CME

Evidence-Based Practices in Facial Reanimation Surgery

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Learning Objectives: After studying this article, the participant should be able to: 1. Describe the causes and preoperative evaluation of facial paralysis. 2. Discuss techniques to restore corneal sensation and eyelid closure, elevation of the upper lip for smile, and depression of the lower lip for lip symmetry. 3. Outline treatment goals, surgical treatment options, timing of repair, and other patient-specific considerations in appropriate technique selection.

Summary: Congenital facial paralysis affects 2.7 per 100,000 children; Bell palsy affects 23 per 100,000 people annually; and even more people are affected when considering all other causes. Conditions that impair facial mimetics impact patients' social functioning and emotional well-being. Dynamic and static reconstructive methods may be used individually or in concert to achieve adequate blink restoration, smile strength and spontaneity, and lower lip depression. Timing of injury and repair, patient characteristics such as age, and cause of facial paralysis are all considered in selecting the most appropriate reconstructive approach. This article describes evidence-based management of facial paralysis. *(Plast. Reconstr. Surg.* 152: 520e, 2023.)

Facial palsy is caused by dysfunction of the facial nerve and/or target musculature.[1–3] The facial nerve is responsible for activating facial musculature that is responsible for a number of key functions, including normal blink and eyelid closure to prevent corneal injury through desiccation and further sequelae; the competency of the oral sphincter to prevent drooling and enable chewing, drinking, speech articulation, and other oromotor functions; and facilitating normal psychosocial functioning through facial symmetry, aesthetics, and coordinated voluntary and involuntary facial movements that convey intended expressions. Reanimation procedures aim to restore these normal functions and muscle tone. It is important to note that psychosocial dysfunction has been consistently reported as the main driver for patients to seek treatment, irrespective of the objective severity of their facial paralysis and asymmetry.[4] Patient-reported outcomes data have confirmed that postoperative Facial Clinimetric Evaluation scale[5] scores are independent of facial movement scores in the pediatric population,[6] highlighting the importance of repair on psychosocial functioning and development.

A number of potential causes for facial paralysis have been reported. These may be classified as congenital or acquired. The latter may result from idiopathic/inflammatory conditions, granulomatous diseases, autoimmune disorders, trauma, infectious processes (ie, Lyme disease, Ramsay-Hunt syndrome, otic infections), and benign or malignant neoplasms.[7] The majority of cases of acquired facial paralysis are from Bell palsy, an inflammatory condition thought to be postviral, associated with herpes simplex virus type 1 or herpes zoster reactivation, versus idiopathic.[8] Bell palsy is a diagnosis of exclusion. Approximately three of four patients with Bell palsy will recover with conservative treatment alone,[9] consisting of ocular lubrication, and corticosteroids; in more severe cases, antivirals may be added.[10]

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In the literature, there are few large analyses of patients with facial nerve palsy.[11–14] A review of 2000 patients treated from 2003 to 2013 at a single tertiary referral center for acoustic neuromas and complex head and neck tumors represents the largest report of patients with facial nerve palsy performed to date.[12] In accordance with earlier work,[11,13] Bell palsy represented the leading cause of facial nerve palsy, representing 38% of cases, with women more commonly affected than men (65% women). The second most common cause of facial paralysis in this series was acoustic neuroma (10%), followed by head and neck cancer (7%). The majority of cases in both oncologic groups developed after resection of the tumor. However, because of a difference in referral patterns, trauma has also been found to account for 10% to 23% of all facial nerve palsies.[11,15]

Cases of congenital and acquired facial nerve paralysis also exist in children, with an incidence of 2.7 per 100,000 in children younger than 10 years and 10.1 per 100,000 in children older than 10 years.[1,2] Half of pediatric cases stem from an unknown cause[16] and congenital causes are more common than acquired, with perinatal trauma representing the most common cause of congenital facial palsy.[2] Fortunately, these cases typically have full recovery of nerve function without surgical intervention.[2]

Reanimation methods vary by location, cause, and timing of facial nerve injury; patient factors such as age; the availability of reconstructive options; and the experience of the surgeon.[17] Numerous surgical and nonsurgical options exist for reanimation, and they may be classified as static or dynamic. Surgical approaches for facial reanimation include approaches that manipulate nerves (ie, primary repair, nerve transfer, nerve graft), muscles (ie, local or regional muscle transfer, free functional muscle transfer), or use a combination of techniques. The choice of approach depends on the timing from injury (Table 1), as this inversely correlates with the progressive atrophy of in situ mimetic musculature.[18] Once this has occurred, preference should be given to a muscle transfer or static sling technique.[19,20] In the case of early injuries (<12 months), however, the patient is likely to retain some viable muscle function and the options for reanimation include a nerve transfer and/or nerve graft.

With respect to Bell palsy, patients who do not recover with conservative management have historically been recommended for surgery within 3 months of onset.[21] However, one group out of Kyung Hee University Hospital found no significant difference in the rate of favorable recovery or in the degree of improvement of House-Brackmann grades between severe cases of Bell palsy that were treated with conservative measures alone versus those that underwent additional decompressive surgery 21 to 70 days after conservative treatment failed.[22] Moreover, a recent Cochrane review deemed there to be low-certainty evidence from randomized controlled trials to decide whether early surgical intervention is beneficial or harmful to patients with Bell palsy.[21] In this CME article, we will outline conservative and surgical options for the management of eyelid closure and reestablishing corneal protective sensation, smile reanimation, and restoration of lip depression.

RESTORATION OF EYELID CLOSURE AND CORNEAL PROTECTION

Paralysis or weakness of eyelid musculature may result in loss of normal blink and corneal protection because of lagophthalmos, paralytic ectropion, and loss of corneal sensation and the tearing reflex.[23] This must be addressed within the context of any other lid abnormality that may be present, such as ptosis, laxity, and/or dermatochalasis.[24] Blink can be restored with either static or dynamic methods, and corneal sensation may be restored with a novel technique using nerve grafts to reneurotize the cornea.[25] Timely intervention prevents corneal desiccation, which may manifest as superficial punctate keratopathy, corneal

Table 1. Timing of Reanimation following Facial Nerve Injury

Time from Facial Nerve Injury	Reconstructive Options
Immediate	• Primary repair for tension-free clean laceration • Nerve autografting from ipsilateral facial nerve proximal to injury
<12 mo	• CFNG if ipsilateral facial nerve is not available and contralateral facial nerve is available • Ipsilateral nerve transfer if distal facial nerve stumps intact (masseteric nerve preferred)
≥12 mo	If muscle intact: • CFNG with or without nerve transfer (nerve transfer may be used temporarily, ie, "babysitter procedures," or may be permanent)
	If muscle not intact: • CFNG plus free functional muscle transfer • Nerve to masseter plus free functional muscle transfer • Static slings

ulcers or, in the most extreme cases, perforation.[23] Establishing ophthalmologic care and regular slit-lamp examinations is necessary to monitor for and avoid ocular complications.

