

Modified Selective Neurectomy for the Treatment of Post-Facial Paralysis Synkinesis

Babak Azizzadeh, M.D.
 Leslie E. Irvine, M.D.
 Jaqueline Diels, O.T.
 William H. Slattery, M.D.
 Guy G. Massry, M.D.
 Babak Larian, M.D.
 Kiersten L. Riedler, M.D.
 Grace Lee Peng, M.D.

Los Angeles, Beverly Hills, and Santa Barbara, Calif.; and Madison, Wis.



Background: To address functional and smile dysfunction associated with post-facial paralysis synkinesis, the senior author (B.A.) has offered “modified selective neurectomy” of the lower division of the facial nerve as a long-term solution. This article examines technical considerations and outcomes of this procedure.

Methods: A retrospective review was conducted of patients who underwent modified selective neurectomy of buccal and cervical branches of the facial nerve performed by a single surgeon over a 4½-year period. House-Brackmann facial grading scores, electronic clinician-graded facial function scale, and onabotulinumtoxinA (botulinum toxin type A) dosages were examined before and after the procedure.

Results: Sixty-three patients underwent modified selective neurectomy between June 20, 2013, and August 12, 2017. There were no serious complications. The revision rate was 17 percent. Temporary oral incompetence was reported in seven patients (11 percent) postoperatively. A statistically significant improvement was achieved in electronic clinician-graded facial function scale analysis of nasolabial fold depth at rest, oral commissure movement with smile, nasolabial fold orientation with smile, nasolabial depth with smile, depressor labii inferioris lower lip movement, midfacial synkinesis, mentalis synkinesis, platysmal synkinesis, static score, dynamic score, synkinesis score, periocular score, lower face and neck score, and midface and smile score. There was a significant decrease in botulinum toxin type A dosage and House-Brackmann score after surgery.

Conclusion: Modified selective neurectomy of the buccal and cervical divisions of the facial nerve is an effective long-term treatment for smile dysfunction in patients with post-facial paralysis synkinesis. (*Plast. Reconstr. Surg.* 143: 1483, 2019.)

CLINICAL QUESTION/LEVEL OF EVIDENCE: Therapeutic, IV.

Components of a natural smile include symmetric superolateral excursion of the oral commissure, appropriate orientation of the

From the Department of Head and Neck Surgery, David Geffen School of Medicine at the University of California, Los Angeles; the Center for Advanced Facial Plastic Surgery; the Santa Barbara Plastic Surgery Center; the Facial Nerve Clinic, University of Wisconsin Hospitals and Clinics; the Department of Neurology, House Clinic; the Department of Otolaryngology, University of Southern California, and Beverly Hills Ophthalmic Plastic and Reconstructive Surgery; Division of Oculoplastic Surgery, Department of Ophthalmology, and Division of Facial Plastic and Reconstructive Surgery, Department of Otolaryngology–Head and Neck Surgery, Keck School of Medicine, University of Southern California.

Received for publication January 10, 2018; accepted September 27, 2018.

Copyright © 2019 by the American Society of Plastic Surgeons

DOI: 10.1097/PRS.0000000000005590

Disclosure: Drs. Massry and Azizzadeh receive royalties from Elsevier and Springer. Dr. Azizzadeh was a co-investigator for the Checkpoint head and neck nerve stimulator/locator product validation study (2004). The other authors have no financial interests or conflicts of interest to disclose. No funding was received for this article.

Supplemental digital content is available for this article. Direct URL citations appear in the text; simply type the URL address into any Web browser to access this content. Clickable links to the material are provided in the HTML text of this article on the *Journal's* website (www.PRSJournal.com).

nasolabial fold, and symmetrical upper and lower teeth show. Spontaneity and simultaneous timing between both sides of the face have paramount importance.^{1,2}

Most facial nerve disorders present initially with flaccid and complete paralysis. Depending on the cause, the majority of patients experience partial or complete recovery. Any form of facial nerve repair (neurorrhaphy, cable nerve grafting), incomplete nerve injury (e.g., Bell palsy, Ramsay Hunt syndrome, acoustic neuroma, temporal bone fracture), or cranial nerve substitution technique (e.g., hypoglossal- or masseteric-to-facial nerve transfer) can lead to synkinesis³⁻⁷ (Fig. 1). The most supported theory of post-facial paralysis synkinesis is aberrant nerve regeneration where proximal axons reroute, sprout, and/or degenerate, leading to abnormal reinnervation of both correct and inappropriate muscles^{3,7-12} (Fig. 2). Simultaneous triggering of the orbicularis oris, platysma, depressor anguli oris, and buccinator muscles resists appropriate activation of key smile muscles such as zygomatic major/minor, levator labii/anguli, and depressor labii inferioris muscles, leading to an inferior and lateral vector of pull on the oral commissure and decreased upper and lower teeth show (Fig. 1).

The ideal facial reanimation technique should improve spontaneous smile mechanism, symmetry of upper and lower dental show, and oral competency.¹²⁻²⁷ This study describes the senior author's (B.A.) current reanimation technique referred to herein forth as "modified selective neurectomy" for patients with post-facial paralysis synkinesis.

PATIENTS AND METHODS

Clinical Indication for Modified Selective Neurectomy

Modified selective neurectomy of the facial nerve is offered to patients who are clinically determined to have synkinesis, active zygomatic major/minor, and access to the distal branches of the facial nerve. Patients with complete flaccid paralysis and malignancies are not candidates for modified selective neurectomy. Patients in the early phase of nerve regeneration are observed until there is a stable pattern of synkinesis. Early in the senior author's experience, patients were also offered simultaneous cross-facial nerve grafting in preparation for a secondary gracilis muscle transfer in case the outcome of the modified selective neurectomy was not satisfactory.

Surgical Procedure

Modified selective neurectomy is performed on an outpatient basis under general anesthesia. A standard rhytidectomy incision is used. Lidocaine hydrochloride is not used, to avoid inadvertent paresis of the facial nerve.

