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Facial Reanimation With Jump Interpositional Graft Hypoglossal Facial Anastomosis and Hypoglossal Facial Anastomosis: Evolution in Management of Facial Paralysis

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When viable proximal facial nerve is inacessible, facial nerve paralysis has been classically managed with the hypoglossal facial anastomosis (HFA) for at least the past 70 years. While this procedure has proven its reliability, its problems with hemilingual atrophy (speech deglutition, drooling, mastication), hypertonia, synkinesis, and mimetic deficits indicate the need for a more perfect solution for facial paralysis. The jump interpositional graft hypoglossal facial anastomosis (JIGHFA) along with gold weight lid implantation and electromyographic (EMG) rehabilitation achieves substantial facial reanimation without hemilingual deficits. We present our results in 18 patients who underwent JIGHFA along with gold weight lid implantation and EMG rehabilitation for facial paralysis. These results were compared with those from published series of 30 patients treated with HFA with EMG rehabilitation evaluated with objective (House-Brackmann) criteria. Anonymous retrospective information from questionnaires from 22 of 48 patients who were treated with the classic HFA was also presented. In properly selected patients, the JIGHFA technique is capable of achieving substantial facial reinnervation (House-Brackmann grade III or better) in 83.3% of the patients without hemilingual sequelae which was seen in 45% of the HFA patients. In contrast to the HFA, this procedure can be used by patients with concomitant lower cranial nerve paralysis (except hypoglossal), and bilateral facial paralysis. Hypertonia, synkinesis, and lagophthalmus were less symptomatic in the JIGHFA patients. Mimetic expression was not improved in the JIGHFA population compared with the HFA group.
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INTRODUCTION

Paralysis of the facial nerve has protean consequences due to multiple, almost infinite, functions under its domain. The physiologic divisions of this neuromotor system are well known: motor efferent (special visceral efferent fibers) to the striated musculature of the face and neck including the stylohyoid, the stapedius, and the posterior belly of the digastric muscles; preganglionic parasympathetic (general visceral efferent fibers) to the lacrimal, submaxillary, sublingual, and nasal seromucinous glands; afferent taste (special sensory fibers) from the lateral anterior two thirds of the tongue, palate, and tonsillar fossa; afferent proprioception (visceral afferent fibers) from facial muscles; and afferent cutaneous sensation (somatic sensory fibers) from the external auditory external canal and the concha.[1]

While facial reanimation surgery is designed to restore facial movement, reconstruction of appropriate emotional facial expression is particularly elusive. Facial expression is a vital form of nonverbal communication whose absence accentuates feelings of isolation in addition to the self-perception of "grotesque." This interpersonal language based on a flow of interactive social signals may be overlooked by objective facial paralysis grading scales and those performing facial reanimation surgery.

Defects in the complex, incompletely understood interactions between facial nerve activity and other paralyzed cranial nerves may impair successful restoration of facial and oral function. Requirements for sucking, speech, chewing, and swallowing involve synergistic activity of the facial musculature (buccinator and orbicularis oris) and muscles innervated by the lower cranial nerves (IX, X, and XII) along with afferent and efferent trigemi-

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nal function.[2] Optimal facial rehabilitation also entails effective afferent trigeminal proprioceptive feedback.[3]

The diversity and complexity of facial function over the past century have spawned multiple ways to manage facial paralysis. Contemporary dynamic facial reanimation is determined by the presence and accessibility of viable structures along the facial neuromotor pathway. Alternative substitution tissue replaces and repairs deficits in the facial neuromotor pathway. For example, if the proximal intracranial facial nerve and distal nerve trunk are accessible, repair can be achieved with primary direct anastomosis or with insertion of an interpositional nerve graft, which can extend distally to within the temporal bone or extratemporally to extrastylomastoid branches.[4–7] If the proximal nerve is not accessible, then ipsilateral crossover cranial nerve (e.g., hypoglossal) and/or an interposition nerve graft from contralateral facial nerve branches can provide proximal nerve substitution.[8–10] Deficient distal facial musculature can be replaced with regional muscle transposition (myoneurotization), free muscle grafts innervated by either proximal facial nerve, or a variety of crossover nerve or facial nerve substitutions.[11–13]

Static procedures can include rhytidectomy and facial regional suspension with autogenous (temporalis muscle and/or fascia) or alloplastic materials (e.g., Gore-Tex).[14]

The purpose of listing methods of facial reanimation is not to be comprehensive but to indicate which positions hypoglossal facial anastomosis (HFA) and jump interpositional graft hypoglossal facial anastomosis (JIGHFA) occupy in the panoply of facial reinnervation procedures.[10,15] As discussed in greater detail below, HFA and JIGHFA are two methods of facial reanimation that can be used when proximal facial nerve is not accessible in the presence of viable distal facial nerve and musculature.

The challenges to complete normal facial function following facial reanimation are, indeed, formidable: ". . . in no case can the final cosmetic result during all activities of the face be expected to equal that found in the presence of a normally functioning facial nerve."[16] Problematic areas of facial reanimation are not limited to the following: extent of muscle tone restoration; degree of voluntary muscle movement; amount of mimetic facial movement; synkinesis; eye protection; speech facility; intraoral management of foods/fluids; and mastication and swallowing. Ideal facial functional rehabilitation should achieve 1) symmetry at rest; 2) symmetry with voluntary motion; 3) symmetry with involuntary expression of emotion; 4) restoration of oral, nasal, and ocular sphincter control; and 5) the above without loss of other function.

When direct facial nerve suture or grafting is not possible in the presence of functioning distal facial nerve and viable musculature, the HFA and its derivative, the JIGHFA, have been used to manage facial paralysis.[10,15] The purpose of this report is to examine the efficacy of JIGHFA, particularly in comparison to HFA, in a retrospective view of patients to ascertain the advantages and the limitations of this method of facial reanimation.

HISTORY

The earliest report of surgical restoration of facial function with alternative crossover cranial nerve motor reinnervation of the facial musculature is attributed to Drobnik in 1879. An anastomosis between the proximal spinal accessory and the distal facial nerve trunk was used to treat his patient's facial paralysis from temporal bone suppuration. According to Sawicki, "it was ascertained that the features had become more symmetrical."[9] Ballance and Duel[17] were not able to find the original account of this case. In 1898, Faure[18] of the Hospital Laënnec reported an attempt to reinnervate a paralyzed facial nerve in a human that did not succeed. This failure was thought to be due to the long period of facial paralysis—18 months.

The first known successful nerve crossover innervation of the facial nerve in humans was reported by Robert Kennedy[19] of Glasgow, to the Royal Society in 1900, as cited by Cushing during his discussion of reanimation with the spinal accessory nerve. Kennedy used this technique to treat facial spasms, which were controlled with this method.