Conservative Management of Eyelid Paralysis

Conservative management prioritizes ocular lubrication using eye drops and ointments, eyelid taping, and/or humidifying goggles.[26] Temporary tarsorrhaphy is indicated if these interventions fail to remedy ocular dryness within a few weeks.[27] Tarsorrhaphy may be a bridge to definitive surgical correction or may be reversed for transient palsies. Permanent tarsorrhaphy is not commonly performed because of unacceptable disfigurement.[7]

Static Methods for Blink Restoration

The standard for restoration of voluntary blink and static lid closure restoration is placement of a gold weight in the upper eyelid. Unlike methods using a magnet or palpebral spring, placement of an upper eyelid weight is straightforward and reversible.[27,28] It is able to correct varying severity of eyelid retrusion using different sized weights, typically between 0.6 and 2.6 g and increasing by 0.2-g increments.[29,30] An appropriate weight centered at the medial limbus is able to achieve complete lid closure while limiting ptosis to no more than 2 mm measured by the margin reflex distance 1.[29,31] Preoperative evaluation using tape to secure various weights to the upper lid aids in determining the correct weight to implant or may be used as a temporizing solution.[32] Platinum is a potential alternative to gold, and has been used to decrease the visibility of the implant and was noted to carry a lower risk of extrusion, although comparative studies between metals have not been performed.[33] In addition, platinum has been associated with allergic conjunctivitis.[34]

Weights can be placed pretarsally[30] and postseptally.[34,35] The pretarsal approach has greater predictability of eyelid position and thus requires fewer early revisions but is associated with increased implant visibility and a 10% risk of extrusion at 5 years.[24,36,37] The postseptal approach has been found to minimize complications associated with pretarsal weight placement,[35] and is well suited to patients who have thinned eyelid skin, such as older patients, mitigating the risk of extrusion.[34] However, postseptal weight placement has historically required intraoperative weaning of sedation for real-time assessment of lid position. More recently, preoperative estimates and actual weights have been correlated, with an average correction of an additional 0.2 g required intraoperatively to achieve desired lid position.[35] Similarly, among revisions, the average correction was an increase of 0.2 g, although most patients had successful blink restoration after the first operation. Among both pretarsal and postseptal techniques, most patients attain complete eyelid closure with voluntary blink.

Alternatives to eyelid weight placement avoid introduction of a foreign body. In one procedure, lengthening of the levator muscle is performed by means of complete disinsertion from the tarsus coupled with interposition of a rectangular piece of autologous graft from the temporalis muscle or fascia lata measuring twice the width of the observed preoperative lid retrusion.[24] For milder lid retrusion of 1 to 3 mm, Müllerectomy may be performed with excellent results.[38,39]

When paralytic ectropion is present, the lower lid may be addressed with medial[29,40] and/or lateral canthoplasty,[27,29,40,41] recession of lower eyelid retractors,[42] suspension with fascia lata or tendon graft,[29] or use of temporalis muscle as a sling to resuspend the lower lid.[41] Most authors recommend lateral tarsal strip tightening in combination with gold weight placement for patients with paralytic ectropion and loss of blink, as these procedures reliably yield reproducible results and are well tolerated.[27,40,43] Ear cartilage grafts have also been used for management of lower lid paralytic ectropion to restore the lower lid height and apposition to the globe, but this approach is more challenging and is reserved for commensurately difficult clinical scenarios where there is a significant paucity of local tissue for successful use of resuspension techniques alone.[44]

Dynamic Methods for Blink Restoration

Neurotization of the orbicularis oculi muscle either directly, through cross-facial nerve grafts (CFNG) from the contralateral zygomaticotemporal branch, or by means of ipsilateral nerve transfers reestablishes the native blink, improves resting tone, and diminishes ectropion with outcomes superior to those achieved with muscle-based procedures.[17,45,46] Patients who undergo combined static and dynamic lid closure restoration experience greater improvements than either technique alone, with improvement in exposure keratopathy and greater and faster palpebral aperture closure.[47]

Targeted reinnervation of the eyelid by means of the zygomaticotemporal branch is typically preferred over coaptation to the main trunk of

the facial nerve because of a lower risk of synkinesis.[19,48] A retrospective study of eight patients with facial paralysis showed improved preoperative eyelid synkinesis following masseteric nerve transfer to the zygomaticotemporal branch of the facial nerve, suggesting that targeted reinnervation of the eyelid may not only prevent postoperative synkinesis following reanimation but may also treat it if present preoperatively.[48]

In cases where the orbicularis oculi muscle is absent or atrophic, regional or free muscle transfer may be required for dynamic blink restoration. The regional options include frontalis and minitemporalis, which are pedicled and reoriented to replace the eyelid sphincter mechanism.[45] Free muscle donors include the platysma[49] or a slip of pectoralis.[46]

Corneal Neurotization

Loss of corneal sensation is associated with potential morbidity to visual acuity.[23] Restoration of protective corneal sensation is now possible through nerve grafting[50] and ameliorates exposure keratopathy.[51–53] Corneal reinnervation procedures are typically performed at the time of other planned operations for facial reanimation.[54] Sensory reinnervation of the cornea may be accomplished by using nerve transfers alone or in concert with a nerve graft. Early efforts in this field used nerve transfers using contralateral supratrochlear and supraorbital nerves.[55] However, these techniques required an invasive bicoronal approach and were not an appropriate option in bilateral trigeminal nerve palsies.[54] In addition, a major drawback of isolated nerve transfers is that they rely on small-caliber distal nerves; this is thought to contribute to the delayed corneal sensibility that has been observed in contralateral supratrochlear and supraorbital nerve transfers, with an average of 2.8 years.[54] The prolonged duration of recovery has been successfully mitigated with the use of nerve grafts coapted to a more proximal and robust donor nerve stump, with return of sensibility as early as 3 months from surgery.[54] The medial cutaneous branch of the sural nerve can be used as an autograft to bridge the sensory donor nerve directly to the cornea.[56] The graft allows for a greater variety of nerves to be used as donors, including maxillary and mandibular divisions of the trigeminal nerve if intact. However, in patients where the ipsilateral trigeminal nerve is spared and demonstrates intact sensation in the V1 distribution, the ipsilateral supratrochlear nerve is the preferred donor for its proximity to the affected cornea.[54] The neurotization of the corneal limbus is performed in collaboration with ophthalmology.[55] If a sural nerve graft is used, multiple fascicles are present (typically, four to eight), and thus neurotization may be distributed to provide sensation to the full surface area of the cornea.[54]