Facial nerve monitoring electrodes (Medtronic, Goleta, Calif.) are placed. The skin flap is elevated for approximately 5 cm. The superficial muscular aponeurotic system (SMAS) is then incised in an oblique vector from the midportion of the zygomatic arch extending inferiorly past the angle of the mandible as it transitions to the platysma (Fig. 3). After the sub-SMAS plane is entered, the dissection continues bluntly on top of the



Fig. 1. (Left) A 28-year-old man with a history of temporal bone fracture with complete right facial paralysis. (Right) Postoperative photograph after hypoglossal-to-facial nerve transfer with right postparalysis synkinesis. Note that there is improved nasolabial fold and tone; however, the patient has facial asymmetry with a "frozen" smile and asymmetric upper and lower teeth show. (Courtesy Facial Paralysis Institute.)

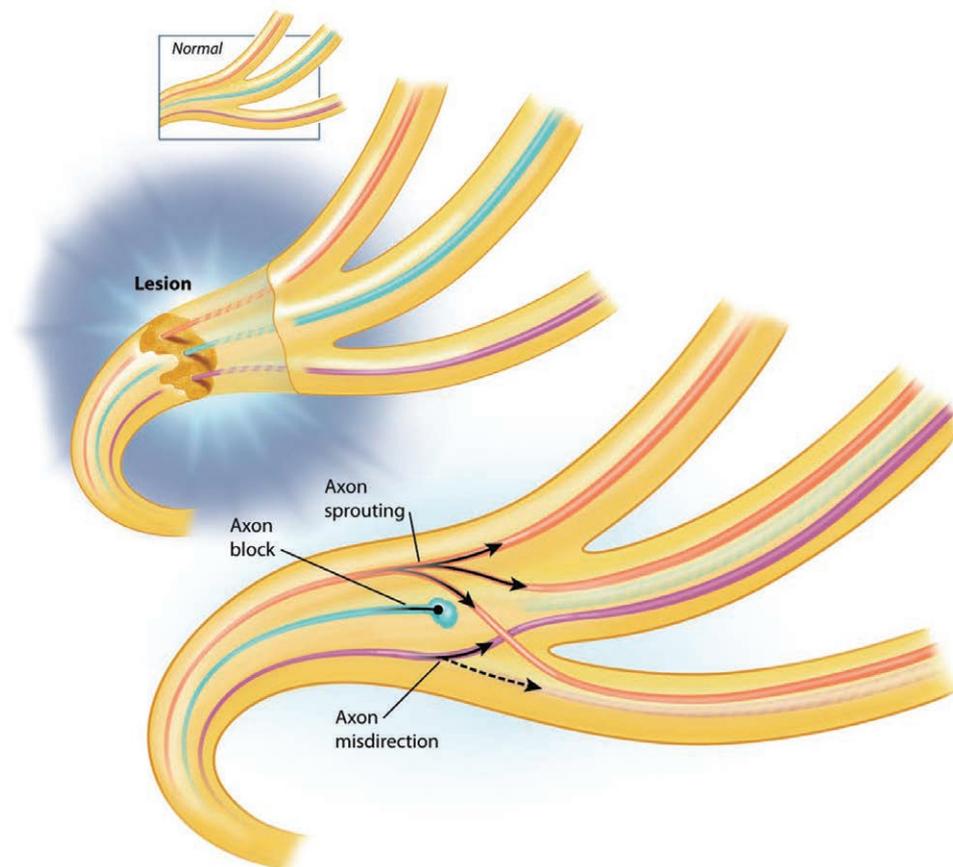


Fig. 2. Aberrant regeneration theory of synkinesis. After nerve injury, proximal axons reroute, sprout along multiple distal pathways, and/or degenerate, leading to reinnervation of both correct and incorrect muscles. (Courtesy Facial Paralysis Institute.)

zygomatic muscle and masseteric fascia. Using a nerve stimulator (Checkpoint Surgical, Cleveland, Ohio), the zygomatic, buccal, cervical, and marginal mandibular nerve branches are identified deep to the masseteric fascia and on the undersurface of the platysma as they exit the parotid capsule.¹⁹ The nerves are stimulated (0.5 to 2 mA) to evaluate the elicited movement. Video documentation is performed. [See Video, Supplemental Digital Content 1, which demonstrates a patient undergoing right modified selective neurectomy. The video demonstrates intraoperative nerve stimulation with split face showing the elicited facial movement. The lower buccal, cervical, and marginal mandibular nerves have been identified in this video. The first facial movement is elicited by stimulating a lower buccal nerve, which elevates the lower lip and depresses the upper lip with lateral oral commissure movement. This nerve is a candidate for neurectomy. The second stimulation is a distal branch that mobilizes the mentalis. This nerve will be sacrificed. The third nerve is another buccal branch that also elevates the lower lip and

depresses the upper lip, thereby counteracting a wide smile. The last split screen shows the depressor labii inferioris activation from stimulation of the marginal mandibular nerve. This nerve will be preserved, <http://links.lww.com/PRS/D438>. (Courtesy Facial Paralysis Institute.)]

Branches that activate the depressor labii inferioris and purely elevate the modiolus and upper lip are preserved. Nerve branches that stimulate the platysma, elevate the lower lip, or cause lateral and downward movement to the oral commissure are candidates for transection (0.5- to 4-cm segment). By design, the facial nerve dissection is not performed in the body of the parotid at the level of the stylomastoid foramen to limit the risk of completely denervating the lower face. Not all the peripheral branches can be feasibly isolated. Branches that activate both the elevators and depressors at the junction of buccal and zygomatic branches may be preserved to maintain neural input. If the patients do not achieve an ideal outcome from the procedure, revision surgery is offered, at which time these watershed



Video 1. Supplemental Digital Content 1 demonstrates a patient undergoing right modified selective neurectomy. The video demonstrates intraoperative nerve stimulation with split face showing the elicited facial movement. The lower buccal, cervical, and marginal mandibular nerves have been identified in this video. The first facial movement is elicited by stimulating a lower buccal nerve, which elevates the lower lip and depresses the upper lip with lateral oral commissure movement. This nerve is a candidate for neurectomy. The second stimulation is a distal branch that mobilizes the mentalis. This nerve will be sacrificed. The third nerve is another buccal branch that also elevates the lower lip and depresses the upper lip, thereby counteracting a wide smile. The last split screen shows the depressor labii inferioris activation from stimulation of the marginal mandibular nerve. This nerve will be preserved, <http://links.lww.com/PRS/D438>. (Courtesy Facial Paralysis Institute.)

branches are often transected. In addition, other buccal and cervical peripheral nerve branches that were not identified in the initial surgery need to be uncovered and transected. Occasionally, the transected buccal branch is rerouted to the zygomatic major/minor (direct muscle neurotization) or zygomatic branch (end-to-side coaptation) to increase neural input into the smile elevators. This approach is used mainly for patients who intraoperatively do not demonstrate adequate superior excursion of the modiolus with direct nerve stimulation.²⁰ Platysmal myotomy is performed from the lateral edge of the platysma to the medial border approximately 3 cm below the border of the mandible, avoiding the marginal mandibular nerve (Fig. 3, *below*).

