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REVIEW

Re-Animation and Rehabilitation of the Paralyzed Face in Head and Neck Cancer Patients

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Facial nerve paralysis can occasionally result from the treatment of head and neck cancer. The treatment of paralysis is patient specific, and requires an assessment of the remaining nerve segments, musculature, functional deficits, anticipated recovery, and patient factors. When feasible, reinnervation of the remaining musculature can provide the most natural outcome. However, the complex and topographic nature of facial innervation often prevents complete and meaningful movement. In these instances, a wide variety of procedures can be used to combat the functional and cosmetic sequella of facial paralysis. Clin. Anat. 25:99–107, 2012.

Key words: head and neck cancer; facial paralysis; facial nerve; facial reanimation

INTRODUCTION

Facial nerve paralysis can be a serious consequence of head and neck cancer and its treatment. In addition to dealing with the reality of having cancer, these patients must deal with the significant emotional, aesthetic, and functional consequences from loss of facial nerve function. With good oncologic outcomes, ongoing treatment focuses on minimizing the disability from the paralysis (Fig. 1).

Paralysis of the facial nerve may occur due to a number of conditions. The most common reasons for paralysis are infectious and traumatic causes. Neoplasms are less common, but can involve the facial nerve anywhere from the brainstem to the peripheral nerve endings. Masses in the cerebopontine angle, including acoustic neuromas and meningiomas, can put the nerve at risk during surgical resection of these lesions. The course of the nerve through the temporal bone places the nerve at risk from malignancies that involve the temporal bone. This includes squamous cell carcinomas, glomus tumors, and sarcomas. Once the nerve exits the stylomastoid foramen, its entry into the substance of the parotid gland make it susceptible to tumors of the gland itself or to metastatic lesions involving the parotid lymph nodes (Fig. 2).

Anatomical variations in the position of the facial nerve make it more vulnerable to injury during surgical procedures. There are extensive reviews on the anatomy of the facial nerve available in the literature (May and Schaitkin, 2000). Variations of the nerve position within the temporal bone not identified preoperatively can result in injury during otologic surgery (Fowler, 1961). Upon exiting the stylomastoid foramen, surgeons generally utilize consistent landmarks to identify the facial nerve and avoid injury. These landmarks include the tympanomastoid suture line, tragal pointer, and the posterior belly of the digastric muscle (Harell et al., 1996; Rea et al., 2010). Variations also exist in the branching patterns of the facial nerve (Davis et al., 1956). Typically, branching patterns are not as relevant to oncologic surgeons who are following standard techniques for identifying and working around the

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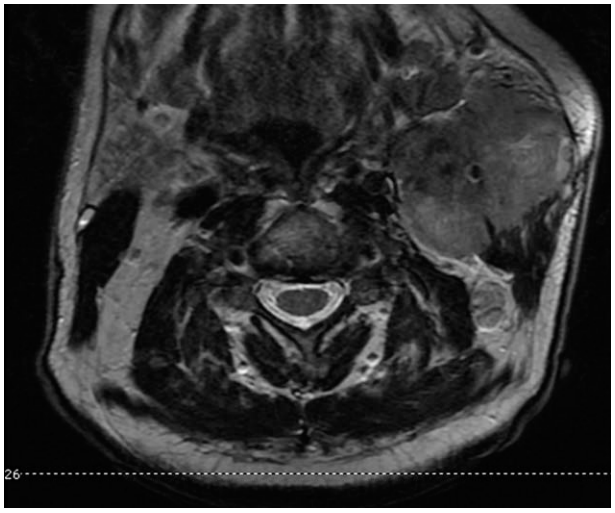

Fig. 1. This patient photo is after right radical parotidectomy, total auriculectomy, radial forearm free flap reconstruction, and now pre-op for facial rehabilitation.

facial nerve, but may be relevant to reconstructive surgeons attempting to identify nerve branches for reinnervation.

Clinical signs of neoplastic involvement of the facial nerve usually differ from other causes of facial nerve weakness. Typically patients report a slow, progressive loss of function with no improvement over time. Pain is a worrisome sign. Extratemporal lesions may present with a palpable mass, while intratemporal lesions may present with additional cranial nerve findings, such as hearing loss, tinnitus, imbalance, and facial numbness.

The treatment of facial nerve paralysis secondary to head and neck cancer depends on a number of variables. The integrity of the nerve at the conclusion of surgery and the possibility for spontaneous recovery can determine the timing of any potential intervention. With disruption of the nerve during oncologic surgery, reinnervation of the musculature may be possible, either through primary repair of the nerve, cable grafting, or by connecting the nerve to a different motor source. In some instances, reinnervation is not feasible or oncologically sound. In both cases, reanimation of the paralyzed face can provide a significant improvement to a patient's quality of life.

TREATMENT OF THE NERVE

The most basic form of reconstruction is direct repair of the disrupted facial nerve. With disruption of the nerve during surgery, first consideration is given to whether or not reinnervation of the distal musculature can be established. This may occur by reestablishing continuity of the severed nerve or by finding a donor motor nerve to provide input.