Postoperatively, patients are monitored for return of corneal sensation using various ophthalmologic tools, such as a von Frey hair or a Cochet-Bonnet esthesiometer, which are designed to quantify sensibility of the corneal surface.[55] Patients in multiple small studies have been found to have improved corneal sensibility with all aforementioned approaches, and some have endorsed improved visual clarity because of decreased corneal clouding and desiccation.[54,55] However, in patients with severe corneal scarring, visual acuity may not be salvageable.[54]

RESTORATION OF SMILE

Smile and oral sphincter restoration enables patients to chew, speak, and emote effectively. Dynamic restoration reestablishes volitionally contractile muscle and yields superior results but demonstrates diminishing improvements with increasing patient age because of loss of neuroplasticity. When dynamic restoration is not possible, static slings and local muscle transfers offer alternatives by restoring resting symmetry. Younger patients with longstanding facial paralysis may require a combination approach to correct facial asymmetry at rest and on smiling. Management of congenital facial palsy is outlined in Supplemental Digital Content 1. (**See Figure, Supplemental Digital Content 1**, which displays the algorithm for reanimation of congenital facial palsy, ***http://links.lww.com/PRS/G72***.)

Static Methods for Smile Restoration

Static slings and local muscle transfers are most appropriate when a rapid solution is required or in older patients who may not have the regenerative capacity to attain significant benefit from neurotizing procedures. Static slings resuspend the oral commissure to address asymmetry of the face at rest,[7] but this is subsequently disillusioned with movement.[57] Original static techniques involved stretching a strip of fascia lata over the zygomatic arch to reach the oral commissure[58] or transferring the entire muscle to the commissure by means of fascia lata after the muscle has been divided from the coronoid process through an intraoral approach.[59]

Autologous palmaris or plantaris tendon-graft slings provide the longest lasting suspension results compared with other materials.[57]

Dynamic Methods for Smile Restoration

Nerve Graft to Existing Facial Muscles

A nerve graft (or cable graft) should be considered in the event of inaccessible proximal and distal nerve stumps or where a wide neural gap cannot be repaired without tension—an antagonist to neuronal sprouting.[60] For a tension-free coaptation, cable grafts should be roughly 25% longer than the defect.[61] The most popular autologous graft is the sural nerve, because of its excellent size match, length, minimal donor-site morbidity characterized by lateral foot numbness, and two-team approach.[7] The length of the sural nerve allows for the creation of two or three independent cable grafts,[62] which can be used to recreate the branching pattern of the original facial nerve during reconstruction.[63] Motor nerves generally carry unacceptable donor morbidity compared with their sensory counterparts.

The best method to achieve symmetric spontaneous excursion is a CFNG, which is a combination of a nerve transfer and graft (Fig. 1).[7] In this type of repair, signals from a branch of the functioning contralateral facial nerve (transfer) are transmitted to muscle on the affected side by means of a neural conduit (graft). Regeneration of the nerve across the CFNG is followed clinically by the Tinel sign and can take approximately 9 to 12 months to heal. A shorter CFNG is preferred to decrease the distance and time required for the nerve to regrow.[20] In case of longer recovery times (>6 months), the use of an ipsilateral nerve transfer should be considered to prevent muscle atrophy while the CFNG is healing.[20] Such a nerve transfer was initially labeled a "babysitter procedure" because the intent was to take down the transfer once the CFNG reinnervated the recipient muscle. However, nerve transfers can also be left in place to augment the power of the CFNG to achieve greater excursion.

Outcomes for cable grafting are often unpredictable.[64] These can be improved by using distal branches of the facial nerve rather than the proximal trunk as the donor site for the CFNG. This approach provides greater specificity of function and less likelihood of donor-site weakness if performing end-to-end coaptations. Many contemporary techniques use concurrent nerve transfer with cable grafting to improve outcomes from immediate repair of killed facial nerves.[64]

Transfer of Nerve to Masseter to Branches of the Facial Nerve

In contrast to a nerve graft, a nerve transfer may be considered in the event of an intact and accessible distal nerve stump. Specifically, unilateral nerve transfers are often performed for traumatic cases of facial palsy that undergo reanimation within 1 year of injury to preserve muscle function on the affected side. Nerves available for transfer include the nerve to the masseter, hypoglossal nerve, spinal accessory nerve, and phrenic nerve.[20,65–68] The nerve to the masseter and hypoglossal nerve are the center of recent research and clinical use and are the focus of this review.

The nerve to the masseter may be identified in the subzygomatic triangle, where it bisects the angle between the temporomandibular joint and the zygomatic arch.[19] The nerve follows an oblique course within the deep substance of the masseter muscle, and gives off a series of small proximal branches, followed by a dominant descending branch.[66] Dissection of the dominant descending branch to a length of up to 3 cm facilitates tension-free coaptation to the main trunk of the facial nerve.[19,66]

The masseter-to-facial nerve transfer produces strong and potentially spontaneous animation with minimal donor-site morbidity and fast reinnervation (Fig. 2).[19,66] [**See Video 1 (online)**, which displays the outcome of fifth to seventh nerve transfer.]

It may also be coupled with CFNG to augment the strength and excursion of the oral commissure (Fig. 3). A systematic review and meta-analysis of 10 studies investigating time from reanimation to facial movement following masseteric nerve transfer reported a pooled outcome of approximately 5 months.[69] A subgroup analysis of transfers to the main trunk versus distal branches of the facial nerve (ie, zygomaticotemporal and buccal branches) identified an even faster time to recovery when the coaptation involved distal branches (main trunk, 5.76 months; distal branches, 3.76 months), although the difference was not statistically significant between the groups.[69]

The nerve to the masseter was shown to have a faster rate of recovery after transfer than the hypoglossal nerve in two studies: those of Albathi et al. (5.6 months versus 10.8 months) and Hontanilla and Marré (62 days versus 136 days).[70,71] This is physiologically intuitive because a coaptation at the main trunk is farther from the facial musculature, resulting in longer distances for nerve regeneration.[70]

