg RA, McCann JD, et al. Suture midface suspension. Head Face Med 2006;2:35–9.
13. Cheney ML, McKenna MJ, Megerian CA, et al. Early temporalis muscle transposition for the management of facial paralysis. Laryngoscope 1995;105(9 pt 1):993–1000.
14. Frey M, Giovanoli P, Tzou CJ, et al. Dynamic reconstruction of eye closure by muscle transposition or functional muscle transplantation in facial palsy. Plast Reconstr Surg 2004;114:865–75.
15. May M, Drucker C. Temporalis muscle for facial reanimation. A 13-year experience with 224 procedures. Arch Otolaryngol Head Neck Surg 1993;119:378–82.
16. Tate JR, Tollefson TT. Advances in facial reanimation. Curr Opin Otolaryngol Head Neck Surg 2006;14:242–8.
17. Yoleri L, Songur E. Modified temporalis muscle transfer for paralytic eyelids. Ann Plast Surg 1999;43:598–605.
18. Boahene KD. Dynamic muscle transfer in facial reanimation. Facial Plast Surg 2008;24: 204–10.
19. Lee EI, Hurvitz KA, Evans GR, et al. Cross-facial nerve graft: past and present. J Plast Reconstr Aesthet Surg 2008;61:250–6.
20. Shah SB, Jackler RK. Facial nerve surgery in the 19th and early 20th centuries: the evolution from crossover anastomosis to direct nerve repair. Am J Otol 1998;19:236–45.
21. Terzis JK, Konofaos P. Nerve transfers in facial palsy. Facial Plast Surg 2008;24:177–93.
22. Frey M, Giovanoli P, Michaelidou M. Functional upgrading of partially recovered facial palsy by cross-face nerve grafting with distal end-to-side neurorrhaphy. Plast Reconstr Surg 2006;117:597–608.
23. Atlas MD, Lowinger DS. A new technique for hypoglossal-facial nerve repair. Laryngoscope 1997;107:984–91.
24. De Cordier BC, de la Torre JI, Al-Hakeem MS, et al. Endoscopic forehead lift: review of technique, cases, and complications. Plast Reconstr Surg 2002;110:1558–68.
25. Steinsapir KD, Shorr N, Hoenig J, et al. The endoscopic forehead lift. Ophthal Plast Reconstr Surg 1998;14:107–18.

Chapter 24

Q. No.	Query
AQ1	Please check the edits made to the sentence “a direct curvilinear incision and skin resection...” for clarity.
AQ2	Please update reference [5].