The best chance for return of function for a severed nerve is primary repair of the nerve (Yarbrough et al., 1993). The most critical factor for a successful outcome is establishing a tension-free repair. Typically three 8–0 or 9–0 non-absorbable sutures are carefully placed into the epineurium to reattach the nerve endings. Nerve gaps up to 2 cm may still be amenable to primary repair, although gaps greater than a few millimeters require significant nerve mobilization. Nerve mobilization is performed by tracing the nerve into the temporal bone via a mastoidectomy. Unfortunately, tumor extirpation typically involves long segments of the facial nerve making primary repair frequently infeasible.

For gaps greater than 2 cm, cable grafting of the nerve can be performed (Fig. 3). This requires that both ends of the nerve be easily accessible for microsurgery, which may not be possible if the nerve has minimal distal branches remaining or if the proximal stump is inaccessible in the temporal bone. Cable grafting is performed in a similar fashion to primary repair. There are many donor nerves described (Table 1). The most accessible is the great auricular nerve, although surgery or oncologic concerns may preclude its use. In select patients, the ansa cervicalis has sufficient size and branching pattern to be an

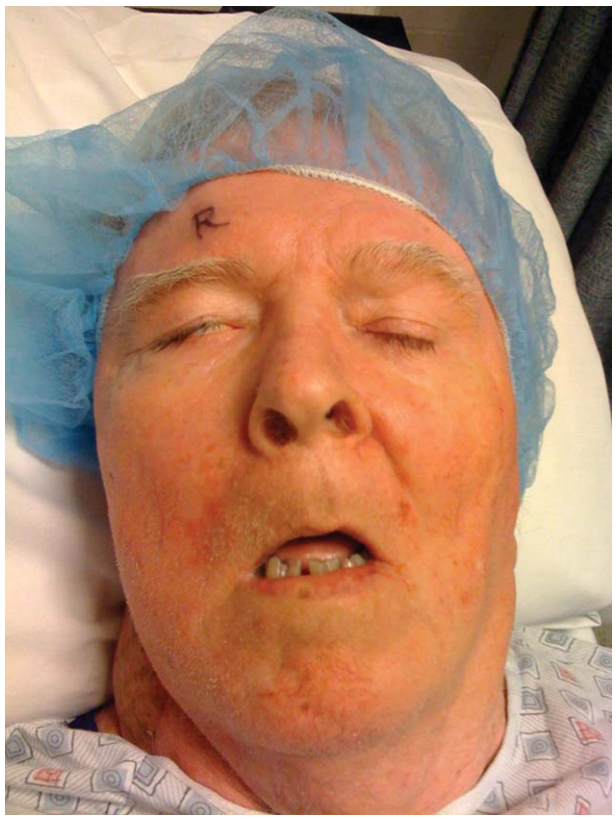

Fig. 2. This T1-weighted MRI scan shows a large malignant mass in the parotid gland encompassing the expected location of the facial nerve. Removal of this tumor required excision of the main trunk of the nerve and proximal branches. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

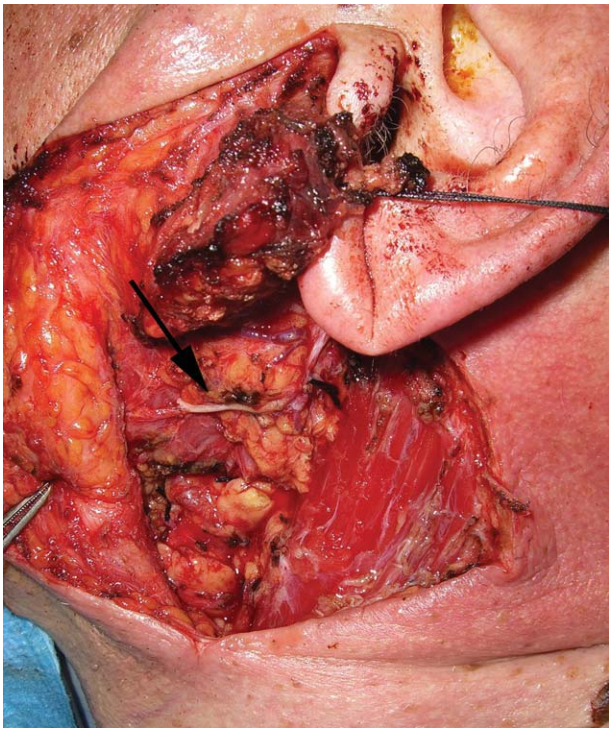

Fig. 3. This image shows a nerve cable graft (black arrow) that reconnects the inferior division of the facial nerve. This segment of the nerve was sacrificed during tumor extirpation. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

excellent option and is readily available during neck dissection. Cutaneous sensory nerves from the limbs such as the sural nerve or the medial or lateral antebrachial cutaneous nerves can also be easily harvested for this purpose, however both require a second donor site.

If the proximal end of the resected facial nerve is not accessible, it is possible to use a different motor nerve to provide neural input. These procedures may be performed at the time of initial surgery or after a delayed interval. Cross-facial nerve grafting uses a donor nerve connected to a branch of the contralateral facial nerve to drive the ipsilateral side. Hypoglossal-facial jump grafts connect a portion of the ipsilateral hypoglossal nerve to the residual distal ends of the resected facial nerve (Pensak et al., 1986; Asaoka et al., 1999). This motor input is more helpful in providing tone than true functional movement. The downsides of tongue weakness and errant facial movements make this less than ideal. The motor nerve to the masseter muscle can also be connected to the distal facial nerve.