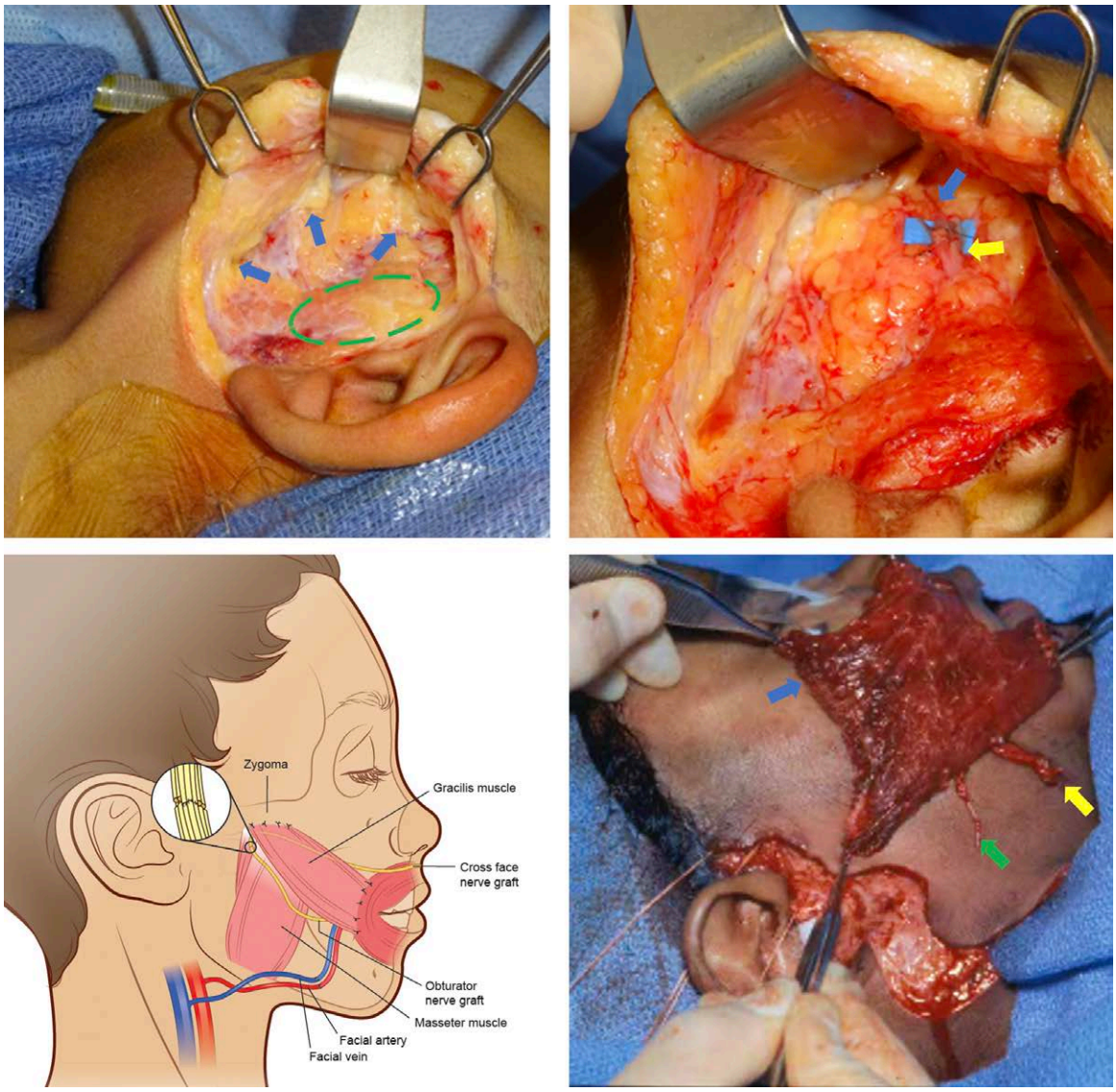

Fig. 1. Illustration of two-stage approach to CFNG and free functional gracilis muscle transfer, showing the first stage, including (*above*, *left*) the subsuperficial musculoaponeurotic system dissection plane (*blue arrows*) on the unaffected left side of the face (parotid shown with *dashed green oval*); (*above*, *right*) coaptation of nerve cable autograft (from sural donor, *blue arrow*) to a branch of the left facial nerve (*yellow arrow*); and second stage, including (*below*, *left*) schematic demonstrating CFNG traveling across the midline from the unaffected to the affected side; and (*below*, *right*) free functional muscle transfer performed at subsequent surgery (*blue arrow*, free gracilis muscle; *yellow arrow*, medial circumflex vascular pedicle before anastomosis with recipient vessels in the face; *green arrow*, obturator nerve before coaptation with recipient nerve in the face).

Concerning reinnervation of smile, masseteric nerve transfer to the buccal branch of the facial nerve has been found to reliably elevate the oral commissure to the preparalysis state.[19] Between the affected and unaffected sides, Hontanilla et al. achieved successful symmetry in 23 cases, as demonstrated by no statistical difference in mean postoperative commissural excursion or commissural contraction velocities between sides.[72]

Other benefits of the masseteric nerve transfer include its relatively low morbidity and lower rate of synkinesis than the hypoglossal nerve transfer. Murphey et al. reported 12 complications among 183 patients, which included infection, hematoma, masseter atrophy, ocular discomfort with chewing, sialocele, and otitis externa.[69] When compared with hypoglossal nerve transfer, the risk of donor-site morbidity is low because the masseteric nerve