At the conclusion, the SMAS is returned to its native position. If the patient is undergoing a simultaneous rhytidectomy, the SMAS is suspended in the appropriate superolateral vector and a deep plane rhytidectomy is performed on the contralateral side.

Patients usually return to social function in 7 to 14 days and may obtain botulinum toxin type A as needed. Neuromuscular retraining may begin

1 month postoperatively, with the goal of reestablishing and coordinating the facial muscle activity and reducing hypercontracted zygomatic major/minor and levator labii/anguli muscles.^{14,21}

Study Design

All patients in this study were from the private practice of the senior author (B.A.). No patients or patient records included in this report were associated with any academic institutions at the time of the study; thus, institutional review board approval was not obtained. Written informed consent was obtained for each procedure from all patients, and the present review adheres to the standards of the Declaration of Helsinki and complies with the Health Insurance Portability and Accountability Act of 1996.

Medical records of patients who underwent modified selective neurectomy between June 20, 2013, and August 12, 2017, were reviewed retrospectively. The subset of patients who concurrently underwent rhytidectomy and direct zygomatic major/minor neurotization or end-to-side nerve coaptation to the zygomatic branch by a cross-facial nerve graft or the proximal portion

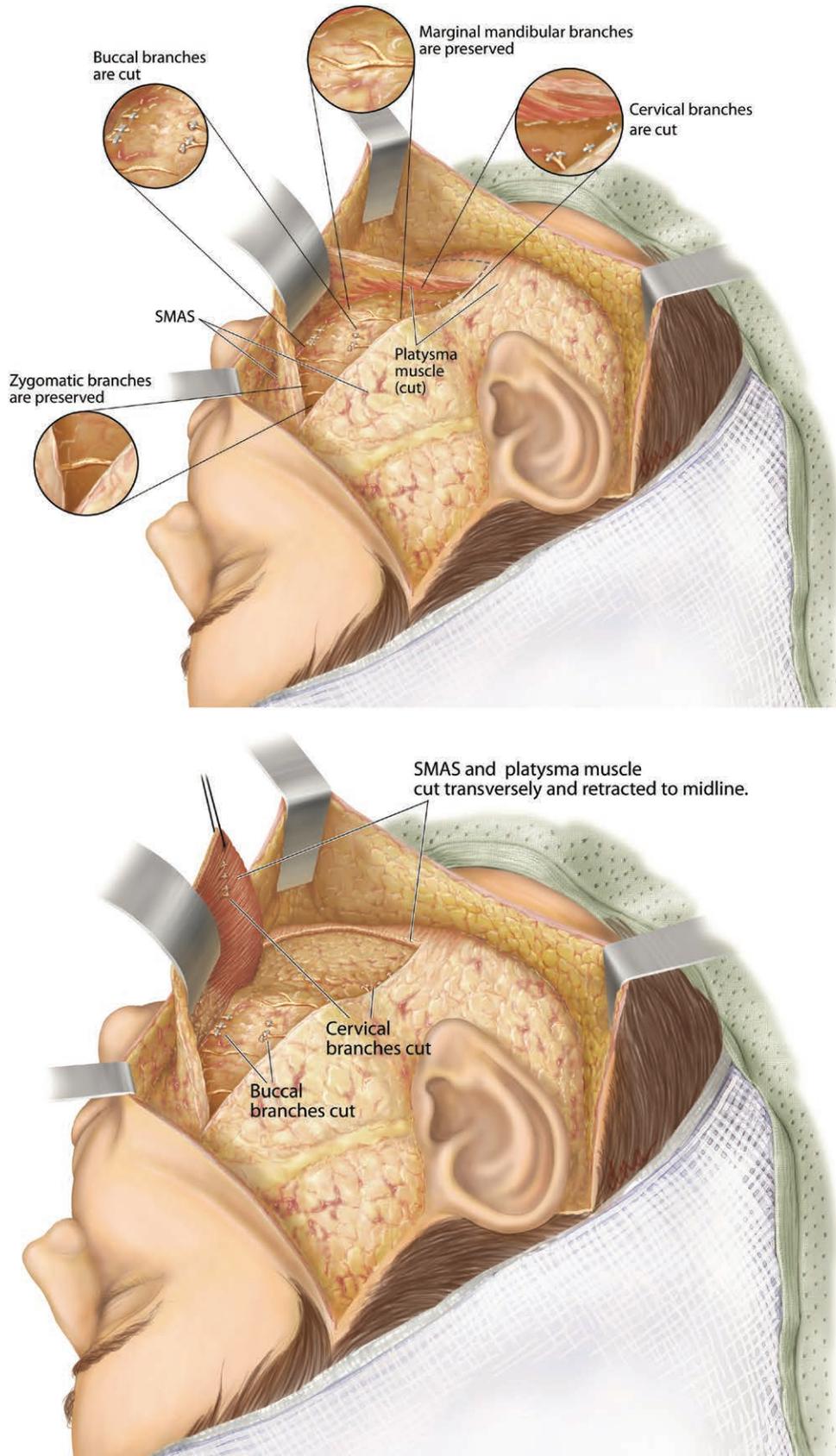


Fig. 3. (Above) Selective neurectomy surgery. After entering the plane deep to the SMAS and platysma muscle, multiple distal nerves are identified and tagged. Stimulation of each nerve is

of a transected ipsilateral buccal branch were included in the study.²² Patients who had gracilis free functional transfer, masseteric-to-facial nerve transfer, or temporalis myoplasty were excluded. Patients who also had selective neurectomy of the contralateral marginal mandibular nerve for the management of congenital unilateral lower lip palsy were excluded.

