SPECIAL COMMUNICATION

Beyond Botox: Contemporary Management of Nonflaccid Facial Palsy

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Abstract

Many transient facial nerve insults, including Bell's palsy, Ramsay Hunt syndrome, Lyme-associated facial paralysis, and temporal bone fractures, ultimately lead to nonflaccid facial palsy (NFFP). NFFP is associated with significant functional, aesthetic, psychological, and social impairments. However, algorithms for comprehensive management of this entity are not well developed and are rapidly evolving. Herein, we present our current approach to NFFP, with the goal of providing a framework that may be applied to this complex patient population.

Background

Historically, treatment for nonflaccid facial palsy (NFFP) was restricted to physical therapy, muscle relaxants, facial massage, acupuncture, and, rarely, myectomy or neurectomy. The introduction of chemodenervation using botulinum toxin revolutionized management for NFFP, as it provided patients a low-risk option to improve facial function and symmetry. Furthermore, results of targeted chemodenervation instructed providers on how *selective* myectomy and neurectomy procedures might ultimately be used to treat the sequelae of NFFP.

Today's treatment of NFFP includes targeted facial neuromuscular therapy, chemodenervation using botulinum toxin, selective myectomies, selective denervation, and nerve and muscle transfers in patients refractory to less invasive procedures. The treatment ladder presented herein is provided as one of many potential frameworks to employ when evaluating and treating patients with this often vexing condition. Our algorithm, derived as much as possible from available evidence, suggests systematic escalation of care in a stepwise manner.

Causes of NFFP

NFFP develops 6–18 months after incontinuity facial nerve insults, and variably after facial nerve repair and grafting, and is characterized by zonal synkinesis, hyper-tonicity, and hypomobility.¹ Bell's palsy results in NFFP in 15–30% of patients.^{2,3} Other common causes of NFFP

include Ramsay Hunt syndrome, acoustic neuroma resection, Lyme disease, trauma, and autoimmune disease.⁴ Furthermore, facial nerve repair, cable grafting, and nerve transfers can all lead to synkinesis.^{5–7} Although there are multiple theories explaining the development of NFFP, the most widely accepted theory is that regenerating axons extend in a multiply terminal manner to both appropriate and inappropriate targets.^{8,9}

Impact of NFFP

Although the morbidity of patients suffering from flaccid facial palsy has been well characterized, patients with NFFP also struggle with significant functional, aesthetic, psychological, and social impairments.^{10–15} Patients affected by NFFP often have a narrowed palpebral fissure, prominent and misoriented nasolabial fold, neck and face tightness, and mentalis dimpling.^{16,17} Furthermore, these patients often have smile asymmetry and restriction due to disruption of normal vector forces on the modiolus. Interestingly, investigators have demonstrated that smiles of NFFP patients convey much higher levels of disgust and contempt than flaccid or normal faces making the same expressions.¹⁸

Management of NFFP

Historical treatments

For debilitating oculo-oral and midfacial synkinesis, Guerrissi described resection of the zygomaticus major

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and minor muscles.¹⁹ More recently, Chuang described radical resection of the cheek and platysma muscles along with the zygomatic, buccal, and cervical branches of the facial nerve to treat patients with severe synkinesis. He reconstructed these patients with gracilis muscle transfer.²⁰ These radical myectomy procedures may carry significant morbidity. Peripheral facial neurectomy has also been used for patients with NFFP. Hohman et al. described highly selective neurectomy for refractory periocular synkinesis in 2013 and found improvement in postoperative palpebral fissure width without lagophthalmos, ectropion, or eye dryness.²¹ However, long-term follow-up demonstrated shortcomings of the original surgical approach.²²

Facial rehabilitation therapy

Targeted facial neuromuscular therapy has gained widespread acceptance as a noninvasive initial treatment for NFFP.^{23–25} A large review demonstrated targeted facial rehabilitation therapy results in quantitative improvement in facial function scores using the facial grading scale in patients with NFFP.²⁵ Coulson et al. demonstrated that targeted facial rehabilitation therapy in conjunction with video self-modeling improves patient quality of life (QOL), smile symmetry, and facial movement control in a prospective blinded manner.²⁶ Others have repeatedly shown how targeted facial therapy improves facial symmetry and patient QOL.^{27,28}