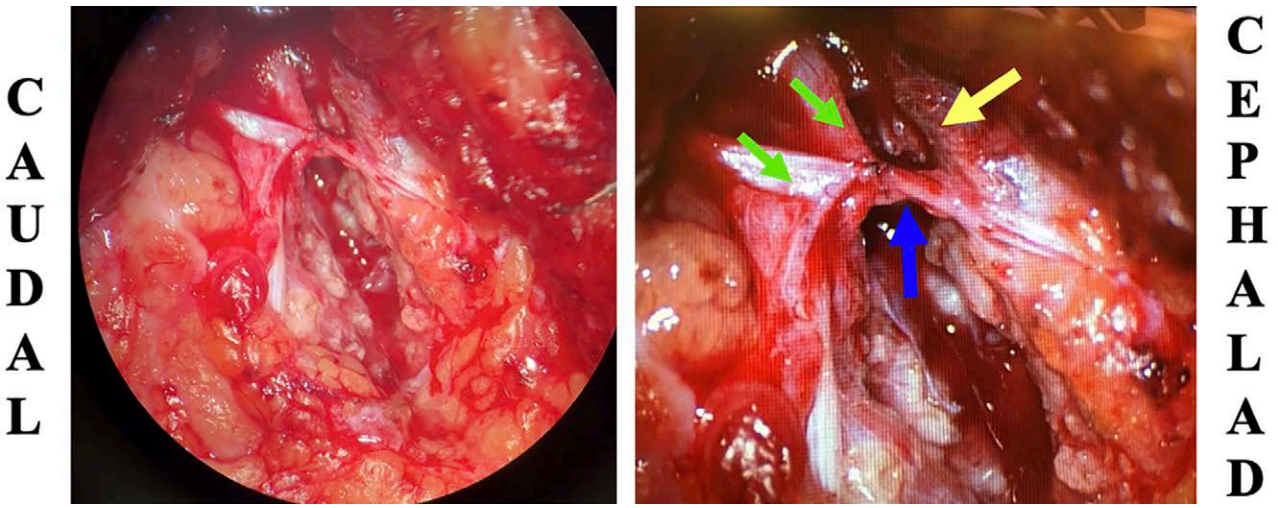

Fig. 2. Transfer of nerve to masseter (*blue arrow*) to buccal and zygomaticobuccal branches (*green arrows*) and frontozygomatic branch (*yellow arrow*).

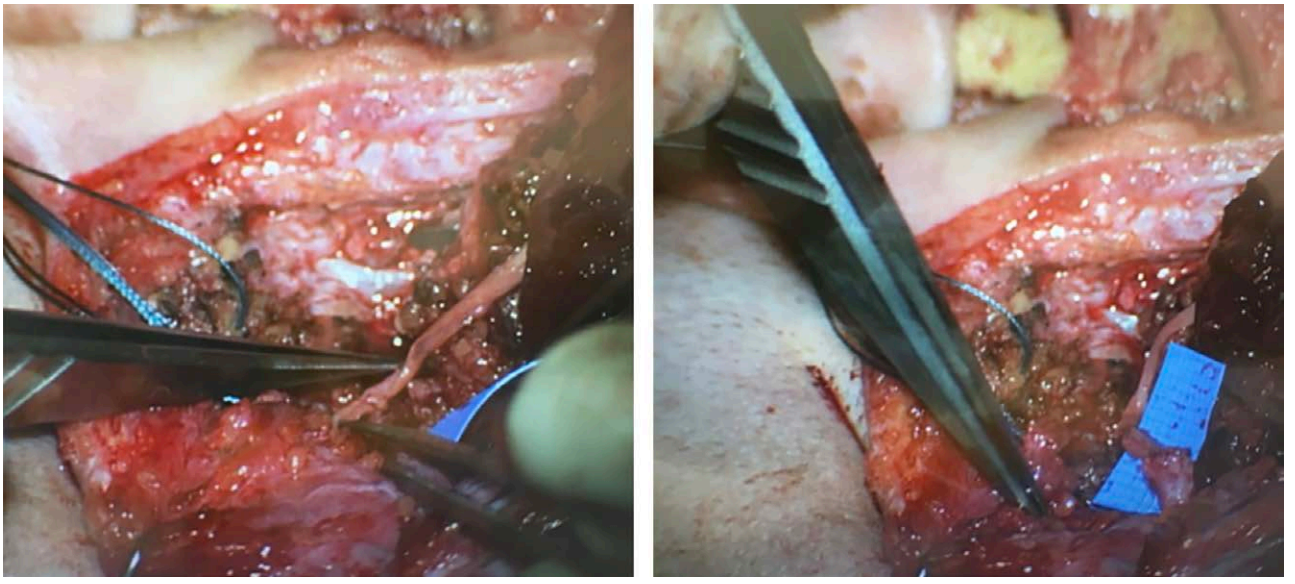

Fig. 3. Augmentation of (*left*) CFNG powering the obturator nerve to the transferred gracilis muscle using (*right*) transfer of nerve to the masseter.

is divided distal to its major motor contributions. For older patients who cannot tolerate general anesthesia, a masseteric nerve transfer can be performed under light sedation and local anesthesia.[73]

Substituting the masseteric nerve instead of the facial nerve to power mimetic musculature requires neuroplasticity such that voluntary biting is uncoupled from voluntary smile. With practice, patients may learn to smile with their mouth open without biting, and approximately half will go on to develop spontaneity of smile in response to joy or humor (Fig. 4).[69,74] [**See Video 2 (online)**, which displays the outcomes of nerve to masseter transfers.] This may take months to years to accomplish and is more efficient in younger patients[75] and women.[69,76]

Transfer of the Hypoglossal Nerve to the Facial Nerve

Unlike the nerve to the masseter, the hypoglossal nerve is most commonly coapted directly to the main facial trunk because of its location.[69] This maneuver is thought to increase the risk of synkinesis compared with patients undergoing a transfer with the nerve to the masseter.[69] [**See Video 3 (online)**, which shows outcome of CFNG,