Patients were required to avoid botulinum toxin type A injections for at least 6 months before the procedure.²³ If patients had been treated with botulinum toxin type A before that time and postoperatively at the Facial Paralysis Institute, the total quantity administered to the entire face before and after the procedure was recorded and analyzed. House-Brackmann scores were recorded by the senior author clinically and by an independent physician observer if consistent preoperative and postoperative photographs were available. The Wilcoxon signed rank test was used for analysis.

Electronic Clinician-Graded Facial Function Scale

In this study, the electronic clinician-graded facial function scale was used for objective smile and synkinesis analysis. The electronic clinician-graded facial function scale application is a digital facial nerve grading system used to analyze 16 important qualities of facial function.^{24,25} Studies have shown excellent interrater and intrarater reliability validated by worldwide facial nerve experts.^{24,25}

Patients were included in the electronic clinician-graded facial function scale analysis if they had consistent preoperative and postoperative photographs taken at the Facial Paralysis Institute. Only patients with at least 3 months of follow-up were included in the electronic clinician-graded facial function scale analysis. Patients who underwent postoperative botulinum toxin type A injection were excluded if it had been administered within a 3-month period of the electronic clinician-graded facial function scale analysis.

Fig. 1. (Continued). performed with video documentation, and cervical and buccal branches that cause downward or lateral excursion of the oral commissure and upper lip are transected. The marginal mandibular branches are identified and preserved along with zygomatic branches. (*Below*) Platysma myotomy is performed from the lateral edge of the platysma all the way to the medial border approximately 3 cm below the border of the mandible with sharp scissors, taking care not to injure the marginal mandibular nerve. (Courtesy Facial Paralysis Institute.)

An independent physician observer not involved in any of the procedures or perioperative management of the patients was familiarized with the electronic clinician-graded facial function scale application. Each patient was evaluated using an iPad (Apple, Inc., Cupertino, Calif.). Preoperative and postoperative scores for each category were compared and a Wilcoxon signed rank test was used to determine significance. Subgroup analyses were performed on the electronic clinician-graded facial function scale data, and an unpaired nonparametric two-tailed Mann-Whitney was used to determine significant differences between subgroups in each electronic clinician-graded facial function scale parameter.

RESULTS

Between June 20, 2013, and August 12, 2017, 65 patients underwent modified selective neurectomy at the Facial Paralysis Institute. Two patients (3 percent) subsequently underwent gracilis free flap and were excluded from the study. Of the 63 included patients, there were 52 women and 11 men. The average age of the patients was 46 years (range, 18 to 71 years). Twenty-eight patients had right and 33 had left post-facial paralysis synkinesis. Two patients had bilateral paralysis. Forty-five patients (71 percent) had a history of Bell palsy. Other causes included Ramsay Hunt syndrome [eight patients (13 percent)], trauma [six patients (10 percent)], benign tumors [three patients (5 percent)], and congenital [one patient (2 percent)]. The average time between onset of facial paralysis and surgery was 9 years (range, 1 to 41 years). The average number of peripheral facial neurectomies was 6.9 (range, two to 14 nerves). The median number of transected nerves was six.

Thirty-five patients (56 percent) had a simultaneous rhytidectomy. Seven patients (11 percent) had cross-facial nerve grafting performed simultaneously. Ten patients (16 percent) had rerouting of the transected ipsilateral buccal branch to the zygomatic branch in an end-to-side manner, and seven patients (11 percent) had rerouting of the buccal branch by means of direct muscle neurotization of the zygomatic major/minor.

There were no serious complications. Three patients (4.8 percent) developed hematoma and one patient (1.6 percent) had a seroma. All were drained in the office and resolved without further sequelae. Two patients (3 percent) had hypertrophic scarring treated conservatively. The mean follow-up period from the time of surgery to the

latest follow-up visit was 373 days (range, 6 to 1377 days). Eight patients (12.7 percent) required two procedures to obtain the desired outcome, and at the time of the data analysis, three patients were planning to undergo revision surgery, for a total revision rate of 17 percent.

Seven patients (11 percent) had temporary worsening of oral incompetence postoperatively. Four patients had mild and three had moderate oral incompetence, as rated by the senior author. Duration of oral incompetence ranged from 7 days to 8 months. Two patients were lost to follow-up. The average age of those with oral incompetence was 58 years (range, 43 to 71 years). The average number of nerves cut in the group with oral incompetence was 8.7, compared to an average of 6.7 nerves cut in the 56 patients who did not report worsening postoperative oral incompetence. Overall, 61 of 62 patients (98 percent) reported satisfaction with the procedure at their last recorded postoperative visit.

House-Brackmann

The average House-Brackmann facial grading scale score as rated by an independent observer improved from 3.9 to 2.8 ($p < 0.001$), with an average follow-up of 370 days (range, 6 to 1377 days). The average House-Brackmann facial grading scale score as rated by the senior author improved from 3.7 to 2.6 ($p < 0.001$).

There was no significant difference in the average preoperative and postoperative House-Brackmann scores between the 31 patients who had greater than the median of six nerve branches transected and 32 patients who had six or fewer nerves transected ($p = 0.5$). There was also no significant difference between the patients with and without face lift ($p = 0.3$), cross-facial nerve grafting ($p = 0.06$), or nerve rerouting ($p = 0.6$). Lastly, there was no statistical difference between the seven patients who had nerve rerouting to the zygomatic major/minor and the 10 patients who had end-to-side neural rerouting ($p = 0.3$).

Botulinum Toxin Type A

Forty-five patients (71 percent) received documented botulinum toxin type A at some point before surgery. Twenty-one patients (33.3 percent) had both preoperative and postoperative botulinum toxin type A at the Facial Paralysis Institute. In this cohort, the average quantity of botulinum toxin type A administered to the entire face decreased significantly from 64 to 34 units ($p = 0.02$).