In 1920, Ballance[20] commented on his experience with anastomosing the facial nerve end to side to the spinal accessory for otogenic facial paralysis, which resulted in response to faradic stimulation. The patient never regained movement dissociated from shoulder movement. Ballance also cited the reports of others including Faure in 1898, Cushing (1903), and Kummer (1920), who used the spinal accessory nerve to reinnervate the distal facial nerve. The imperfect results of the spinal accessory—facial anastomosis were noted by Ballance:

> After spinal accessory facial anastomosis, associated movements of the face and shoulder are always the first, and sometimes the permanent, result of anastomosis. Dissociated movements of the face and shoulder are rare, requiring a long patient period of reeducation and may never be regained even after years of effort . . . Again symmetrical and subconscious emotional movements of both sides of the face—in which the cortical centers of the opposite sides of the brain must cooperate in unison—the movement that is most desired, is the one that frequently eludes recovery.[17]

Although Furet suggested to Faure in 1901 that facial paralysis might be corrected with a hypoglossal-facial anastomosis, Korte was the first to describe performing this procedure, which occurred December 20, 1901, for a facial paralysis due to temporal bone resection following infection.[10,21] Professor Bernhardt, a neurologist at the University of Berlin, in a letter to Ballance, reported his observations of Korte's patient:

> The patient in laughing moves the mouth and movements of expression can also be carried out through the facial. I believe, however, there are associated movements of the tongue which escape observation as the tongue is confined within the mouth, but they in no way trouble the patient, nor are they noted by those around her. From all that I have observed, I entirely agree with you that in grafting the facial for relief of facial palsy, it is better, not withstanding the opinion of Korte to chose the hypoglossal rather than the spinal accessory.[17]

At this time Korte also contemplated the possibility of central nervous system plasticity:

> It is a most remarkable physiologic fact that ganglion cells of the hypoglossal or the accessory nerve-nucleus can acquire under voluntary influence the ability to induce contractions of muscles originally innervated from other nerve centers. We cannot yet decide whether there are connections between the two areas of nerve ganglia or whether by will-power the nerve center can adopt or become accustomed to stimulating a newly acquired muscle system [translated by E. Stennert[2]].

After citing their own and the experiences of colleagues with end-to-side hypoglossal facial anastomosis, Ballance declared:

> . . . we have performed end to side facial anastomoses, and the experience has persuaded us that it is a mistake to attempt the great problem of curing facial palsy and the same time to endeavor to preserve the function of the tongue unimpaired. We think, too, objectives in surgery, as in war, mean, as a rule, partial success or failure. The curing of facial palsy justifies the sacrifice of the movements of half the tongue. The end to side anastomosis may militate against a more perfect restoration of the facial movement. The only operation therefore that should be carried out is end to end anastomosis. On this earth ours what great good has ever been or can be gained except by sacrifice? It is, in every era, and in every zone, the law of life.[17]

Over the following several decades, many authors observed that HFA proved to be a reliable method to achieve strong reinnervation of facial musculature. In 1940, Coleman,[22] while extolling the vigorous reinnervated muscle activity associated with the HFA, commented on its lack of regionalization (failure to achieve discrete regional movement independent of other areas in the face), and absent emotional expression in the paralyzed side. He thought that his most successful patients were those who attained minimal asymmetric facial movement essentially through maximal suppression of lingual activity. Coleman declared that the success of anastomosis operations depends on the patient himself or herself and ". . . the loquacious patient is the most unfavorable type for facio-hypoglossal anastomosis." With the misdirection of regenerated nerve fibers throughout all ipsilateral muscles making the entire facial musculature on the affected side a single functional unit resulting in facial movement en masse, Coleman believed that the patients with HFA would be unable to achieve a complete dissociation of action of various muscle groups. Therefore, he advocated that "suppression of facial movements should become a fixed habit." It is certainly odd to recommend suppression of movement to achieve symmetry which seemingly works against the ultimate goal of facial reinnervation: facial movement. Throughout the literature on HFA we frequently see this contradictory theme of achieving symmetry at the cost of movement.

At the same meeting in which Coleman spoke, Lahey expoused his clinic's preference for the spinal accessory nerve for facial reinnervation based on its experience with 19 patients resulting in fewer speech and swallowing difficulties. Coleman responded that hypoglossal function has a closer functional similarity to the facial nerve, therefore implying that it is capable of producing more appropriate facial movement. He also noted that his patients did not suffer speech defects based on monitored preoperative and postoperative voice recordings. Hemilingual atrophy was present in all cases, and distal hypoglossal reinnervation with a crossover from the hypoglossal descendens branch did not prevent this atrophy. Coleman concluded that HFA provided facial symmetry in repose, restored voluntary movement of paralyzed muscles, and prevented further disfigurement from atrophy of facial musculature.

Later in 1954, Alexander[16] reported results as "excellent" in 12 patients, "good" in 14, and "fair" in 7 from a total of 33 of 42 patients who were treated with HFA. Criteria for these ratings were not given. In all cases there was some form of facial movement. Hypertonia was observed in a few but an unspecified number of cases. Forehead movement was usually absent and markedly impaired when present in a few cases. Alexander[16] also believed that individuals who rarely smiled and who displayed little emotional expression were likely to show the best results after HFA. He wrote that no function appeared on the side of the anastomosis with the outburst of laughter which pulled the face to the normal side. He thought that hemilingual atrophy did not interfere with talking or eating. The author verified that there was good or excellent reinnervation in at least 75% of the patients resulting in facial symmetry in repose and some animation during conversation. Alexander found that comparable results were obtained if HFA was performed anytime within 2½ years after loss of facial function—even though he recommended that HFA be performed as early as possible for the best results. He did not delineate an absolute time beyond which HFA should not be performed.

Falbe-Hansen[23] observed that all of the 23 followed patients of the 25 who underwent HFA in his study had signs of reinnervation. Their results were classified as "good" for 12; "between good and poor," 8; and "poor," 3. The good group achieved facial symmetry at rest, voluntary eye closure, voluntary corner of the mouth movements, mimetic-like synkinesis during speech, and no sequela of ipsilateral hypoglossal dysfunction. The poor group had one or more of the following: severe asymmetry at rest, orbicularis oculi closure defects, pronounced hypertonic (grimacing) facial movement, and discomfort from the hypoglossal deficit. The intermediate group did not satisfy all the criteria for the good group but did not qualify for the poor group criteria. In two subjects in the poor group, the anastomosis occurred 6 years after loss of facial function, and in one patient the anastomosis was deemed technically insufficient due to tension at the anastomotic site. In contrast to Coleman's report,[22] there were many examples of discrete regional movements when submaximal lingual force was elicited. Mass movements (synkinesis), however, always occurred with maximal force. Fifteen patients had observable movement concurrently with speech, which gave the face a dynamic expression. Vigorous emotional facial movements had no corresponding movement on the reanimated side, which produced severe asymmetry.