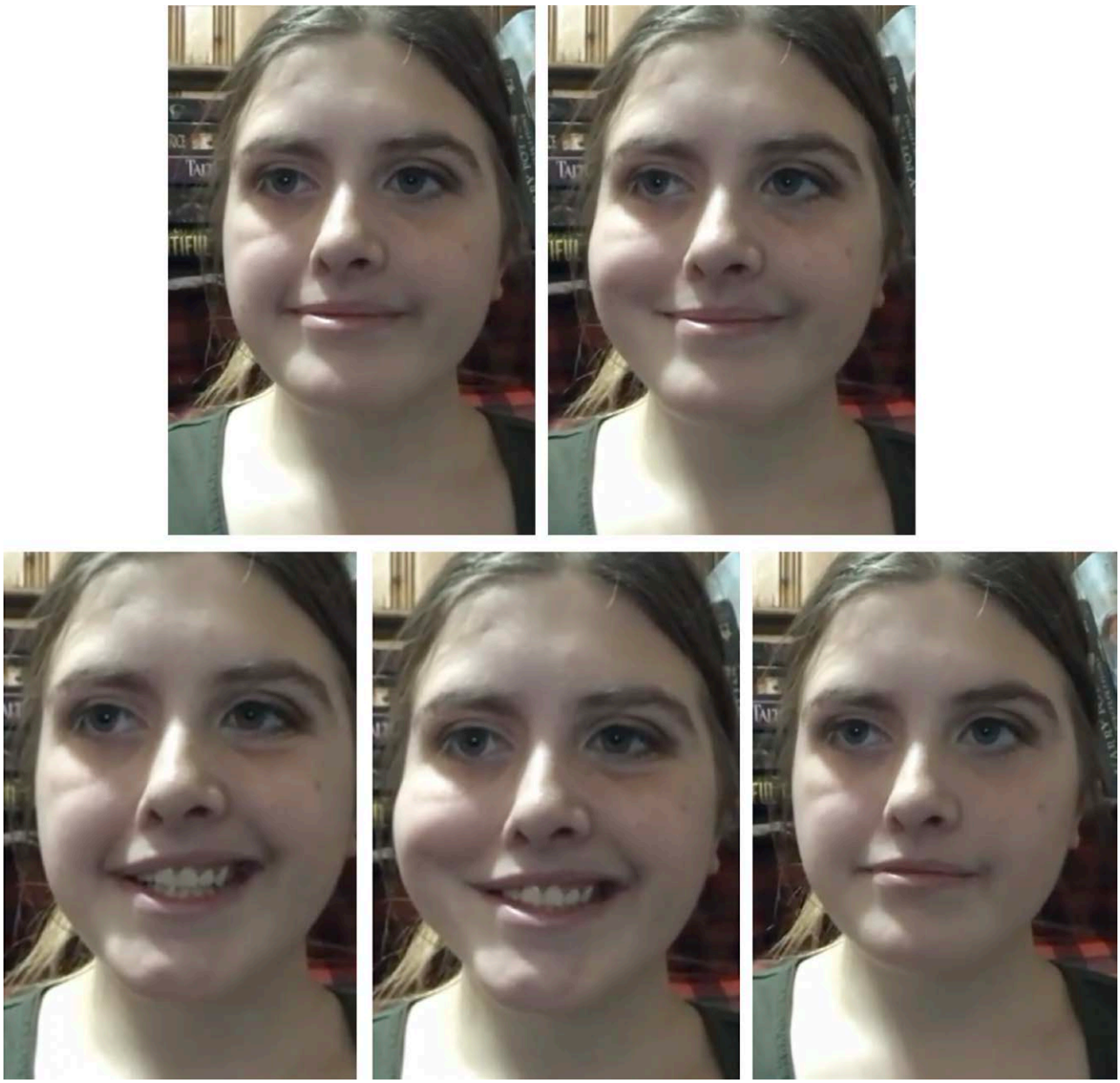

Fig. 4. Patient who received free functional gracilis innervated by nerve to masseter for right hemifacial paralysis secondary to Moebius syndrome. (*Above, left,* and *below, left*) The patient was asked to smile. (*Above, right,* and *below, center*) The patient was asked to smile and bite down simultaneously, demonstrating augmentation of the excursion of the right oral commissure when voluntary bite is elicited. (*Below, right*) The patient was asked to stop smiling and only bite down, demonstrating that she has uncoupled these two distinct functions.

free gracilis, and hypoglossal-to-facial nerve transfer.]

Despite equivalent outcomes for facial expression and symmetry as with masseteric nerve to facial nerve transfers, patients may experience hemiglossal atrophy secondary to functional impairment of the hypoglossal nerve itself.[77] With this in mind, this technique may not be appropriate for patients with Moebius syndrome, whose condition leads to inherent speech and swallowing impairments in the majority of patients.[20]

Strategies to minimize donor-site morbidity include end-to-side coaptations[78–80] and transferring part of the hypoglossal nerve, leaving behind fascicles that remain in continuity to maintain tongue function.[61,81,82] Another approach that may be taken to avoid tongue atrophy involves

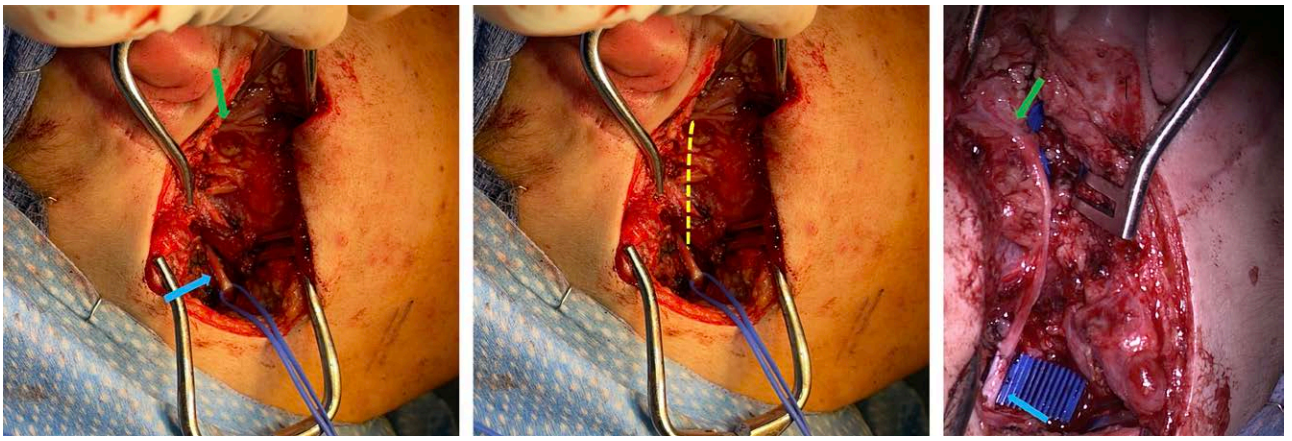

Fig. 5. (*Left*) The hypoglossal nerve (*blue arrow*) and the facial nerve (*green arrow*) are identified. (*Center*) These nerves can be bridged by an interposition nerve graft (*dotted yellow line*), usually taken from the sural nerve, and arranged in end-to-side fashion at both coaptations. Use of an interposition nerve graft in end-to-side arrangement circumvents the morbidity associated with traditional hypoglossal nerve transfers (ie, babysitter procedures). This approach also does not require division of the facial nerve trunk or its branches and thereby preserves the potential for functional recovery of the facial nerve, which is possible in the case of multiple causes of facial paralysis. (*Right*) The hypoglossal (*blue arrow*) and facial nerve (*green arrow*) are bridged by a sural nerve graft.

indirect hypoglossal-facial coaptation with interposition of a nerve graft (Fig. 5).[83,84] Overall, findings of facial synkinesis, tongue atrophy, and varying degrees of speech and swallowing impairments favor the masseter-to-facial nerve transfer.