Whether or not these procedures are a viable option to establishing innervation to the remaining facial musculature is very patient dependent. These procedures do not always produce adequate functional outcomes and can leave the patient with ongoing disability. In these instances or when these procedures are not performed, the next step in treatment involves facial reanimation or rehabilitation.

REANIMATION AND REHABILITATION

Treatment of facial nerve paralysis has two basic forms; re-animation and rehabilitation. The goal of reanimation is to restore meaningful and volitional movement to the paralyzed face to restore function. Rehabilitation consists of directed surgical efforts to minimize or reverse the negative functional consequences of the paralysis without the restoration of movement. Since true purposeful reanimation of the entire facial musculature is not possible without topographic neural input, these procedures are designed to compensate for the functional loss and provide an improved aesthetic outcome. It is helpful to divide the face into three horizontal zones to help separate out the regions that are typically addressed during facial reanimation and rehabilitative surgery (Fig. 4).

The decision to perform reanimation and rehabilitative procedures may be done at the time of initial surgery or delayed. Several considerations such as status of the cancer, life expectancy, need for postoperative radiation, monitoring for disease recurrence, and likelihood of nerve graft success affect the decision of which procedures to do and when. In some instances, only a subset of nerve branches will be lost.

Upper Face

Loss of innervation of the frontalis muscle causes ptosis of the brow. The degree of ptosis and resulting functional impairment and asymmetry varies

TABLE 1. Potential Donor Nerves for Cable Grafting

Donor nerve	Advantages	Disadvantages
Great auricular nerve	Easy accessibility	Non-branching pattern Short length May be contraindicated for oncological reasons
Ansa cervicalis	Available with neck dissection	Small caliber
Sural nerve	Long length Good caliber	Requires second donor site Lateral foot numbness
Medial/Lateral antebrachial Cutaneous nerve	Branching pattern Long length	Requires second donor site

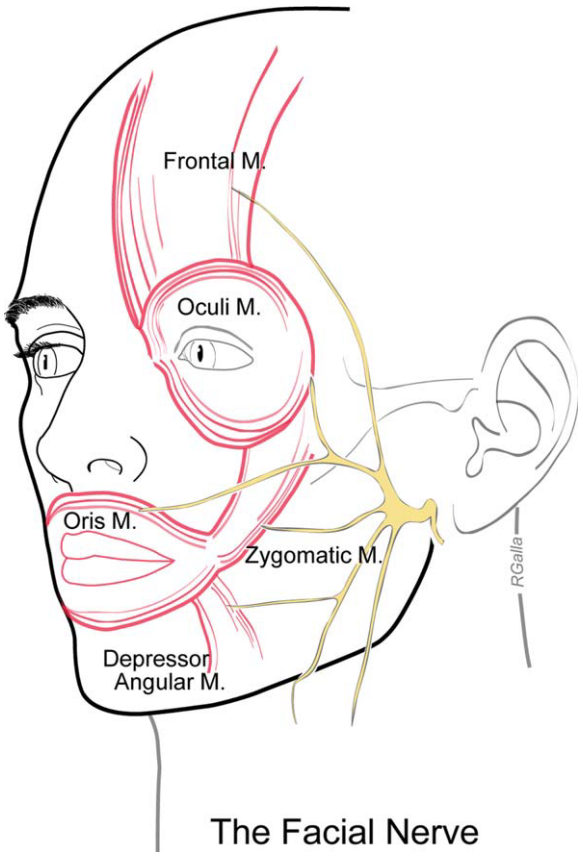

Fig. 4. Facial nerve and target musculature. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

between patients, and is predominantly dependent on age and skin laxity. In addition to the cosmetic appearance of brow ptosis, the overhanging tissue can impinge on the eye causing decreased field of vision.

Rehabilitation centers on elevation of the brow and there are many methods described for this, most having their origins in cosmetic surgery. The coronal, pretrichal, midforehead, and direct brow lift all attempt to elevate the brow by removing a strip of excess tissue which allows the brow to advance superiorly. These approaches primarily differ in the location of the incisions. Endoscopic approaches attempt to elevate the brow through a minimally invasive approach and without skin excision. Each of these methods has advantages and disadvantages that are well described in the cosmetic literature.

The method most frequently advocated by reconstructive surgeons for facial nerve paralysis is a direct brow lift (Booth et al., 2004). This allows for excellent unilateral control of brow height. Disadvantages include a facial incision that may not be concealed in patients with thin eyebrows as well as loss of velous hair superior to the brow.

Before surgery, the patient is best assessed in the upright position to determine the extent of brow elevation necessary. With a direct brow lift, the skin excision takes the form of a crescent moon (Fig. 5). The inferior incision parallels the upper aspect of the eyebrow at its margin so as to hide the final scar within this hair. The superior limb creates a gentle arc. The exact shape of the excision is designed to create symmetry between the two sides, therefore, the final shape will vary between patients. Care is taken to not make the angles at either end of the excision >20–30° to avoid creating a dog-ear deformity.