Botulinum toxin therapy

Chemodenervation using botulinum toxin (e.g., Botox, Dysport, Myobloc, and Xeomin) has become the mainstay of treatment for improving synkinesis, hypertonicity, and facial symmetry in patients with NFFP. Numerous studies have demonstrated the ability of botulinum toxin to treat synkinesis and hypertonicity in patients with NFFP.^{9,10,29,30} Chemodenervation for NFFP is associated with improved clinician-graded assessment of facial palsy severity and patient-reported QOL scores.^{31,32} Not only can botulinum toxin be used to improve hypertonicity and synkinesis, but it can also be administered to musculature on the healthy side to improve symmetry.^{33,34} It can also be injected into the depressor anguli oris (DAO) muscle and buccinator to release patients' smiles.^{35,36}

Current surgical therapies for hypertonicity and synkinesis

Although facial rehabilitation therapy and chemodenervation remain important treatment strategies for NFFP, emerging surgical therapies are broadening contemporary NFFP management. Knowledge gained from chemodenervation is informing the development of pioneering

selective myectomies and neurectomies. Surgeons have also begun to employ procedures formerly restricted to patients with FFP to improve the smiles of patients with NFFP.

Myectomy

Selective myectomy procedures are relatively low risk, can relieve neck tightness, and improve smile symmetry in patients, and can often be performed under local anesthesia. If patients demonstrate a favorable response to local anesthesia and/or botulinum toxin administration in the healthy-side depressor labii inferioris (DLI) muscle, it can be excised, leading to a permanent improvement in lower lip symmetry.^{37,38} Henstrom et al. demonstrated that platysmectomy under local anesthesia leads to improved QOL in patients with platysmal synkinesis.³⁹ Weakening the affected-side DAO using local anesthesia leads to improvement in oral commissure excursion, smile angle, and ability to express positive emotions with smile such as joy and happiness.⁴⁰ Labbe et al. have reported successful surgical resection of this muscle in a small number of patients.³⁵

Neurectomy

Although myectomy procedures target the dysfunctional end organs in NFFP, the aberrantly regenerated nerves can also be eliminated to relieve hyperactive and synkinetic facial muscles. In 2012, Terzis and Karypidis briefly mentioned selective neurectomy in their review of NFFP treatments, and then in 2018, Azizzadeh and Frisenda published their surgical technique for modified selective neurectomy.^{41,42} Azizzadeh et al. subsequently demonstrated that modified selective neurectomy leads to improved eFACE scores and decreased Botox requirement, although there is a risk of temporary oral incompetence and need for revision procedures.⁴³

Upregulation of smile

In patients who fail to achieve improved smiles after decreasing the activity of smile antagonists through myectomy and neurectomy procedures, augmentation of the smile elevators can be performed. Cross-face nerve grafting (CFNG) has been used to “supercharge” existing musculature, though no large study has convincingly demonstrated improvements in smile excursion using this approach.⁴⁴ Banks et al. demonstrated a low success rate for masseteric-facial nerve transfer in improving smile excursion in patients with NFFP.⁴⁵ Recently though, Vincent et al. reported improved eFACE scores in patients who underwent masseteric-facial nerve transfer simultaneously with selective neurectomy.⁴⁶ Further study will determine whether simultaneous selective

denervation can eliminate zygomaticus major synkinesis, making the 5–7 nerve transfer procedure more effective in patients with NFFP. Ultimately, if nerve transfers are unlikely to sufficiently augment smile elevators, Lindsay et al. demonstrated that gracilis muscle transfer improves QOL, facial function, and smile symmetry when used for reanimation in patients with NFFP.¹⁶

Treatment Ladder for NFFP

Despite significant advances in therapies for NFFP, there is no widely accepted treatment algorithm. Our approach to managing ~5000 patients with facial palsy over the past several decades has evolved; our most contemporary strategy is highlighted conceptually as an NFFP treatment ladder (Fig. 1).

The reconstructive ladder, a term familiar to all reconstructive surgeons, serves as a framework for decision-making when approaching surgical defects.⁴⁷ The fundamental principal is that one should consider the simplest procedure that will effectively address the problem. Subsequent modifications have emphasized how surgeon experience, technical advancements, and patient risk must also be considered.^{48,49} Similar to the reconstructive ladder, the NFFP treatment ladder provides a framework for clinicians treating patients with NFFP, but does not represent a rigid structure; each patient must be evaluated in the context of his or her diagnosis, examination, comorbidities, and goals for treatment.

Our algorithm begins with targeted facial rehabilitation, as long-lasting improvement in facial function has been demonstrated in a large review, and it has minimal

risk as long as patients are able and willing to meet with a therapist and perform at-home exercises.²⁵ Furthermore, targeted physical therapy works synergistically with chemodenervation and surgical therapies to improve outcomes. We re-evaluate patients 3–4 months after their first facial rehabilitation session for initiation of chemodenervation therapy with botulinum toxin if necessary. Initiating facial rehabilitation before chemodenervation allows patients to understand the different facial muscles, their own synkinesis patterns, and develop skills to isolate and selectively downregulate certain muscles, before chemically weakening them.