Electronic Clinician-Graded Facial Function Scale Analysis

Forty-six patients (73 percent) had satisfactory photographic documentation and were included in the electronic clinician-graded facial function scale analysis. There were 38 women and eight men in this subgroup analysis. The mean age of patients included in this analysis was 47 years (range, 18 to 70 years). The average time from surgery to follow-up electronic clinician-graded facial function scale analysis was 499 days (range, 95 to 1509 days). A statistically significant improvement was achieved in oral commissure position at rest ($p = 0.04$), nasolabial fold depth at rest ($p = 0.01$), oral commissure movement with smile ($p < 0.001$), nasolabial fold orientation with smile ($p = 0.01$), nasolabial fold depth with smile ($p = 0.004$), depressor labii inferioris lower lip movement ($p = 0.01$), ocular synkinesis ($p = 0.03$), midfacial synkinesis ($p < 0.001$), mentalis synkinesis ($p < 0.001$), platysmal synkinesis ($p < 0.001$), static score ($p = 0.002$), dynamic score ($p < 0.001$), synkinesis score ($p < 0.001$), lower face and neck score ($p < 0.001$), midface and smile score ($p < 0.001$), smile score ($p < 0.001$), and periocular score ($p < 0.001$) (Table 1).

In the subgroup analyses, there was no significant difference in any of the electronic clinician-graded facial function scale parameters between the seven patients who had simultaneous cross-facial nerve grafting and the 39 who did not, between the six patients who had nerve rerouting and the six who had rerouting by direct zygomatic major/minor neurotization, between the 25 patients who had rhytidectomy and the 21 who did not, or between the 23 patients who had fewer than the median of six nerves transected and the 20 patients who had more than six neurectomies.

Of the 46 patients included in electronic clinician-graded facial function scale analysis, 22 (48 percent) had more than 1 year of follow-up (average, 786 days; range, 375 to 1509 days). There was significant improvement in all electronic clinician-graded facial function scale categories except nasolabial fold depth/orientation and ocular synkinesis (Table 2). Thirty-eight of 46 electronic clinician-graded facial function scale patients (79 percent) had a viral cause for their paralysis. In this group, the average time to follow-up was 469 days (range, 95 to 1509 days). All electronic clinician-graded facial function scale categories except depressor labii inferioris lower lip movement ($p = 0.08$) and oral commissure position at rest ($p = 0.27$) improved significantly in this group.

Table 1. Electronic Clinician-Graded Facial Function Scale Results

eFACE Parameter	Average Preoperatively	Average Postoperatively	<i>p</i>
Nasolabial fold depth at rest	111	106	0.01
Oral commissure position at rest	95	96	0.04
Oral commissure movement with smile	43	70	<0.001
Nasolabial fold orientation with smile	80	92	<0.001
Nasolabial fold depth with smile	95	96	0.006
DLI lower lip movement with EEEE	66	79	0.01
Midfacial synkinesis	80	93	<0.001
Mentalis synkinesis	79	93	<0.001
Platysmal synkinesis	82	99	<0.001
Static score	91	94	0.002
Dynamic score	73	84	<0.001
Synkinesis score	76	89	<0.001
Lower face and neck score	76	90	<0.001
Midface and smile score	76	86	<0.001
Smile score	79	87	<0.001
Ocular synkinesis	63	70	0.03
Periocular score	83	88	<0.001

eFACE, electronic clinician-graded facial function scale; DLI, depressor labii inferioris.

Of the original 46 electronic clinician-graded facial function scale patients, 26 did not have any other dynamic procedures such as cross-facial nerve grafting or rerouting. Average time to follow-up in this group was 478 days (range, 95 to 1509 days). All electronic clinician-graded facial function scale parameters improved significantly, except nasolabial fold depth with smile, ocular synkinesis, depressor labii inferioris lower lip movement, and oral commissure position at rest.

DISCUSSION

Historically, facial nerve experts have had diverse management protocols for patients with post-facial paralysis synkinesis.¹³⁻¹⁷ The current

treatment options include observation, physiotherapy, botulinum toxin type A, and procedures such as free functional muscle transfer and temporalis myoplasty to increase superior excursion forces on the oral commissure.

Because patients with synkinesis have innervated facial musculature, the treatment philosophy should be different than those who have long-term flaccid paralysis without any functioning muscles. Treatments that can reduce but not completely eliminate the activity of the depressor anguli oris, platysma, orbicularis oris, and buccinator while preserving smile elevators and the depressor labii inferioris should enhance the smile mechanism and dental show. Powering or augmenting the elevators—which is the basis of

Table 2. Electronic Clinician-Graded Facial Function Scale Subgroup Results

eFACE Parameter	>1 Year Follow-Up (<i>n</i> = 22) <i>p</i>	No CFNG or Rerouting (<i>n</i> = 26) <i>p</i>	<i>p</i>	
			Viral Cause (<i>n</i> = 38)	Viral Cause, No CFNG Rerouting (<i>n</i> = 20)
Nasolabial fold depth at rest	0.002	0.005	0.02	0.007
Oral commissure position at rest	0.009	0.27	0.27	0.95
Oral commissure movement with smile	0.04	0.03	0.001	0.4
Nasolabial fold orientation with smile	0.17	0.03	0.005	0.25
Nasolabial fold depth with smile	0.6	0.38	0.008	0.48
DLI lower lip movement with EEEE	0.04	0.49	0.08	2
Midfacial synkinesis	0.02	0.04	0.005	0.19
Mentalis synkinesis	<0.001	<0.001	<0.001	0.007
Platysmal synkinesis	0.001	<0.001	<0.001	0.002
Static score	0.002	0.008	0.03	0.04
Dynamic score	0.004	0.008	<0.001	0.13
Synkinesis score	<0.001	<0.001	<0.001	0.002
Lower face and neck score	0.003	0.003	<0.001	0.02
Midface and smile score	0.006	0.03	0.002	0.24
Smile score	0.003	0.02	<0.001	0.13
Ocular synkinesis	0.8	0.3	0.002	0.07
Periocular score	0.003	0.02	<0.001	0.07

eFACE, electronic clinician-graded facial function scale; CFNG, cross-facial nerve grafting; DLI, depressor labii inferioris.

almost all facial reanimation procedures—may not be necessary.