In the operating room, the skin incisions previously marked are made down to the subcutaneous layer. This skin is then excised staying superior to the frontalis muscle and SMAS layer. Inferiorly, the superior aspect of the orbicularis oculi muscle may be seen. This layer can help with maintaining an elevated brow position and take tension off the closure by placing two sutures into the muscle and tacking it superiorly to the periosteum of the frontal bone. The skin is then closed in a layered fashion by placing absorbable sutures in the subdermal layer, and a subcuticular stitch for final closure (Fig. 6).

Rehabilitative treatment of the eyelids is required secondary to loss of function of the orbicularis oculi muscle. With loss of the only sphincter muscle of the eye, the primary concern becomes corneal exposure

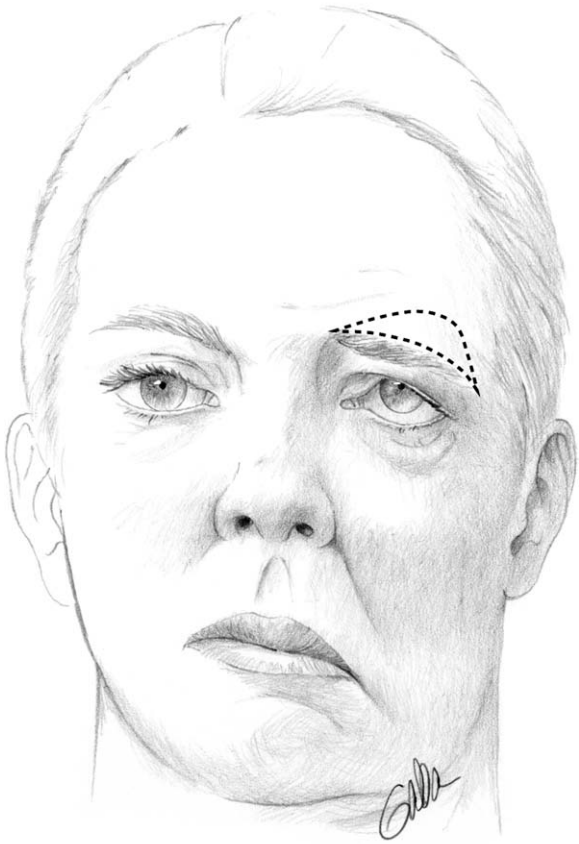

Fig. 5. The direct brow lift incision is designed in a half-moon shape with the inferior limb approximating the superior aspect of the brow. This approach gives excellent control of brow height.

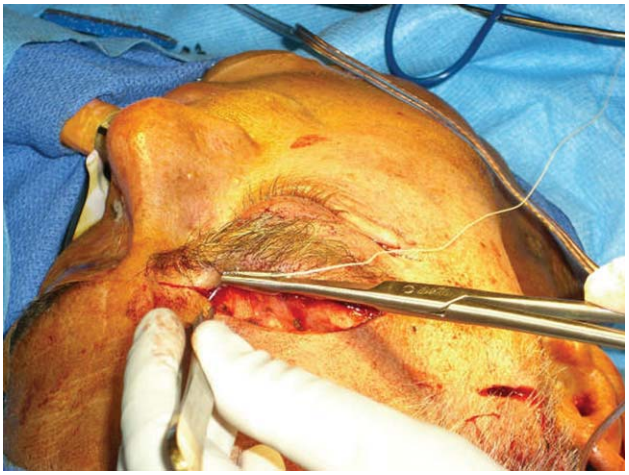

Fig. 6. This intraoperative photo shows buried stitches being placed to close the direct brow lift incision after skin removal. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

and maintenance of a tear film (Kinney et al., 2000). Exposure keratitis can lead to pain and potentially long-term visual impairment. This is especially problematic when there is also dysfunction of the Vth cranial nerve. Delayed or absent eye closure is obvious to an observer and can be aesthetically concerning.

Upper lid closure can be assisted in a static fashion, using a tarsorraphy stitch, or dynamically. Although gravity is typically the enemy in facial nerve paralysis, it can be used to aid upper eyelid closure. Since control of eye opening remains secondary to the uninterrupted innervation of the levator muscle by the cranial nerve III, placement of a weight in the upper eyelid helps bring the lower lid closed once the opposing force of the levator muscle

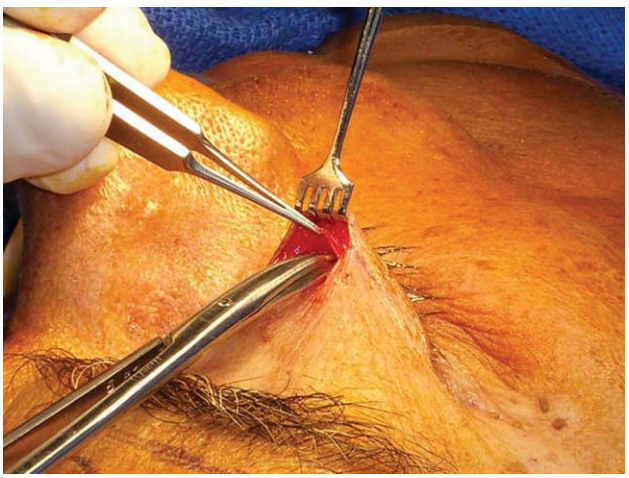

Fig. 7. The pocket for gold weight placement is developed between the tarsal portion of the orbicularis oculi muscle and the tarsal plate. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