Outcomes of using a CFNG have been less favorable than nerve transfer techniques using the nerve to the masseter, which exceed CFNG in both time to nerve recovery and improvement in oral commissure excursion (Fig. 6).[69] The number of axons present in the donor nerve is one predictor of strength of muscle reinnervation following nerve-based techniques.[85] Axon counts are lower in the branches of the contralateral functioning facial nerve to which the CFNG is coapted (100 to 200 axons)[69,86] compared with 1250[86] to 2700[18] axons in the masseteric nerve and 9200

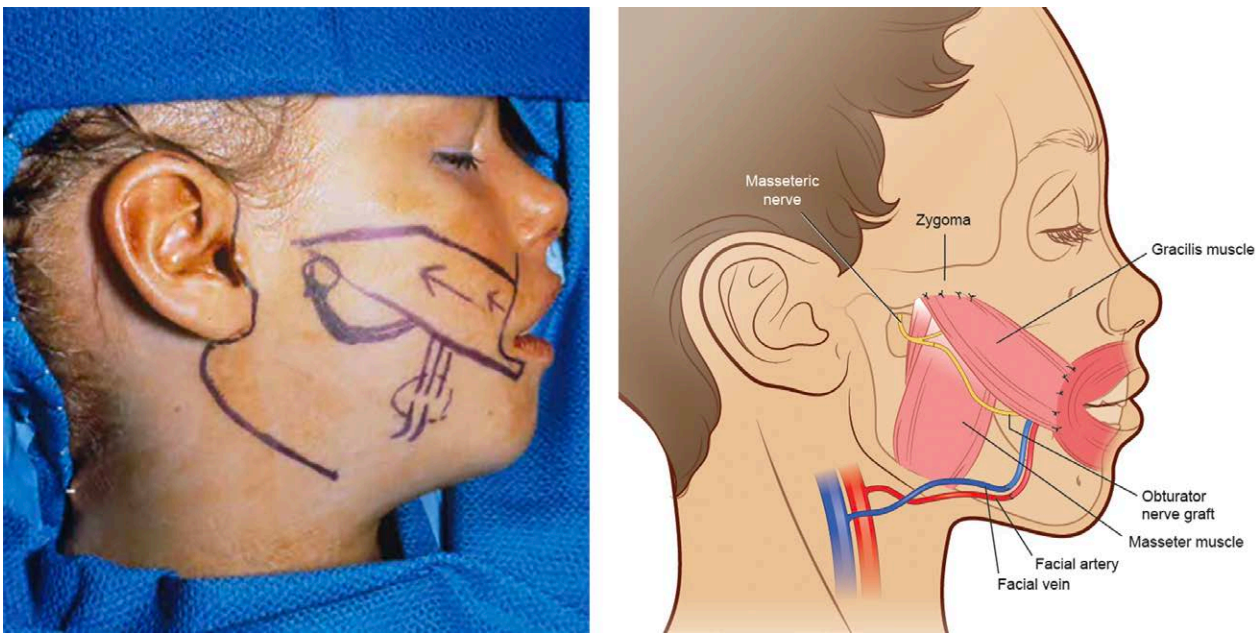

Fig. 6. (*Left*) Intraoperative markings and (*right*) schematic representation of free functional gracilis muscle transfer powered by coapting the ipsilateral nerve to the masseter to the obturator nerve.

axons in the hypoglossal nerve.[87] To achieve maximum axon counts distal to the neurorrhaphy site and minimize loss of regenerating axons, nerve grafts are typically reversed in anatomical orientation during coaptation.[20]

Local Muscle Transfer

Several regional muscle transfer techniques have been described to dynamize the face in clinical situations that lack viable muscle on the affected side. First described in 1998, the Labbé procedure involves transferring the fixed coronoid process to the lips.[88] Lengthening temporalis myoplasty is a one-stage outpatient procedure with subtle incisions and prompt results, without the need for fascia lata extension or the bulk of the traditional transposition technique.[89] In Labbé's original case series of 10 patients with unilateral facial paralysis, strength of muscle contraction was at least 1.5 cm in amplitude after 6 months of physical therapy, surpassing the force elicited by partial temporalis muscle-tendon transfer with a fascia lata sling.[88] With respect to other temporalis myoplasty techniques, outcomes following the Labbé procedure are also more aesthetically pleasing than the Gillies[58] or McLaughlin[59] procedure, which creates a muscular bulge over the zygomatic arch or falls short in its recreation of the nasolabial fold, respectively.

Similarly, the pedicled masseter muscle transfer technique repositions the muscle following detachment of both the origin and insertion to achieve an appropriate contraction vector for smiling.[90] When both muscles are available for use, however, the temporalis is generally preferred to the masseter because it allows for a 45-degree traction in the occlusal plane[88] versus the unsuitable horizontal pull of the pedicled masseter muscle transfer.[91]

Free Functional Muscle Transfer

Free muscle transfer powered by a nerve transfer or CFNG is indicated for patients with good regenerative capacity and chronic denervation of facial mimetic musculature. This approach offers a chance to achieve a more natural, spontaneous smile. The gracilis muscle is used most often and is anchored to recreate the nasolabial fold and simulate the vector of a natural smile (Fig. 7). The transferred muscle can be powered by means of CFNG, the ipsilateral nerve to the masseter, or other nerves. We provide an overview of the technique of a free gracilis muscle transfer innervated by CFNG or nerve to the masseter. [**See Video 4 (online)**, which displays the surgical technique for sural nerve harvest for CFNG. **See Video 5 (online)**, which displays the surgical technique for free gracilis and nerve to masseter part 1. **See Video 6 (online)**, which displays the surgical technique for free gracilis and nerve to masseter part 2.]

In comparison to the nerve to the masseter, innervation of the gracilis muscle by CFNG has been criticized for producing significantly less excursion of the oral commissure during attempted smile.[92] In a review of 166 free segmental gracilis muscle transfers innervated by either a CFNG or nerve to the masseter, the extent of oral commissure movement when innervated by the nerve to the masseter was found to not only exceed that of the CFNG (nerve to the masseter, 14.2 mm; CFNG 7.9 mm), but to also approximate normal ranges (15.2 mm).[93] As long as the free functional muscle transfer is adequately revascularized and reinnervated, it will gradually recover function over 1.5 to 2 years, beyond which the aesthetic and functional outcomes are believed to be maintained in the long term.[94]

RESTORATION OF LIP DEPRESSORS

Clinical scenarios that specifically impair activation of the lower lip by means of paresis or paralysis of the depressor anguli oris (DAO) muscle, as seen in asymmetric crying facies,[95] also warrant reanimation. Restoration of lower lip depressors may be achieved by contralateral paralysis of the unaffected side using botulinum toxin injections[96] or denervation, or by ipsilateral reconstruction using a local muscle transfer.[97] We favor the latter approach because of the need for repeated botulinum toxin injections every 3 months, which is prohibitive in children, and the potential for long-term morbidity to oral sphincter competence with denervation.