Botulinum toxin therapy is initiated to improve hypertonia, synkinesis, and asymmetry, and to inform subsequent surgical procedures. Similarly, injection of local anesthetic agents can be used to weaken the affected-side DAO and/or healthy-side DLI muscles to demonstrate the potential benefits of chemodenervation and myectomy.^{40,50} If patients demonstrate hyperactivity of their buccinator and/or mentalis on the affected side, they can also be transiently weakened to observe the effects on smile (Fig. 2). We administer a total of 1–3 cc of 1% lidocaine with 1:100,000 epinephrine during a weakening trial.

In our experience, muscle excision has the potential to provide greater improvements in smile excursion, dental show, and smile angle than the administration of local anesthetic or botulinum toxin (unpublished data). We typically perform excision of the affected-side DAO muscle (including excision of a segment of buccinator muscle) and/or the healthy-side DLI muscle under local

Fig. 1. NFFP treatment ladder. NFFP, nonflaccid facial palsy.

Fig. 2. (a) Patient with left NFFP before local anesthetic injection. (b) Patient with left NFFP after 3 mL 1% lidocaine with 1:100,000 epinephrine injection into the right DLI, left DAO, left mentalis, and left buccinator (intraoral injection) muscles. DAO, depressor anguli oris; DLI, depressor labii inferioris; NFFP, nonflaccid facial palsy.

anesthesia (Fig. 3). The risks of oral incompetence and articulation issues are minimal with targeted muscle excision, yet the benefits can be significant.^{37,38}

If targeted myectomy does not provide significant smile improvement or patients are affected by facial tightness, selective denervation may be an excellent option. Azizzadeh demonstrated that selective denervation

Fig. 3. (a) Patient with left NFFP before left DAO resection. (b) Patient with left NFFP after left DAO resection.

Fig. 4. (a) Patient with left NFFP before selective denervation. (b) Patient with left NFFP after selective denervation.

results in improved clinician-graded facial symmetry using the eFACE. Patients also had significantly less mentalis, mifacial, and platysmal synkinesis after selective neurectomy.⁴³ We have also found that selective denervation leads to improved QOL for patients as determined by the Facial Clinimetric Evaluation Scale (Fig. 4, unpublished data). However, risks of oral incompetence, articulation difficulties, and need for revision procedures should be discussed with patients before performing this procedure. If clinical circumstances warrant (NFFP with significant superimposed hypotonicity in zygomaticus muscles), we place a CFNG at the time of denervation, in preparation for a CFNG-driven or dual innervation gracilis muscle transfer, in case denervation fails to improve smiling.

In some patients with hypotonic NFFP, weakening smile antagonists using chemodenervation, muscle excision, and/or denervation are inadequate to achieve a satisfactory smile. In these cases, augmentation of the smile elevators is required to improve smile symmetry. Some have advocated “supercharging” the existing musculature with CFNG, although no large study has convincingly demonstrated improvements in smile excursion using this approach.⁴⁴ Although Vincent et al. recently published their success in masseteric-facial nerve transfer in conjunction with selective denervation, their cohort was small and further study will elucidate whether selective denervation can treat the synkinetic zygomaticus major muscle that likely limits isolated masseteric-facial nerve transfer in patients with NFFP.⁴⁶ Gracilis muscle transfer in patients has been shown to improve smile excursion, smile symmetry, and QOL in patients with NFFP, and may represent a viable option for patients wherein less invasive procedures have not produced a satisfactory smile.¹⁶

Conclusions

Patients with NFFP may suffer significant functional, aesthetic, psychological, and social impairments. Similar to other treatments in medicine, the principles of maximizing patient benefit while minimizing risk should be respected when treating patients with NFFP. We propose the NFFP treatment ladder as a framework to use when evaluating and planning treatments for patients with NFFP. Although other factors such as surgeon experience, patient goals, and patient history and examination should ultimately determine the best treatment for patients, the NFFP treatment ladder encompasses the spectrum of treatments for NFFP and suggests potential treatments to optimize patient outcomes while minimizing risk.

Authors' Contributions

M.Q.M. and T.A.H. contributed to the design of this study, drafting of the article, and critical revisions. Both have approved the final version of the article for publication.

Author Disclosure Statement

No competing financial interests exist.

Funding Information

No funding was received for this article.

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