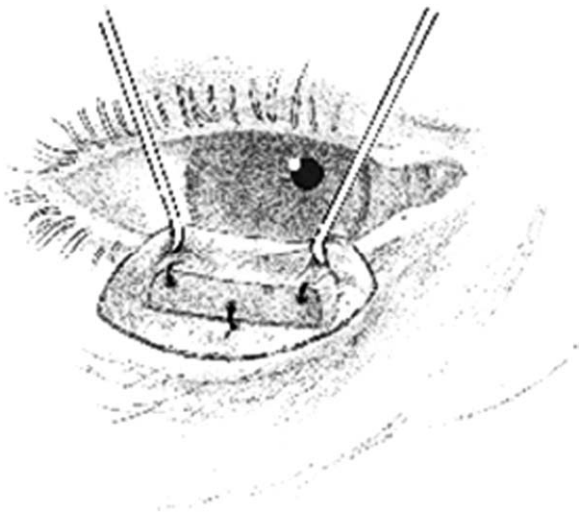

Fig. 8. The placement of the gold weight on the inferior-most aspect of the tarsal plate is secured with 6–0 sutures.

is relaxed. These weights have traditionally been made from gold, although platinum is becoming more popular given its lower profile and lower allergenicity (Berghaus et al., 2003).

Before placement of the weight, the appropriately sized implant is measured on the patient. This is done in clinic by having the patient in an upright position and taping weights to the upper eyelid. The ideal weight is the minimum size that allows for passive eye closure. In the case when weight placement is done at the time of resection, the weight is estimated, typically 1.0 gm for females, and 1.2–1.4 for males. Minor oversizing of the implant is acceptable in most cases since the levator muscle can hypertrophy to a small degree and account for the added weight on opening.

The most frequent approach for weight placement is via an incision at the supratarsal crease, although methods of retrograde placement using an incision at the ciliary margin have also been described (Kao and Moe, 2004). After injecting local anesthetic, an incision is made in the supratarsal crease, which sits approximately at the superior margin of the tarsus. The incision should be through the thin skin and the orbicularis oculi muscle. Care must be taken to not extend the incision deeper and disrupt the levator attachment to the tarsus. Once through the orbicularis oculi, a small skin hook is used to retract the skin and muscle superiorly while dissection is carried out along the tarsal plate (Fig. 7). The skin and orbicularis muscle is elevated off the anterior aspect of the tarsal plate almost to the lid margin to create a pocket in which the gold or platinum weight will be inserted.

The center of the pocket should be slightly medial to the pupil. Once an adequate pocket is created, the weight is inserted. It is secured into position by placing 6–0 silk sutures to the underlying tarsal plate (Fig. 8). Care must be taken to not pass the sutures through the tarsal plate and underlying conjunctiva. After the weight is secured, the incision can be

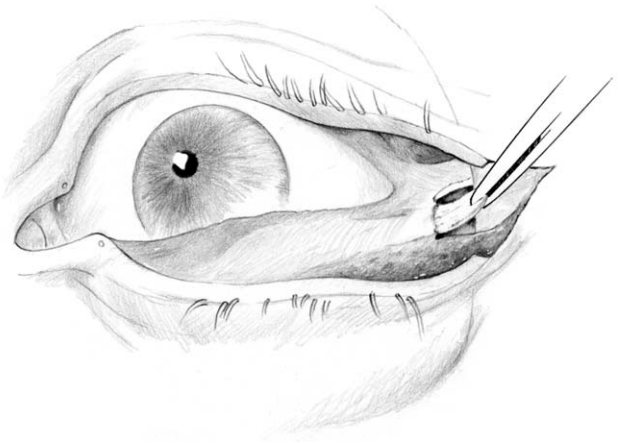

Fig. 9. With the lower lid disconnected laterally, the anterior and posterior lamella of the canthal tendon are removed leaving a strip of tendon which can be shortened and reattached.

closed by approximating the muscle layer and then using a 6–0 fast-absorbing suture to close the skin.

The lower lid faces some unique problems over the upper lid. With the loss of tone in the orbicularis muscle, the weight of the lid and the midfacial tissues pulls inferiorly on the lower lid. Any degree of laxity in the canthal system will subsequently lead to the lid pulling away from the eye, initially beginning with scleral show and progressing to frank ectropion. Over time the canthal tendons stretch further worsening the problem. With loss of apposition of the lid to the globe, increased corneal exposure, loss of the tear film, and epiphora further contribute to the drying of the corneal surface.