There were 10 patients with slight difficulty in eating or blurring of consonants. Falbe-Hansen[23] thought that this was not remarkable because half of these patients had reduced or abolished trigeminal sensory function, which can contribute to oral dysfunction. Frontalis movement was obtained with voluntary hypoglossal activity in 4 out of 23 patients following HFA.

In 1974 Evans[24] reported the outcomes in 13 patients with HFA with follow-up of at least 2 years. Their etiologies for facial paralysis were acoustic neuroma in 12 and herpes zoster oticus in 1. After outlining specific criteria, HFA was noted to be effective in producing near complete facial symmetry at rest in nearly all cases and limited purposeful movement in a few. There were four patients who were enthusiastic, eight were pleased with slight reservations, and one said that there was no improvement in facial appearance. Intraoral sticking of food in the vestibule bothered five patients. Dysarthria was present in two patients and barely noticeable in five. Lingual hemiatrophy was visible in most cases but not particularly symptomatic.

All but one patient had partial tarsorrhaphies as part of the initial management of facial paralysis. No mention was made of subsequent tarsorrhaphy release. Four patients were able to close their affected eye completely, with the remaining nine achieving partial voluntary closure without keratitis. Two patients were unable to close the affected eye except with lingual protrusion—producing a massive facial grimace. Evans concluded that eye closure was more likely to occur when the HFA was performed within 6 months of the onset of facial paralysis.

Evans observed complete facial symmetry at rest in six patients, slight asymmetry in six, and severe unaltered paralysis in one. While facial movement was detectable in four patients, it was inadequate to achieve facial symmetry with expression—fine movements with speech nevertheless implied some facial animation to the observer. In this series, Evans recalled that one of his best results occurred in a "voluble talker," suggesting that such lingual activity can contribute to excellent return of facial tone. This observation should be contrasted to Coleman's reservations about "loquacious" patients. Evans thought pre-existing difficulty with manipulating food was the primary contraindication to HFA and advocated an alternative cranial nerve motor source, that is, spinal accessory, with sparing the trapezius muscle. He quoted Stookey's conclusions[25] about the differing suitability of these two reinnervating crossover nerves: "In a singer the spinal accessory should be used for crossing. A hod carrier could better sacrifice his hypoglossal."

In 1959 Kessler et al.[26] reported successful (normal facial tone in repose, eye closure, movement, corner of mouth movement, and no gross speech impairment) in 14 of 17 patients. The reasons for the three poor results were not made available.

Hemiatrophy of tongue was observed without gross dysfunction. Electromyographic recordings from the tongue (and the face) revealed motor unit discharges with volition in three patients without iatrogenic lingual reinnervation (with the hypoglossal descendens branch) suggestive of cross-lingual neurotization from the contralateral hypoglossal nerve. In five patients the hypoglossal descendens branch was anastomosed to the distal hypoglossal nerve with documented lingual reinnervation in only two patients. No functional differences were noted between those with and without lingual atrophy.

In a series of 30 of 40 patients treated with HFA over a 20-year period, Gavron and Clemis[27,28] reported excellent results in 12 patients; "good" results in 10,"fair" in 6, and "no improvement" in 2 after HFA. Their criteria were similar to earlier reports with the excellent category achieving facial symmetry in repose, strong voluntary motion in midface, minimal synkinesis, inappropriate and dissociated movements with lingual activity. The "fair" patients had poor to good facial tone and symmetry at rest but limited facial improvement or good facial motion with significant synkinesis and or marked dissociated facial movements. Those rated "good" were between excellent and fair, primarily with deficits involving dissociated facial movements or synkinesis, despite good symmetry at rest and strong voluntary facial movement.

All but one facial paralysis was due to cerebellopontine angle tumor. No relation was found between tumor size, surgical approach for tumor removal, sex, or variation in anastomotic technique and ultimate degree of facial reinnervation. There was a trend suggesting poorer results associated with increasing age, although there were patients of all ages in each of the categorized groups. Intervals greater than 1 year between the onset of facial paralysis and anastomosis adversely affected the prognosis, although the authors could not establish an absolute interval beyond which HFA was definitely contraindicated. Most of the patients required supplementary procedures for physiologic protection of the eye, and some desired corrective cosmetic surgery.

In their experience, Clemis and Gavron viewed hemiatrophy of the tongue as an enhancement of lingual function by reducing the bulk of the abnormal side and "thereby the work load of the normal side." While speech and swallowing were affected in the early postoperative course, these problems diminished with reinnervation of the midface from improved buccinator function and pursing action of the mouth.

Clemis and Gavron stressed accurate preoperative assessment of lower cranial nerves to avoid potential exacerbation of dysphagia and aspiration from the additional loss of ipsilateral hypoglossal innervation to the tongue. While one of their patients had ipsilateral loss of cranial nerves IX and X, they did not report postoperative dysphagia or masticatory difficulties in this patient.

Reference to the incidence of facial paralysis secondary to intracranial posterior fossa surgery was made by both Coleman and Clemis, whose juxtaposed comments place this issue in unique historical perspective. In 1940, Coleman[22] observed that intracranial injuries of the facial nerve formerly were rare but were going to become more frequent due to the increasing number of operations for cerebellopontine angle tumor, producing destruction of the facial nerve associated with complete removal of the tumor and its capsule. Forty-four years later, Clemis and Gavron[28] observed that with improved diagnostic (magnetic resonance imaging [MRI] to identify smaller tu-

mors) and surgical techniques (operating microscope, better understanding of surgical anatomy and intraoperative facial nerve monitoring), the incidence of facial nerve paralysis would decrease and the need for HFA would decline until it would be a "rare operation."

In a retrospective review of 61 cases of HFA performed within 5 to 7 days of facial paralysis associated with cerebellopontine angle tumor removal, Pensak et al.[29] reported that 90% of the patients gained some facial movement. The onset of facial reanimation occurred within 9 months in 61%. The most frequent region of initial reanimation was in the mouth, which occurred in 58%, followed by primary movement in ocular sphincter (18%) and the nasal fold (15%). Forehead activity was noted by 9% of the respondents.