Because the peripheral nerve branches are not anatomically distorted in most patients with post-facial paralysis synkinesis, the root cause of synkinetic patients can potentially be addressed by directly reducing the neural input of the counterproductive muscles. If the marginal mandibular nerve and zygomatic branches are preserved, the oral region will be able to function more normally while maintaining adequate neural input to avoid serious consequences of denervation.

In 1937, Coleman first described neurectomy of the trunk of the facial nerve for hemifacial spasm.²⁶ Greenwood in 1946 advocated partial neurectomy of the intraparotid or postparotid branches.²⁷ In 1950, Marino and Alurralde performed peripheral selective neurectomy for spastic facial palsy.²⁸ In the upper face, multiple reports of selective neurectomy for blepharospasm have been reported.^{29,30}

Myectomy of the zygomatic major/minor has also been advocated for treatment of midface synkinesis.³¹ Neurectomy of the cervical branches of the facial nerve has been described to specifically improve synkinetic platysmal banding.^{32,33} Hestrom et al. also described the use of platysmectomy for the treatment of platysmal hypertrophy.³⁴ Comprehensive myectomy of synkinetic facial muscles along with transection of peripheral branches of the facial nerve followed by reconstruction with a gracilis muscle flap innervated by the masseteric or spinal accessory nerve was described by Chuang et al. for patients with synkinesis.³⁵ None of these studies included a smile analysis of the neurectomy and/or myectomy alone. In 2012, Terzis and Karypidis discussed selective neurectomy as one of many strategies for post-facial paralysis synkinesis, but it was performed on only six patients with minor synkinesis, and the surgical details were not outlined.¹⁵

Modified selective neurectomy is the first peripheral facial neurectomy technique to specifically improve smile function in a large group of post-facial paralysis synkinesis patients.^{15,26–35} We found objective improvement (electronic clinician-graded facial function scale) in lower facial synkinesis and in smile mechanism. The key differentiating factors in the modified selective neurectomy versus other neurectomy procedures described in the literature include identification of a significant number of peripheral branches, preservation of the marginal mandibular nerve, and ablation of multiple buccal and cervical branches that cause inferior and lateral excursion of the oral commissure. Modified selective

neurectomy as described in this study unlocks the patient's natural smile mechanism by reducing the activity of antagonistic muscles while preserving the neural input into the key smile muscles.

Modified selective neurectomy achieves results rarely seen in traditional facial reanimation procedures by producing a well-timed, natural, spontaneous, and symmetric smile (Figs. 4 and 5). [See **Video, Supplemental Digital Content 2**, which shows a preoperative video of the patient in Fig. 4, *above* (courtesy Facial Paralysis Institute), <http://links.lww.com/PRS/D439>. See **Video, Supplemental Digital Content 3**, which shows a postoperative



Video 2. Supplemental Digital Content 2 shows a preoperative video of the patient in Figure 4, *above* (courtesy Facial Paralysis Institute), <http://links.lww.com/PRS/D439>.



Video 3. Supplemental Digital Content 3 shows a postoperative video of the patient in Figure 4, *above*, 1 month after right modified selective neurectomy, platysma myotomy, revision bilateral rhytidectomy, autologous fat grafting, and botulinum toxin type A injection into the periorbital region and contralateral face performed by the senior author (B.A.) (courtesy Facial Paralysis Institute), <http://links.lww.com/PRS/D440>.



Fig. 4. Photographs obtained (*above, left*) preoperatively and (*above, right*) 9 months postoperatively after right modified selective neurectomy, platysma myotomy, revision bilateral rhytidectomy, and autologous fat grafting (see **Video, Supplemental Digital Content 2**, <http://links.lww.com/PRS/D439>; see **Video, Supplemental Digital Content 3**, <http://links.lww.com/PRS/D440>). Photographs obtained (*below, left*) preoperatively and (*below, right*) 10 months postoperatively after right modified selective neurectomy, platysma myotomy, bilateral rhytidectomy, upper blepharoplasty, autologous fat grafting, and fractionated carbon dioxide laser resurfacing. Both patients had not had any neuromodulators at the time the postoperative photographs were taken for at least 6 months. Note that the *blue dots* in *below, right*, are planned sites of botulinum toxin type A injections at the time of this postoperative visit. (Courtesy Facial Paralysis Institute.)

video of the patient in Fig. 4, *above*, 1 month after right modified selective neurectomy, platysma myotomy, revision bilateral rhytidectomy,

autologous fat grafting, and botulinum toxin type A injection into the periorbital region and contralateral face performed by the senior author (B.A.)



Fig. 5. (Left) Preoperative and (right) postoperative photographs 5 months after revision left modified selective neurectomy with improvement in left lower teeth show and left lower lip inferior excursion (see Video, Supplemental Digital Content 4, <http://links.lww.com/PRS/D441>; see Video, Supplemental Digital Content 5, <http://links.lww.com/PRS/D442>). The patient does not have significant alteration in pursing of the lips postoperatively. (Courtesy Facial Paralysis Institute.)

(courtesy Facial Paralysis Institute), <http://links.lww.com/PRS/D440>. See Video, Supplemental Digital Content 4, which shows a preoperative video of the patient in Fig. 5 (courtesy Facial Paralysis Institute), <http://links.lww.com/PRS/D441>. See Video, Supplemental Digital Content 5, which shows a postoperative video of the patient in Fig. 5 following revision modified selective neurectomy and platysmal myotomy procedure performed by the senior author (B.A.) (courtesy Facial Paralysis Institute), <http://links.lww.com/PRS/D442>.] Unlike most facial reanimation procedures, the entire neural pathway of the smile mechanism is maintained, as the native facial musculature is motorized by the facial nucleus by means of an uninterrupted facial nerve. Because most facial

palsy patients have post-facial paralysis synkinesis, modified selective neurectomy can potentially have a tremendous overall impact for practitioners in this field.

In addition, a subset of post-facial paralysis synkinesis patients with apparent marginal mandibular nerve dysfunction were also found to have improvement in depressor labii inferioris motion and lower teeth show (Fig. 5) (see Video, Supplemental Digital Content 4, <http://links.lww.com/PRS/D441>. See Video, Supplemental Digital Content 5, <http://links.lww.com/PRS/D442>). Marginal mandibular nerve rehabilitation has significant relevance because asymmetry of lower teeth show has been found to be one of the main factors influencing disfigurement after facial paralysis.³⁶



Video 4. Supplemental Digital Content 4 shows a preoperative video of the patient in Figure 5 (courtesy Facial Paralysis Institute), <http://links.lww.com/PRS/D441>.