Reconstruction of the lower lid has generally focused on tightening the lateral canthal tendon, thus helping reestablish lid contact with the globe and elevating the lid margin to a more normal position (Fedok and Ferraro, 2000). This is most commonly achieved by a lateral tarsal strip procedure (Anderson and Gordy, 1979). In the lateral tarsal strip procedure the lateral canthal tendon is isolated, shortened, and refixed. Attempts have been made to use innervated muscle flaps to provide dynamic rehabilitation of the eye closure, however, these are frequently not successful (Frey et al., 2004).

The lateral tarsal strip procedure begins by making a 1 cm incision from the lateral canthus laterally over the bony orbital rim. The incision is made down through the skin and orbicularis oculi muscle. At this point, the orbital septum is encountered, and the insertion of the septum into the orbital rim forming the arcus marginalis is identified. Dividing through this layer, the lateral canthal tendon is identified attaching to the inside surface of the orbital rim at Whitnall's tubercle. The tendon can be identified by grasping the lower lid gently and retracting medially. The tendon is felt as a band deep to the plane of the orbital rim. Once the inferior limb of the lateral canthal tendon is identified, it can be divided (Gioia et al., 1987). Division of the tendon frees the lower lid laterally, and allows for it to be rolled outward.

The lateral canthal tendon is then pulled laterally as the lower lid is assessed to determine the degree of shortening that must be performed. Overshortening of the tendon can result in the lower lid not being able to conform to the convexity of the globe, therefore causing the lower lid margin to slip inferiorly and rest beneath the curvature of the globe. Once the optimum tendon length is established, the skin and muscle are removed from the anterior surface of the lid, and conjunctiva is removed from the posterior surface (Fig. 9). This allows for isolation of the tendon. The tendon is the refixed laterally with a non-absorbable suture. Placement of this suture is important to reestablishing appropriate positioning of the lid. In order for the lid to fully oppose the globe, the suture needs to be placed inside the orbital rim approximately in the area of Whitnall's tubercle. Posterior and superior attachment of the tendon allows for the lid to maintain contact with the globe laterally (Fig. 10). After the tendon is refixed, the muscle and skin layers are then closed.

Mid Face

Addressing the midface in facial nerve paralysis includes both the cheek and the nasal opening. Ptosis of the midfacial tissue not only exacerbates lower lid dysfunction, but also poses a significant aesthetic challenge. Rehabilitation centers on elevation of these tissues and is challenging primarily due to the lack of strong supporting structures to suspend an anchor from. Treatment of this area with a midface lift can be performed by suspending the periosteum and midfacial tissues to the zygoma or inferior orbital rim.

Ptosis of the midface also causes impingement on the nasal valve, leading to nasal obstruction. While elevation of the midface tissue may improve this area, suture suspension of the nasal valve may need to be performed. This technique is performed by using a suture to hook the upper or lower lateral

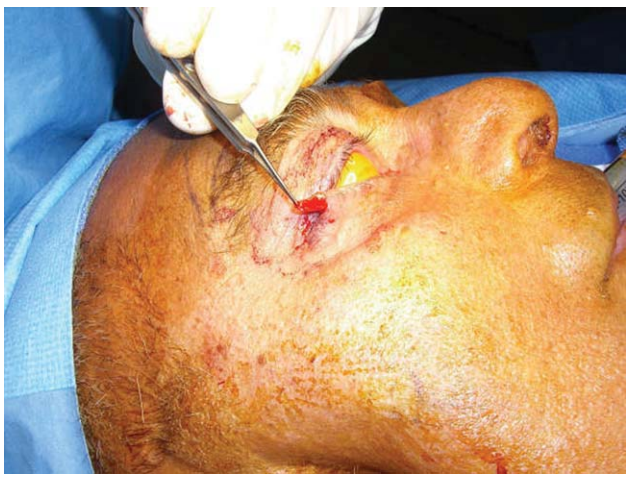

Fig. 10. After the lateral tarsal strip is fashioned, it is reaffixed in a superior and posterior direction to allow for elevation and tightening of the lower lid, and better apposition to the globe. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

cartilages and affixing them to a bony anchor at a vector that allows for opening of the valve (Rizvi and Gauthier, 2003).

Lower Face

Treatment of the lower face is centered around the oral stoma and includes functions of the orbicularis muscle and the muscles that act on the mouth. One the most significant aesthetic and emotional disabilities related to facial nerve paralysis is loss of facial expression related to the oral cavity—primarily the smile (Seiff et al., 1989; Cross et al., 2000). The loss of ability to smile is a devastating consequence. Treatment of the oral stoma functionally is related to maintenance of oral competence.

Addressing the smile complex or superior stoma can be done in a static or dynamic fashion. The choice of procedure depends on the specific clinical scenario. Rehabilitation with static treatment allows for elevation of the oral commissure. These procedures are simpler than dynamic procedures, and may be especially useful with nerve grafting and anticipated return of some nerve function. Multiple materials have been described for static suspension. Autologous fascia is an ideal substance, although the harvest of which may require a second surgical site. Allografts are also an option, including products such as Alloderm and Durepair. Finally, synthetic products such as PTFE (Teflon) sheets do not reabsorb like allografts, however these alloplasts are vulnerable to infection.