Measurement of facial reanimation in the series of Pensak et al. used criteria similar to earlier reports.[27,28] Three percent of the patients were rated excellent, 39% were good, 49% were fair, and 10% were poor. Synkinetic activity was noted in 67% of corresponding patients. Complete volitional control of mimetic activity was achieved by 27%, in contrast to 73% with incomplete voluntary mimetic emotional response. This implies that involuntary mimetic movements were not present.

In Pensak's survey,[29] two specific problem areas were noted: ophthalmologic disability and difficulty with intraoral management of food. While 75% of the patients had eye closure, 21 encountered difficulty with eye dryness (29%), visual problems (11%), eye irritation (17%), ocular pain (9%), and excessive tearing (4%). Corneal abrasions, neutrophic keratitis, and discomfort with persisting lateral tarsorrhaphy were also identified. Moderate tongue atrophy was reported in 53% of the patients with only 21% debilitated by hemilingual atrophy. Dysphagia was noted by "few patients."

In this series of patients, 23% of the respondents believed that the paralysis affected their social lives and 21% observed an adverse effect on their livelihood. Those with strong emotional support systems from family and co-workers, close physician-patient relationships, and realistic expectations tended to adapt better compared with those with anger, depression, prolonged grieving, and despair. Nevertheless, 74% of the respondents believed that HFA was very helpful.

Because the majority of those reinnervated with HFA have no forehead motion, Brudny modified the House-Brackmann facial grading system, which was originally adapted by the Facial Nerve Disorder Committee of the American Academy of Otolaryngology Head and Neck Surgery.[30,31] With this six-point grading scale of facial function, (grade I being normal and VI representing total paralysis), those without forehead function would automatically be placed into grade IV or worse, regardless of how successful was the remaining facial function. This modified system also graded for dissociated facial movements (grimacing) from tongue activity (Table I). In 30 patients, who had EMG rehabilitation following HFA, Brudny noted that 10 patients achieved grade II/VI; 17 patients had grade III/VI, and 3 had grade IV/VI facial function (Fig. 1).

In contrast to the previously cited reports of HFA for facial paralysis secondary to intracranial (usually acoustic neuromas)/temporal bone lesions, Conley's experience with 137 cases were mostly from patients with extracranial lesions (cancer or infection): 52 temporal bone resections, 18 radical parotidectomies, and 10 ear resections.[32,33] It is possible that there were fewer cases with impaired trigeminal function in this series of extracranial facial paralysis compared with the other cited series of intracranial facial paralysis managed with HFA. There were, however, in this series 16 cases of facial paralysis from acoustic neuroma surgery. HFA was performed in 94 patients within 2 years after the onset of facial paralysis. In 43 patients, the HFA was delayed beyond 2 years.

Conley[32] observed that 95% of the patients attained good muscle tone in repose, along with some mass movement. He found, however, that 98% of the patients who underwent "immediate" HFA (within 2 years) for facial paralysis attained movement in contrast to the 90% achieving the same following delayed HFA (beyond 2 years). In the immediate surgical group, good or better movement was attained in 77%, whereas in the delayed group similar results were obtained in 41%. Extreme reinnervation activity (hypertonia) was noted in some of the better-performing groups, but the number of patients with this difficulty was not specified. Although severe lingual hemiatrophy was documented in 25% of the patients, functional problems were not observed, especially in the patients undergoing immediate reanimation surgery. Conley commented on the variability of hemilingual atrophy and averred that "cross glossal axonal" growth from the normal side of the tongue might neurotize the surgically

Fig. 1. Progressive facial electromyographic (EMG) feedback rehabilitation. **A** and **B.** Asymmetric movement after hypoglossal facial anastomosis (HFA) but before EMG feedback rehabilitation. **C.** Surface electrodes in place for audible and visual EMG waveform feedback comparison between normal and reinnervated sides. **D.** Eye closure with lower face symmetry and "relaxation" compared with **B. E.** Balanced smile with more symmetry compared with **A.** (Reprinted with permission from Brudny, et al.[30])

TABLE I.
Brudny's Modification of House-Brackmann Scale for Grading Facial Paralysis for Reanimation With Hypoglossal Facial Anastomosis.

Grade	Normal	II Mild Dysfunction	III Moderate Dysfunction	IV Moderately Severe Dysfunction	V Severe Dysfunction		VI Total Paralysis
					Short S/P	Long S/P	
A. At repose							
1. Appearance	N	N	Mild asymmetry	Marked asymmetry	Very marked asymmetry		Disfiguring asymmetry
2. Tone	N	N	Good	Decreased	Decreased	Increased	Absent
B. Selective volitional motion of							
1. Forehead	N	Some	Absent	Absent	Absent	Absent	Absent
2. Eye closure	N	Full effortless	Full, with effort, or tongue motion	Incomplete, upon tongue motion only	Absent	Absent	Absent
3. Blinking	Spontaneous	Volitional	Absent	Absent	Absent	Absent	Absent
4. Upper lip elevation	N	Modest	Modest	Absent	Absent	Absent	Absent
5. Lower lip depression	N	Modest	Minimal	Absent	Absent	Absent	Absent
6. Smile	Spontaneous, symmetrical, synchronous	Spontaneous, symmetrical, synchronous	Spontaneous (modest), asymmetrical, synchronous	Upon tongue motion, asymmetrical asynchronous	Absent	Absent	Absent
7. Lips pursing	N	N	Full, with sealing	Incomplete, no sealing	Absent	Absent	Absent
C. Synkinesis due to							
1. Tongue movement	No abnormal facial movement	Minimal lower lid and mouth	Moderate lower lid and mouth	Slight lower lid and mouth	Slight mouth	Severe around eye and mouth	No facial movement
2. Swallowing	No abnormal facial movement	Minimal lower lid	Moderate lid and mouth	Slight lower lid and mouth	No facial movement	Severe around eye and mouth	No facial movement

Note that absent forehead movement still permits attainment of grade III in contrast to original House-Brackmann scale, which grades these as class IV.[30]
S/P = status post anastomosis (short ≤ 1 y, long > 1 y); N = normal.

denervated hemitongue. He believed that those with the least lingual atrophy were thought to have the best facial movement and optimal intraoral competency.

Those with delayed anastomosis complained of difficulty with mastication (16%), swallowing (12%), and speech (10%). All patients in this delayed group who had preoperative problems with mastication and speech noted improvement after HFA, while deficiencies with swallowing increased.

Along with previous authors, Conley strongly advocated his preference for the hypoglossal nerve over the spinal accessory, phrenic, and cross facial-facial nerve grafts.