To our knowledge, no previous studies have addressed this issue in patients with synkinesis without nerve grafting or muscle transfers.

The authors believe that the results of this procedure are long-lasting. Almost half the patients in the electronic clinician-graded facial function scale analysis had more than 1 year of follow-up and still showed persistent improvement in all synkinesis and smile categories.

Further advantages of modified selective neurectomy include quick patient recovery and the ability to perform the procedure on an outpatient basis, unlike most reanimation options. The study did not show a statistical difference in outcome when patients underwent concomitant cross-facial nerve grafting, direct muscle neurotization, or ipsilateral buccal-to-zygomatic end-to-side nerve coaptation. Nerve rerouting and cross-facial nerve graft procedures are likely unnecessary, and the senior author no longer performs these procedures on a routine basis. Despite the reduction of neural input into various muscles of facial expression, temporary worsening of oral competency, articulation, or lip pursing function was not common.

In this study, postsurgical global botulinum toxin type A doses to the entire face decreased significantly. Because modified selective neurectomy does not address periorbital synkinesis, botulinum toxin type A is still required to manage narrowing of the eyelid aperture. Botulinum toxin type A may also be required for the contralateral side of the face to further improve symmetry. Furthermore, patients may also have residual lower facial synkinesis because not all the peripheral facial nerve branches can be feasibly isolated or are not



Video 5. Supplemental Digital Content 5 shows a postoperative video of the patient in Figure 5 following revision modified selective neurectomy and platysmal myotomy procedure performed by the senior author (B.A.) (courtesy Facial Paralysis Institute), <http://links.lww.com/PRS/D442>.

ablated by choice to maintain adequate perioral neural input.

The senior author and his junior associates and physician extenders perform botulinum toxin type A injections at the Facial Paralysis Institute. This may add variability to patient outcome; however, the ancillary staff follow strict treatment protocols set by the senior author. In this study, only patients that were treated at the senior author's practice were included in the botulinum toxin type A analysis.

The use of neuromodulators in the postoperative period may potentially impact the findings of this study; however, the majority of the patients received botulinum toxin type A to only the periorbital region and contralateral side, which should not have an impact on the electronic clinician-graded facial function scale smile analysis. The authors also did not include any patients who had been treated with postoperative botulinum toxin type A in the prior 3 months. A prospective double-blinded study using a contralateral frontalis model by Nestor and Ablon showed that the median duration for "full efficacy" of botulinum toxin type A was only 87 days.³⁷ Using Jankovic scores for blepharospasm, Bihari showed that the duration of effect by botulinum toxin type A was only 62.2 days.³⁸ In addition, to reduce adverse events, the standard dosages used for synkinetic patients by the senior author are relatively low, which have shown to further reduce the duration of botulinum toxin type A effect.³⁹

Despite very promising results and limited risks, the outcomes for modified selective neurectomy are variable. The revision rate of 17 percent is an indicator of this unpredictability and the significant learning curve to this procedure. Anecdotally, we have noted very good results following revision surgery and a decreased revision rate as the senior author has performed more procedures (Fig. 5).

The study did not determine the contribution of the platysmal myotomy to the outcome of the modified selective neurectomy. Prior studies have shown only limited improvement of platysmal synkinesis without significant enhancement of smile mechanism.⁴⁰ The electronic clinician-graded facial function scale and House-Brackmann analysis was performed by an independent physician but not blinded to whether the patient photographs were preoperative or postoperative, which can potentially add to the bias of the data. The retrospective nature and lack of quality-of-life instruments in this study further limit its application. In addition, neuromuscular retraining was not standardized because most patients did not live in the local area. The authors plan on performing a long-term prospective study with blinded observers and quality-of-life assessments to definitively assess the permanence and significance of this surgical technique.

CONCLUSIONS

Modified selective neurectomy improves spontaneous smile mechanism and synkinesis in patients with post-facial paralysis synkinesis. This technique should be considered as an alternative to more invasive surgical options as a standalone treatment for patients who develop synkinesis with partial facial palsy.

Babak Azizzadeh, M.D.

The Center for Advanced Facial Plastic Surgery
9401 Wilshire Boulevard, Suite 650
Beverly Hills, Calif. 90212
drazizzadeh@gmail.com

PATIENT CONSENT

Patients provided written consent for the use of their images.

REFERENCES

1. Neely JG, Neufeld PS. Defining functional limitation, disability, and societal limitations in patients with facial paresis: Initial pilot questionnaire. *Am J Otol.* 1996;17:340–342.