The goal of static suspension is to elevate the oral commissure to a position that is a balance between the resting and smiling positions of the contralateral side. Technique for static suspension is similar regardless of what product is used. The vector of pull is designed to extend from the oral commissure to the body of the zygoma (Fig. 11). The procedure is begun by elevating a skin flap in the preauricular area just above the level of the SMAS. This flap may have been previously elevated for tumor extirpation or for nerve grafting. The skin flap must be elevated to adequately expose the malar prominence and zygoma, which will serve as the anchor point for the sling. A subcutaneous tunnel towards the oral commissure can then be made.

At the oral commissure, there are multiple ways in which to insert the sling. The skin flap elevation can proceed to the orbicularis oris muscle and the modiolus. This muscle then becomes the insertion point for the sling. Alternatively, a separate incision can be made in the vermillion border or in the melolabial crease. Exposure of the underlying muscle through this method can provide different anchor points as well. Some authors describe using extensions which connect the sling towards the midline of the lip. Once the distal end of the sling is secured, the proximal portion is elevated in the supero-lateral direction towards the malar prominence. The degree of excursion and tension placed on the sling will vary between patients. In general, overcorrection is recommended due to the effects of tissue relaxation with time (Figs. 12 and 13). Once the tension is established, the proximal sling can be fixed to the zygoma using two 1.3 mm titanium screws and small miniplate, allowing for stable anchoring of the sling superiorly in a broad fashion that should not tear through.

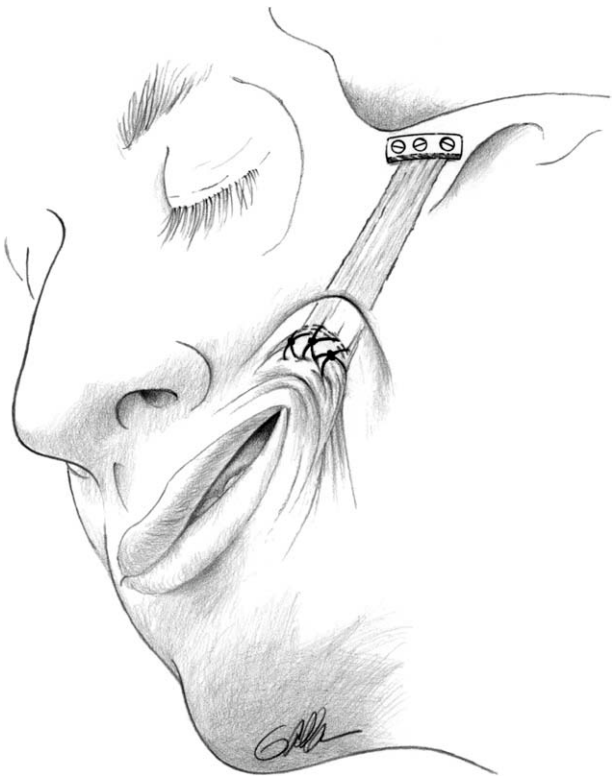

Fig. 11. The corner of the mouth is elevated superiorly by the facial sling and affixed to the zygoma with a mini-plate.

Reanimation with dynamic suspension allows for movement of the oral commissure and is primarily designed to allow patients to smile, in addition to providing some degree of static suspension. It can be performed using regional or free tissue transfer. In regional tissue transfer, the muscles of mastication are transposed to allow for movement of the oral commissure. The temporalis muscle and the masseter muscle are both candidates. The temporalis transfer can be performed by using a section of muscle detached from the temporal fossa and taken over the zygomatic arch. Alternatively, the temporalis tendon can be detached from its insertion on the coronoid process and brought forward through the buccal space (Croxson et al., 2000). The attachment of the muscle or tendon is similar to that described for the static sling technique.

Free tissue transfer for reanimation is an evolving area. The muscle most commonly used is the gracilis, although the pectoralis minor, latissimus, serratus, and rectus femoris muscles have also been described (Terzis and Noah, 1997). Before transfer of the muscle, adequate neural input must be planned. This may come from either the contralateral facial nerve via a cross facial nerve graft, or from the nerve to the ipsilateral masseter muscle. Once there

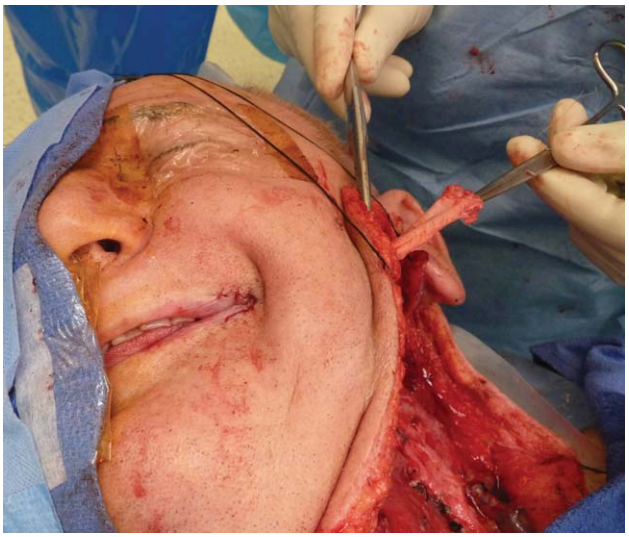

Fig. 12. Tightening of the facial sling intra-operatively is done with the intent of over correction of the corner of the mouth to account for anticipated relaxation of tissues. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

is viable nerve input to the free tissue transfer, the muscle is transplanted and the neural connection is established (Fig. 14).