In a retrospective review of 66 cases of HFA, Luxford and Brackmann[34] were able to obtain information for 48 patients. Excellent results were obtained by 12 (25%), good results by 17 (35.4%), fair results by 15 (31.2%), and poor results by 4 (8.3%). They also found that 89% of the patients achieved good to excellent facial reanimation if their HFA surgery occurred within 24 months of facial paralysis. When the HFA surgery occurred after 36 months of paralysis, 29% attained good to excellent results. Criteria for classification were similar to those used by Gavron and Clemis.[27]

Kunihiro et al.[35] reported HFA postoperative results in 28 of 34 patients in whom he could follow up. Using the House-Brackmann scale, their facial function was classified as follows: grade III for 10 patients; grade IV in 14; and for the remaining 4, grade V. There was a positive correlation between the patients' subjective evaluations and their overall satisfaction. In contrast, there was no significant correlation between objective and subjective evaluations or overall satisfaction. Kunihira suggested that normal function is the standard by which the patients evaluate themselves and measure their satisfaction. Since the HFA cannot achieve normalcy due to persistent sequelae of various degrees (i.e., synkinesis, hypertonia, lagophthalmos, and distal hypoglossal nerve deficits), these patients will always have some degree of dissatisfaction. Evaluation by objective criteria (House-Brackmann grading system) was performed by people other than the patients.

The cumulative results of the above-cited HFA reports (Table II) totaling 418 subjects from multiple surgeons illustrate a summary trend with the following classifications: good to excellent in 64.6%, fair in 24.2%, and poor in 11%. The results displayed in Table II are extrapolated based on similar criteria. Good to excellent results demonstrate facial symmetry at rest with a good voluntary motion of eye and mouth, along with absence of severe dissociated facial movements (grimacing) and minimal synkinesis. Poor results were defined by severe facial asymmetry at rest, minimal voluntary motion of face including eye and mouth, severe synkinesis, and severe grimacing with lingual activity. Fair results encompassed those who did not completely satisfy the requirements of either the good to excellent or poor group. There was a summary incidence of 23.5% for problems associated with hemilingual atrophy, of 76% for synkinesis, and of 29% for ophthalmic problems (Table III.)

The extrapolated results listed in Table II have limited precision, which is intrinsic to the multiple, sometimes conflicting variables assessed. For example, Pensak's data were based on subjective responses by the patients. In contrast, Brudny's series used specific criteria based on the House-Brackmann scale. Other shortcomings of this summary pertain to inability to separate the different surgical techniques and modifications of the HFA. For example, Conley's series included 12 cases with longitudinally split hypoglossal nerves, 3 cases that used the hypoglossal descendens branch, and 19 cases with anastomosis to facial nerve branches distal to the main trunk. Many reports were limited by the number of patients available for follow-up. It is conceivable that those who failed to participate in follow-up clinical examinations or response to questionnaires were those discouraged or angry with poor results. On the other hand, Brudny's series self-selected out the successful cases: only those with evidence of reinnervation were referred for EMG rehabilitation. Nevertheless, the

TABLE II.
Cumulative Facial Reanimation Results From 10 Published Series Following Hypoglossal Facial Anastomosis.

			Results		
Author	No. Reported	Total	Excellent/Good	Fair	Poor
Alexander and Davis[16]	33	42	26	7	0
Falbe-Hansen[23]	23	25	18	2	3
Evans[24]	13	20	12	0	1
Kessler and Pool[26]	14	14	11	0	3
Conley and Baker[32,33]	137	137	89	25	23
Gavron and Clemis[27,28]	30	40	22	6	2
Pensak and Glasscock[29]	61	61	26	29	6
Brundy[30]	30	30	27	3	0
Kunihiro and Matsunga[35]	28	34	10	14	4
Luxford and Brackmann[34]	48	66	29	15	4
Total	418	469	270	101	46
%	100	112.2	64.6	24.2	11.0

TABLE III.
Summary Incidence of Complications (%) Associated With Hypoglossal Facial Anastomosis.

Study (N)	Speech	Hemilingual Atrophy		Synkinesis	Eye
		Swallowing	Chewing		
Falbe-Hansen (23)	43.4	—	—	—	—
Evans (13)	38.5	—	—	—	—
Pensak (61)	21.0	—	—	67	29
Conley (137)	10.0	12	16	—	—
Stennert (11)	—	—	—	85	—
Average incidence	—	23.5		76	29

Data are in percentages from individual study cited unless indicated as "average."[2,23,24,29,33]

general trend of this series is one of substantial "dynamic" facial reanimation in 88.8% (64.6 24.2) of patients within the above-cited limited parameters.

HYPOGLOSSAL FACIAL ANASTOMOSIS

Timing of Repair: Basic Science and Clinical Experience

While optimal timing for the successful facial reanimation with HFA has not been established, general guidelines have developed based on clinical experience. These reports primarily delineate a time beyond which attempts at facial reinnervation with HFA are unlikely to attain a reliable, satisfactory result. Ultimately, timing of the HFA must occur before loss of viable recipient facial musculature and fibrosis of the distal facial nerve impede the axonal ingrowth from the proximal hypoglossal nerve.

Classic neuropathology has established that distal to the sites of injury, the axon and its surrounding myelin sheath degenerate (wallerian degeneration) and its remanents are phagocytized. Schwann cells hypertrophy and Bunger bands form to guide regenerating axons. If distal endoneural tubules are not reinnervated within several months, they are slowly replaced by fibrous tissue which interferes with future axonal regeneration.[36–38] In HFA, axonal sprouting occurs from the proximal cut end of the hypoglossal nerve. The number of axonal sprouts that appear after nerve transection are thought to greatly exceed the original fiber count.[39]

In the cell body of transected facial motor neurons, there is an increased production of cytoskeletal proteins, which are important components of a structural framework to facilitate nerve regeneration.[40] Within 5 hours after injury, elevated levels of messenger RNA (mRNA) were observed, which are also thought to be involved with triggering the neuronal regeneration. Haas et al.[41] in 1993 reported a unique pattern of early gene (C-jum, junB,TIS) expression after rat facial nerve transection. Molecular biology studies of nerve repair demonstrate that rat facial motor neuron death following transection can be prevented with in vivo exposure to neurotransmitter 4/5, insulin-like growth factor 1 (IGF-1), and leukocyte inhibitory factor (LIF).[42] Neurotrophic factors influencing nerve growth in adult systems have been identified; for example, ciliary neurotrophic factor (CNTF) when applied to rodent muscle can induce motor nerve sprouting.[43] Insulin-like growth factors are signals from denervated muscle cells to promote nerve sprouting.[44] Brain derived neurotrophic factor (BDNF) has been found to prevent neonatal motor neuron death and loss of choline acetyltransferase (CHAT) following peripheral adult axonotomy.[45,46] Spector et al.[47] found that early myelinated axon counts were higher in regenerated facial nerves utilizing nerve growth factor (NGF). Differences, however, in gross muscle activity or muscle innervation patterns were not noted.