2. Helwig NE, Sohre NE, Ruprecht MR, Guy SJ, Lyford-Pike S. Dynamic properties of successful smiles. *PLoS One* 2017;12:e0179708.
3. Takeda T, Takeda S, Okada T, Kakigi A, Yamasoba T. Experimental studies on the recovery processes from severe facial palsy and the development of its sequelae. *Otol Neurotol.* 2015;36:896–903.
4. Nash JJ, Friedland DR, Boorsma KJ, Rhee JS. Management and outcomes of facial paralysis from intratemporal blunt trauma: A systematic review. *Laryngoscope* 2010;120:1397–1404.
5. Beurskens CH, Oosterhof J, Nijhuis-van der Sanden MW. Frequency and location of synkineses in patients with peripheral facial nerve paresis. *Otol Neurotol.* 2010;31:671–675.
6. Yamamoto E, Nishimura H, Hirono Y. Occurrence of sequelae in Bell's palsy. *Acta Otolaryngol Suppl.* 1988;446:93–96.
7. Celik M, Forta H, Vural C. The development of synkinesis after facial nerve paralysis. *Eur Neurol.* 2000;43:147–151.
8. Moran CJ, Neely JG. Patterns of facial nerve synkinesis. *Laryngoscope* 1996;106:1491–1496.
9. Guntinas-Lichius O, Irintchev A, Streppel M, et al. Factors limiting motor recovery after facial nerve transection in the rat: Combined structural and functional analyses. *Eur J Neurosci.* 2005;21:391–402.
10. Eekhof JL, Aramideh M, Speelman JD, Devriese PP, Ongerboer De Visser BW. Blink reflexes and lateral spreading in patients with synkinesia after Bell's palsy and in hemifacial spasm. *Eur Neurol.* 2000;43:141–146.
11. Bratzlavsky M, vander Eecken H. Altered synaptic organization in facial nucleus following facial nerve regeneration: An electrophysiological study in man. *Ann Neurol.* 1977;2:71–73.
12. Brach JS, VanSwearingen J, Delitto A, Johnson PC. Impairment and disability in patients with facial neuromuscular dysfunction. *Otolaryngol Head Neck Surg.* 1997;117:315–321.
13. Dall'Angelo A, Mandrini S, Sala V, et al. Platysma synkinesis in facial palsy and botulinum toxin type A. *Laryngoscope* 2014;124:2513–2517.
14. Diels HJ, Beurskens CHG. Neuromuscular retraining: Non-surgical therapy for facial palsy. In: *The Facial Nerve*. Slattery WH III, Azizzadeh B, eds. New York: Thieme; 2014:205–213.
15. Terzis JK, Karypidis D. Therapeutic strategies in post-facial paralysis synkinesis in adult patients. *Plast Reconstr Surg.* 2012;129:925e–939e.
16. Azizzadeh B, Pettijohn KJ. The gracilis free flap. *Facial Plast Surg Clin North Am.* 2016;24:47–60.
17. Moubayed SP, Labbé D, Rahal A. Lengthening temporalis myoplasty for facial paralysis reanimation: An objective analysis of each surgical step. *JAMA Facial Plast Surg.* 2015;17:179–182.
18. Cabin JA, Massry GG, Azizzadeh B. Botulinum toxin in the management of facial paralysis. *Curr Opin Otolaryngol Head Neck Surg.* 2015;23:272–280.
19. Tzafetta K, Terzis JK. Essays on the facial nerve: Part I. Microanatomy. *Plast Reconstr Surg.* 2010;125:879–889.
20. Terzis JK, Karypidis D. Outcomes of direct muscle neurotisation in adult facial paralysis. *J Plast Reconstr Aesthet Surg.* 2011;64:174–184.
21. Diels HJ. Facial paralysis: Is there a role for a therapist? *Facial Plast Surg.* 2000;16:361–364.
22. Viterbo F, Trindade JC, Hoshino K, Mazzoni Neto A. End-to-side neurotomy with removal of the epineurial sheath: An experimental study in rats. *Plast Reconstr Surg.* 1994;94:1038–1047.
23. Toffola ED, Furini F, Redaelli C, Prestifilippo E, Bejor M. Evaluation and treatment of synkinesis with botulinum toxin following facial nerve palsy. *Disabil Rehabil.* 2010;32:1414–1418.

24. Banks CA, Bhamra PK, Park J, Hadlock CR, Hadlock TA. Clinician-graded electronic facial paralysis assessment: The eFACE. *Plast Reconstr Surg*. 2015;136:223e–230e.
25. Banks CA, Jowett N, Hadlock TA. Test-retest reliability and agreement between in-person and video assessment of facial mimetic function using the eFACE facial grading system. *JAMA Facial Plast Surg*. 2017;19:206–211.
26. Coleman CC. Surgical treatment of facial spasm. *Ann Surg*. 1937;105:647–657.
27. Greenwood J Jr. The surgical treatment of hemifacial spasm. *J Neurosurg*. 1946;3:506–510.
28. Marino H, Alurralde A. Spastic facial palsy; peripheral selective neurotomy. *Br J Plast Surg*. 1950;3:56–59.
29. Hohman MH, Lee LN, Hadlock TA. Two-step highly selective neurectomy for refractory periocular synkinesis. *Laryngoscope* 2013;123:1385–1388.
30. Fisch U, Esslen E. The surgical treatment of facial hyperkinesia. *Arch Otolaryngol*. 1972;95:400–405.
31. Guerrissi JO. Selective myectomy for postparetic facial synkinesis. *Plast Reconstr Surg*. 1991;87:459–466.
32. Laskawi R, Rohrbach S, Rödel R. Surgical and nonsurgical treatment options in patients with movement disorders of the platysma. *J Oral Maxillofac Surg*. 2002;60:157–162.
33. Nakamura K, Murakami S, Kozawa T, Yanagihara N. Surgical treatment of synkinesis. *Eur Arch Otorhinolaryngol*. 1994;Dec: S380–S382.
34. Henstrom DK, Malo JS, Cheney ML, Hadlock TA. Platysmectomy: An effective intervention for facial synkinesis and hypertonicity. *Arch Facial Plast Surg*. 2011;13: 239–243.
35. Chuang DC, Chang TN, Lu JC. Postparalysis facial synkinesis: Clinical classification and surgical strategies. *Plast Reconstr Surg Glob Open* 2015;3:e320.
36. Banks CA, Jowett N, Hadlock CR, Hadlock TA. Weighting of facial grading variables to disfigurement in facial palsy. *JAMA Facial Plast Surg*. 2016;18:292–298.
37. Nestor MS, Ablon GR. Duration of action of abobotulinumtoxin and onabotulinumtoxin: A randomized, double-blind study using a contralateral frontalis model. *J Clin Aesthet Dermatol*. 2011;4:43–49.
38. Bihari K. Safety, effectiveness, and duration of effect of BOTOX after switching from Dysport for blepharospasm, cervical dystonia, and hemifacial spasm dystonia, and hemifacial spasm. *Curr Med Res Opin*. 2005;21:433–438.
39. Poewe W, Deuschl G, Nebe A, et al. What is the optimal dose of botulinum toxin A in the treatment of cervical dystonia? Results of a double blind, placebo controlled, dose ranging study using Dysport. German Dystonia Study Group. *J Neurol Neurosurg Psychiatry* 1998;64:13–17.
40. Hadlock TA, Greenfield LJ, Wernick-Robinson M, Cheney ML. Multimodality approach to management of the paralyzed face. *Laryngoscope* 2006;116:1385–1389.