Addressing the inferior oral stoma includes dealing with the loss of function of the orbicularis oris and depressor anguli oris. The impact of this on oral competence can be variable. If loss of orbicularis function causes decreased tone and oral competence due to a flaccid segment of the oral stoma, wedge excision of the affected area of the lip can improve function. The lower lip can tolerate resection of up to 1/3 without decreasing oral opening and with minimal cosmetic impact. By removing the atonic segment of the lower lip, the remaining innervated muscle in the lower lip can better establish competence.

Fig. 13. Static facial sling on right side 8 years post-surgery. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

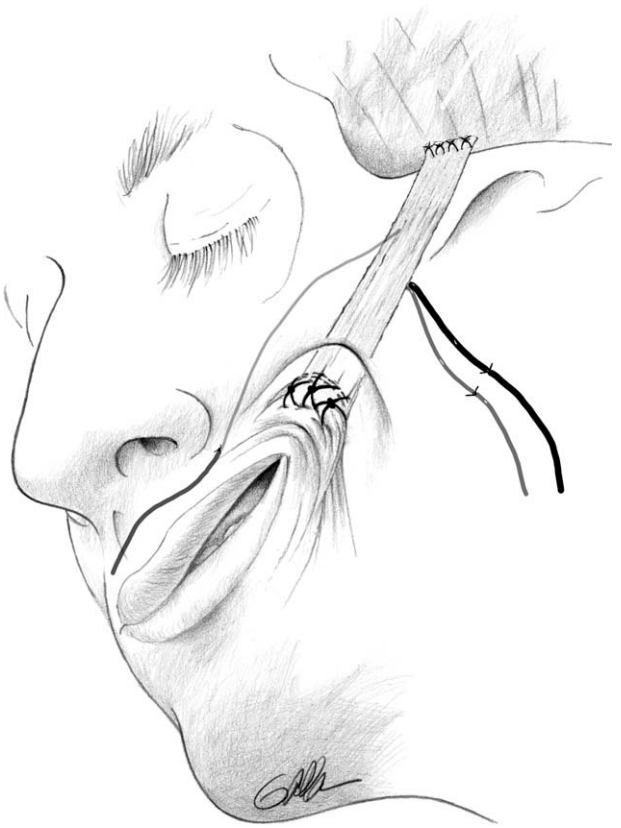

Fig. 14. Gracilis free flap inset with a cross facial nerve graft.

Loss of depressor anguli oris function is predominantly an aesthetic issue. Without this muscle, the smile and facial expressions can be significantly asymmetric. Although this may be less noticeable in patients with a complete facial nerve paralysis, it can be quite obvious in a patient with isolated marginal mandibular nerve weakness.

To deal with the asymmetry, the contralateral side can be addressed. By decreasing function of the contralateral muscle, the ipsilateral weakness becomes less noticeable. Initially, chemical dennervation of the contralateral muscle with a botox injection is attempted. If this produces favorable results, the contralateral depressor anguli oris muscle can be severed using a transoral incision, although this muscle can frequently grow back. Other options, such as transposition of the ipsilateral anterior belly of the digastric muscle, have been described with variable success (Tulley et al., 2000).

EVALUATION OF OUTCOMES

Multiple scales have been designed to quantify facial nerve injuries based on movement and disability. Objective evaluation of movement is measured from the perspective of the physician, who assesses-independent zones of movement to quantify residual facial nerve function. Commonly used examples include the House-Brackmann scale (House and Brackmann, 1985), Sunnybrook Facial Grading Scale (Ross et al., 1996), and the Syndey grading system, and more continue to be proposed (de Ru et al., 2006). These validated scales provide reliable measurement of volitional movement, although are not as effective at evaluating synkinesis (Coulson et al., 2005).

However, what is more relevant to the patient is the subject assessment of outcomes from their perspective, which is measured by various self-reported quality of life questionnaires. These questionnaires assess the physical and psychological impact of nerve dysfunction, and include the Facial Clinical Evaluation scale (Kahn et al., 2001) and the Facial Disability Index (VanSwearingen and Brach, 1996). Unfortunately, evaluation of outcomes of surgical procedures is relatively limited, with most surgeons using objective measures and not disability scales. The majority of the quality of life data relates to the ocular complex, and does show statistically improvement in symptoms and disability (Golio et al., 2007; Henstrom et al., 2011).

A comprehensive rehabilitation program is not limited to surgical treatment. Additional strategies such as targeted physical therapy, biofeedback, and neuromuscular retraining exercises can be a useful adjunct to recovery and have been shown to provide a statistically significant improvement in objective measures (Lindsay et al., 2010).

CONCLUSION

The challenges of facial nerve paralysis management are complex and patient specific. Rehabilitative and reanimation procedures can provide significant benefit to the patients from a functional and aesthetic standpoint. Multiple techniques are available and must be tailored to the specific clinical scenario.

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