Denervation of skeletal muscle ultimately leads to irreversible fibrosis following loss of contractible proteins and muscle mass, particularly if reinnervation is not achieved in a "timely" manner. Irreversible muscle fibrosis can occur 3 years after denervation, although this time profile can vary up to 20 years.[48] Such variability indicates that laboratory investigation has not clearly established a consistent end point beyond which facial muscle cannot undergo reinnervation or functional recovery. It has been shown that the contractile properties of reinnervated muscle fiber and its type of myosin can be influenced by the reinnervating motor nerve. For example, fatigue-resistant fast-acting muscle fiber may be changed to a more easily fatigued, slower-contracting one by a reinnervating nerve anatomically different from the original nerve of innervation.[49–51] Denervation of muscle can affect an increase in DNA, mRNA synthesis, and reappearance of cell adhesion molecules in muscle cell membranes; the latter are usually found only in the embryologic muscle.[52] Skeletal muscle denervation may activate genes for acetylcholine receptors, which are also thought to maximize reinnervation of muscle.[53]

Despite the above examples of incipient advances in molecular biology of nerve and muscle injury and regeneration, clinical reports of HFA still form the basis for establishing the optimal time for HFA repair following the onset of facial paralysis.

In 1974 Conley[54] reported results of HFA in 10 patients, without evidence of meaningful electrodiagnostic activity, with time periods of facial paralysis extending from 2 to 33 years. One patient with paralysis for 10 years, however, achieved good improvement following treatment with HFA. Poor results were seen in patients with paralysis over a period of 10 to 33 years in whom severe atrophy of facial nerve was clinically observed at time of surgery. No correlation with age was noted.

Gagnon[55] had similar findings in five patients with paralysis of 2.5 to 7 years: the degree of nerve atrophy was more important than the duration of paralysis. His results also seemed better in younger patients, although his study was too small for statistical validity. He concluded that gross intraoperative assessment for nerve atrophy was the best available method for predicting successful facial reinnervation following HFA.

Alexander[16] noted that his best results occurred if the HFA was performed within 2½ years. This is consistent with the work of Yanigahara,[56] who also found that his best results were obtained if HFA was performed within 3 years following paralysis. Others advocated performing the anastomosis as early as possible or within 1 year of paralysis [23,26,57–59]

Ylikoski[60] histopathologically examined distal facial nerves from patients with facial paralysis ranging from 17 days to 30 months before undergoing HFA. Fibrosis was found in one patient (with paralysis of 7 months) who failed to improve in facial function after HFA. Endoneural tubule formation was observed in three of the remaining four facial nerves, and all of these patients had clinically improved facial function after HFA. Collagenization of the endoneural structures in the distal facial nerve was completed within 3 months followed by little change for, at least, up to 30 months. It was thought that severe fibrosis of the distal facial nerve of the single patient without facial improvement might be due to a clinically undetected infection following tumor resection or significant "individual variation in the extent of collagenous change."

Electromyographic assessment of facial muscle ability to receive reinnervation after periods of facial paralysis greater than 18 months was suggested by Crumley.[61] Fibrillation potentials certainly would allow one to anticipate optimal reinnervation in contrast to the absence of "electrical silence" EMG activity. Prognostic information is less clear when intermediate waveforms (polyphasic or giant waves) are observed. It must be kept in mind that EMG evaluation for muscle viability is a function of which muscle fibers are sampled. The EMG activity for a given muscle sampled may not reflect the status of other muscles throughout the face, possibly leading to an unreliable assessment of facial musculature viability.

Other authors have alluded to muscle biopsy to determine the extent of muscle degeneration and fibrosis.[59] Again, this technique can be plagued with sampling problems, since there can be wide variability in muscle survival throughout the face. Other factors affecting facial muscle degeneration include degree of facial nerve injury with variable quantity of nerve fibers surviving, collateral innervation from other cranial nerve (trigeminal) systems in the face, neurotization from adjacent nerve or muscle, and rich vascular support in the face.

The clinical experiences cited suggest that facial muscle reliably retains potential for vigorous reinnervation up to 30 months. Nevertheless, there seems to be a meaningful amount of early-onset muscle degeneration, as demonstrated by Terzis' technique[62] of "babysitting" newly denervated facial muscle with immediate ipsilateral hypoglossal reinnervation until contralateral cross facial-facial axonal growth is completed over several months. The success of "babysitting" technique suggests that a critical mass of viable muscle fibers is required for the successful reinnervation by the limited number of axons seen with the cross facial-facial grafts. It is assumed that the classic HFA has a far greater number of axons available for reinnervation of the distal facial nerve, making moot this early onset, limited loss of muscle fibers. This large number of regenerating axons may be a factor in the reliability of HFA for facial reinnervation when compared with other methods of reanimation.

Despite the above ancillary techniques to assess neuromuscular capability for reinnervation and clinical reports of successful facial reanimation after many years of paralysis, there is an informal concensus that for reliable results, HFA should be performed within 3 years of the onset of paralysis. Gross intraoperative assessment of facial nerve atrophy is recommended, but realistically, it is nearly impossible to differentiate intraoperatively the degrees of atrophy to predict reliably whether reanimation will be successful.

Synkinesis

Mass movements of the face reanimated by HFA were noted in all clinical reports.[16,17,22–30,32–35] Lipschitz,[63] as cited by Ford and Woodhall,[64] in 1906 postulated that misdirected regenerating nerve fibers that grew into all parts of the degenerated facial nerve trunk produced a diffuse spread of stimuli. This synkinesis, noted with proximal facial nerve injury and regeneration, certainly will be present, if not exacerbated, in cases with crossover reinnervation from alternative motor neurons (i.e., the hypoglossal nerve) whose proximal tubules certainly are not organized relative to the distal facial nerve. Miehlke[65] commented on the inevitable dilemma of synkinesis following HFA:

> . . . the better the reinnervation, the more pronounced are the secondary deficits. In a uniform assessment of innervation and deficit healing, therefore, the positive point of good reneurotization is cancelled out by the negative point of accompanying synkinesis.

He believed that slight synkinesis should be considered acceptable as opposed to "pathological synkinesis," which he reviewed as an important parameter of the severity of paresis. If synkinesis involved three or more regions of the face, it was considered "pathological" in his grading scale.