One option is transfer of the anterior belly of the digastric muscle because it is innervated by the trigeminal nerve.[98] Another valuable option, although rarely described in the literature, is transfer of the ipsilateral platysma muscle, as fibers of the platysma interdigitate with those of the DAO and have a depressor effect on the commissure.[99] The difficulty of this maneuver, however, pertains to establishing integrity of the platysma muscle preoperatively. Because both the platysma and DAO muscle yield similar depression of the lower lip when activated, many mistake paralysis of the DAO, which is innervated by the marginal mandibular branch of the facial nerve, for paralysis of the platysma.[100] Superiorly, the platysma is co-innervated by the marginal mandibular

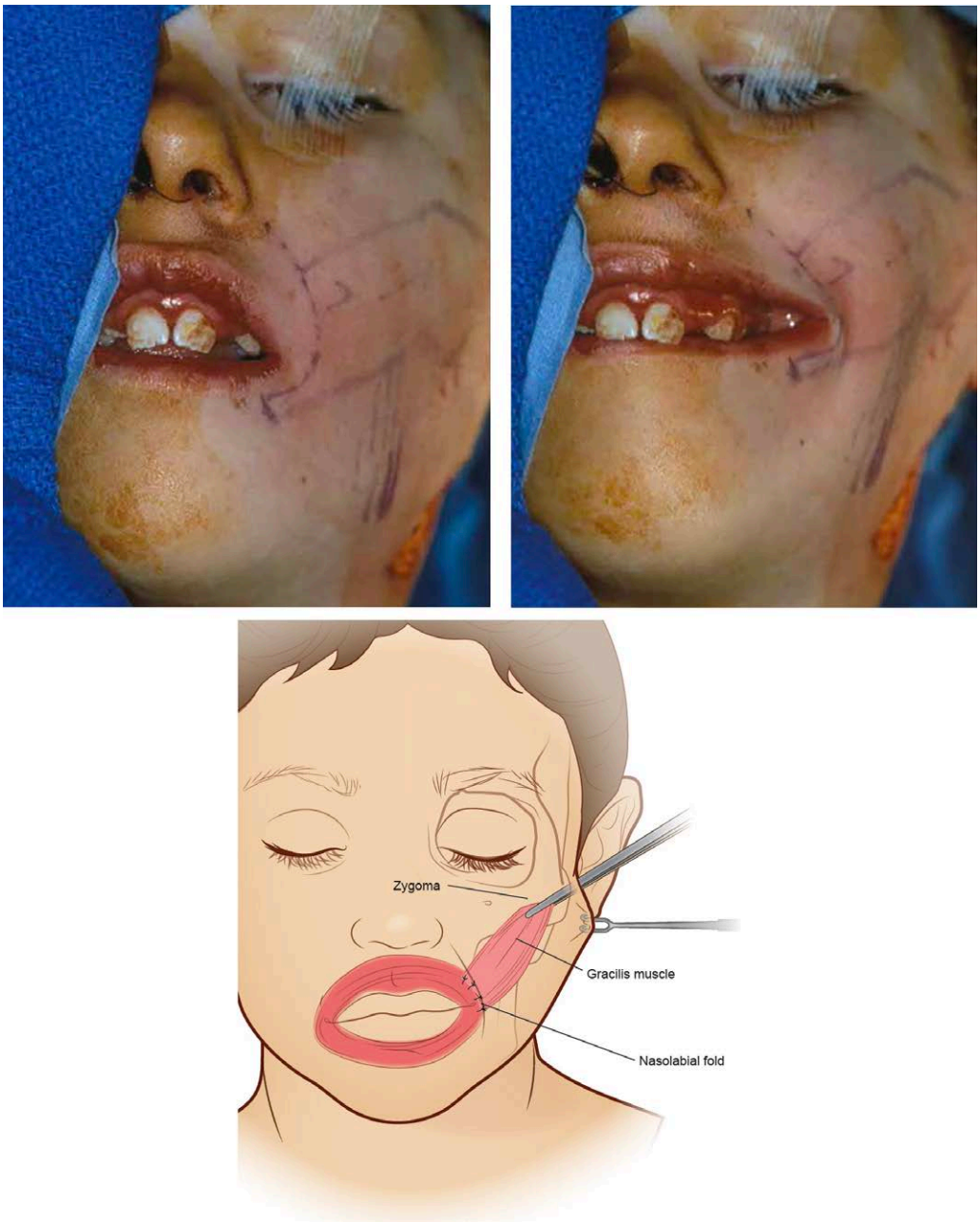

Fig. 7. The gracilis muscle is anchored to the deep surface of the anticipated nasolabial fold (mirrored from the normal side wherever possible). The muscle is then tensioned (*above, left*) with a superolateral vector and secured to the periosteum of the zygomatic arch to recreate the nasolabial fold (*above, right*). (*Below*) Illustration of muscle tensioning.

and cervical branches of the facial nerve; however, inferiorly, at the level of the hyoid, cervical branches fan out and innervate the remaining muscle.[101] Therefore, facial electromyography should be used to localize denervation, evaluate muscle atrophy, and prognosticate functional outcomes.[102,103] Results of platysma transfer for DAO paralysis (using clockwise rotation of the platysma to the lower lip) can be seen. [**See Video 7 (online)**, which displays the outcome of platysma transfer for lip depressor restoration.]

SUMMARY AND FUTURE OF FACIAL REANIMATION

Patient selection is paramount to appropriate management of facial palsy and relies on the surgeon's understanding of the various clinical

findings, cause, and expected clinical course; and possession of a broad array of technical approaches to apply to these diverse clinical scenarios. Procedure selection may be variable between adult and pediatric patients, who warrant independent consideration. In congenital patients, we prioritize dynamic procedures that result in the greatest spontaneity and symmetry, whereas in adults, we tend to choose what will give them the most immediate restoration. In addition, although facial reanimation is the primary aim, this must be coupled with an effort to minimize morbidity of donor sites and donor nerves that may be used in the reconstructive approach. A major obstacle to the development of robust evidence-based guidelines for facial reanimation is the variability of existing severity grading scales to describe preoperative and postoperative asymmetry and functional facial expression. This has limited comparisons between studies and techniques.[24,104–107] Small sample sizes further inhibit the generalizability of most studies. It is clear, however, that earlier interventions yield improved functional outcomes, and every effort must be made to intervene without delay.[17,108–110]

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DISCLOSURE

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PATIENT CONSENT

Parents or guardians provided written informed consent for use of patients' images.

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