Synkinesis is frequently encountered with extreme tongue movements to produce "grimacing." Falbe-Hansen,[23] however, identified many examples of isolated, discrete movements without synkinesis. He did find that when maximal voluntary force was used to produce an isolated regional movement, generalized associated mass movements always resulted. He suggested that synkinesis be managed with reduced activity from the tongue. Reduced activity from the contralateral normal side was also necessary to minimize asymmetry. Alexander[16] observed that the best functional results after HFA were with patients who rarely smiled and displayed little emotional expression, since strong involuntary facial movements of expression (smile and laughter) accentuated the assymetry, particularly at the mouth. In fact, he noted the best symmetry in

two patients who were blind with quiescent facies and little visually perceived emotional expression. It is implied that facial expression is learned behavior based on visual observation during human interaction, which may explain why the blind patients had the least facial expression.

Electromyographic recordings from the tongue and facial musculature after HFA revealed that voluntary lingual movements and mimetic muscles can function independently to an amazing degree.[2] Stennert[2] noted that hypoglossal motorneurons can be so "autonomous that single facial areas can be activated, up to a certain degree, independently from each other." He allowed that he "never before observed such innervatory independence of separate areas of expression after direct facial nerve reconstruction" (primary facial nerve or interpositional graft anastomosis). Stennert suggested that the hypoglossal nucleus has the potential for re-education, in contrast to the facial nuclei. If we recall that much of hyoglossal activity in humans involves continuous learning and modification, especially with regard to speech and language, it is not unreasonable to contemplate the educable potential of this cranial nerve system. Stennert hypothesized that any reduction of synkinesis following direct facial nerve reconstruction was probably from a more efficient use of musculature instead of modification of neural activity in different regions of the face.

Regional control of discrete muscle groups can be increased with anastomosis between the regenerating motor nerve source and peripheral facial nerve branches instead of the distal nerve trunk.[27,66] Separate subsystems "reinnervating" different areas of the face should reduce synkinesis. For example, the eye can be managed with a gold weight with upper lid implantation and the mid/lower face is reinnervated with HFA. Others have found that it is helpful to have neural reinnervation of the ocular sphincter for the infrequent case of gold weight rejection.[67]

Botulinum toxin is an adjunct to manage synkinesia by reducing regional activity.[68] This method has the risk of weakening muscle activity to the point of deinnervating the specific area under consideration. Reduction of axonal input to the distal facial nerve trunk may prevent severe synkinesia.[59]

While strong mass movements can be seen after HFA, there is one area of the face that rarely shows muscle activity: the forehead.[16,17,23,30,69] Most series reported few cases, if any, of forehead reinnervation, which is usually only with maximal tongue movement. Gavron and Clemis[27] contemplated that the temporalis frontal branch's "right angle take off" from the main trunk was too acute for the reinnervating axon to negotiate. Stennert,[70] however, demonstrated EMG recordings from the frontalis, procerus, depressor supercilii, and components of orbicularis oculi muscles showing evidence of "reinnervation" in postoperative HFA patients. Cross innervation of all these antagonistic muscles of the forehead lead to ineffective function with contraction, a process he called "autoparalysis," in which the components of reinnervated frontalis musculature cancel out each other's activity. Electrophysiologic studies after HFA also recorded highest amplitudes, lowest thresholds, and the longest latencies from the mentalis muscles compared with shorter latencies, higher thresholds, and lowest amplitudes from the orbicularis oculi region.[71] Axonal regeneration and muscular reinnervation were most vigorous in the lower facial area, least in the upper region, and midway in the midface.

Function: Hemilingual Atrophy

Distal hemilingual denervation raises concern about dysfunctional intraoral manipulation of food, swallowing, and speech. Ipsilateral hemilingual atrophy and tongue biting also may be bothersome. Pensak[29] found that 74% of respondents had some degree of difficulty with eating, especially with mobilization of food on the paralyzed side. An unspecified "few" also had problems with swallowing. Clemis and Gavron[28] noted that their patients had immediate HFA postoperative problems with intraoral manipulation of food warranting intensive oral hygiene (i.e., use of water pick and close dental follow-up). These problems diminished as buccinator and lip musculature became reinnervated. While all 43 patients who underwent delayed HFA had masticatory problems, Conley[33] noted that only 16% had a similar problem after early facial reanimation. In his report, Conley wrote that 12% had swallowing difficulties compared with none before HFA. This finding was in contrast to the experience of those who underwent immediate HFA, postoperatively producing a much lower incidence of problematic mastication (3%), swallowing (2%), and speech (2%).

Of patients who had preoperative difficulties with swallowing, particularly those with cranial nerve IX and X deficits, dysphagia persisted or worsened following HFA.[28] Lower cranial nerve dysfunction may account for Evans' single case[24] of exacerbated dysphagia after HFA, although he did not comment on lower cranial nerve function in his series.

Ballance[17] did not believe that hypoglossal dysfunction was severe enough to warrant "preservation" of ipsilateral lingual function with an end-to-side anastomosis. In his report, problems associated with hemilingual dysfunction were not addressed. It is possible that patients at the turn of the century were less assertive about this problem. Functional results following the end-to-side anastomosis may have been unsatisfactory to Ballance, since he did not have access to the operating microscope, which became available in the 1950s. Lahey's concerns about speech problems following HFA were dismissed by Coleman, who cited his experience with voice recordings from his patients to demonstrate unaffected speech.[22] Other reports of blurring of consonants in almost half their patients categorized this deficit as subtle. Falbe-Hansen[23] contemplated that the speech and intraoral problems might be associated with the trigeminal nerve deficits found in a large number of his patients.

Attempts to reinnervate the ipsilateral tongue with hypoglossus descendens branch to the distal hypoglossal trunk were generally met with either failure or equivocal results. Conley's three cases[33] did not alter ipsilateral lingual bulk when compared with their noninnervated cases. Kessler[26] achieved function verified by EMG recordings in only two of five cases. Most authors agreed that there was no meaningful difference after reinnervation with the hypoglossus descendens, even when it was "successful,"

when compared with actual functional results following lingual dennervation.[61]

Most reporters have observed that loss of hypoglossal function following HFA have minimal disabling sequelae unless there are concomitant ipsilateral lower cranial nerve deficits. Clemis[28] even suggested that the loss of tongue bulk contributed to successful intraoral manipulation of food. The overall lack of sequelae associated with hemilingual function following hemiglossectomy for cancer was cited by Conley, which was to suggest that post-HFA hemilingual symptoms would be minimal.[23] While all series emphasized successful restoration of speech and oral function, for some there was still a lingering doubt about hemiglossal deinnervation. As observed by Evans,[24] "the unwanted effects of the operation are unescapable, but in general are well compensated for the advantages."

Lagophthalmos

Facial paralysis adversely affects ocular protection with loss of tearing following lacrimal gland deinnervation and failure of active lid movement to lubricate the cornea with tears and protect it. Insufficient eyelid sphincter support causes ectropion, epiphora, conjunctivitis, and keratitis leading to visual impairment. Loss of vision from neuroparalytic keratopathy is most likely in those with the dry eye, poor Bell's phenomena, and corneal anesthesia from loss of trigeminal function.[72] While some series cited were notable for absence of problems due to lagophthalmos,[16,23–28,33–35] 21% of respondents to a follow-up questionnaire encountered ophthalmologic deficiencies including dryness (29%), irritation (17%), visual impairment (11%), ocular pain (9%), and epiphora (4%). Others were bothered by corneal abrasion, neurotrophic keratitis, and problems with lateral tarsorraphy.[29]

Many of the reports reviewing experience with HFA did not specifically address lagophthalmos. Evans[24] acknowledged that all but one of his patients had lateral tarsorrhaphy. Other procedures to provide static support include canthoplasty and implantation of autologous tissue.[73,74] Dynamic support can include implantation of upper lid gold weights, magnets, palpebral springs, silicone bands, temporalis muscle suspension, and free muscle grafts.[75–78]

Extrusion of nonautologous implants have occurred.[79] Therefore some authors have preferred to rely more on dynamic autologous reinnervation (e.g., free muscle grafts or nerve-to-nerve anastomosis. EMG biofeedback rehabilitation was believed to contribute toward achieving satisfactory orbicularis oculi function to permit reversal of tarsorrhaphy in 13 of 17 patients after HFA dynamic reanimation.[30] Artificial tears, lubricants, moisture chambers, wraparound glasses, and windshield barriers are adjuncts to the management of lagophthalmos that are not usually mentioned in reviews on HFA.[73]

Emotional Expression

Early advocates of HFA commented on the closer anatomic relationship between facial motor cortex and hypoglossal motor cortex when compared with other cranial nerves (i.e., the spinal accessory or glossopharyngeal), which might suggest closer temporal and functional similarity from a potential association at the cortical level. Others have observed that involuntary intraoral lingual movements concomitant with emotional liability might effect "appropriate" facial movements following HFA.[25,80,81] While Shroder[82] and others[83] have commented on direct ipsilateral projections from spinal trigeminal nucleus (STN) to the hypoglossal nucleus (HN) in cat and rat, there is still a paucity of information about the neuroanatomy and regulation of emotional expression.

Voluntary facial movement and involuntary emotionally induced facial movement use separate pathways. Efferent fibers for voluntary facial motor function emanate from the motor cortex and course to the facial nuclei through the corticobulbar projections, also known as pyramidal tracts. Impulses for emotional facial movements arise from the phylogenetically older extrapyramidal system, which commences from subcortical regions to travel to the facial nucleus.[84]

Patients with lesions of the cortical motor strip or corticobulbar projections do not have contralateral voluntary lower facial movements but do retain bilateral involuntary emotional movements. In this instance of corticobulbar paralysis, the facial nucleus is still innervated by a separate pathway, the extrapyramidal system. Conversely, patients with parkinsonian lesions of the basal ganglion of the extrapyramidal system lack spontaneous facial movement ("mimetic facial paralysis") while preserving voluntary facial function. The pyramidal (corticobulbar) system from the cortex is phylogenetically more recent and is associated with learned "cortical" behavior. Patients with corticobulbar (internal capsule) lesions seen with multiple sclerosis, amyotrophic lateral sclerosis, anoxia, or stroke of the internal capsule have pseudobulbar palsy, which lacks inhibitory cortical control of inappropriate nonemotional laughing or weeping facial expression.[85]

The corticobulbar system is capable of modulation of facial movement based on cortical motor input, which is associated with frontal lobe development in the human. This cortical behavior forms the basis of learned social etiquette of facial behavior. In contrast, the subcortical extrapyramidal emotional response is a more direct primitive reflex that can be seen in infants or congenitally blind children crying or laughing. These genuine, deep emotional facial movements are not cortically mediated nor learned through imitation.[86] While for the most part there is modulation of behavior and facial expression, there are times during which it is difficult to inhibit genuine expression.

Schemm[87] observed strong voluntary movement, but he did not see genuine emotional movement after nerve crossover facial reinnervation surgery. Rinn[88] postulated that the more primitive motor centers of the emotional extrapyramidal pathway lack the educable plasticity of the cerebral cortex. Voluntary facial movement following crossover nerve anastomosis can be learned. In contrast, the subcortical centers for emotional movement continue to send their signals to the disconnected proximal facial stump, instead of learning to activate the alternative crossover nucleus.

Although the concept of separate facial pathways can explain why there is little emotion expression following a crossover anastomosis, it cannot account for the failure to

elicit emotional movement after a primary direct facial nerve reconstruction or repair with an interpositional graft.[2] One would anticipate some degree of emotional movement in this instance, since the subcortical extrapyramidal pathways to the facial nerve are intact. Distal facial nerve injury and the misdirection of regenerating fibers must inexplicably influence the mechanics of emotional movement. Elucidation of these affects on facial expression with "intact" proximal facial nerve await the results of further investigation.

Clinical reports have commented on mimetic movements observed in association with talking, which can give the face an "incipient" expression. All reports, however, have declared that there is no emotional expression except for what accompanies lingual movement. The absence of facial movement associated with strong spontaneous contralateral activity centered around the mouth (i.e., during laughter) is accentuated because this central facial region requires synergistic bilateral muscle activity for symmetry, unlike other parts of the face. The myth of restoration of natural emotional expression by a type of surgical endeavor has been replaced by the understanding that there is no known procedure that can return the face to emotional normalcy.[83]

Hypertonia

Several authors commented on hypertonus in their series, but the precise number of cases affected was not made available.[16,22,30,33] Facial grimacing, with tongue activity, extreme facial movements, or contractures and facial spasms, may be seen with hypertonia. Facial spasms and contractures may more commonly be seen with recovered injured facial nerves and rarely with HFA. Facial spasms are more accurately thought to be secondary to synkinetic clonic movements produced by "maldeveloped motor neurons by which the R2 component of the trigeminal-facial reflex (nictitation) is conducted."[65] Consequently, these movements are synchronized with nictitation and should be termed "generalized blinking" instead of facial spasm.[65] Excessive facial movement is considered a function of neural input exceeding that required for optimal facial activity. Conversely, hypotonia is related to an insufficient number of reinnervating axons and viable facial muscle fibers. Modulation of hypertonia can be achieved with reduced axonal supply, as seen with May's jump graft procedure, delay in anastomosis to allow for nerve and muscle fibrosis, inhibition of neuromuscular function with botulinum toxin, and EMG rehabilitation for modification of facial function.[15,59,68] Hypotonus may be modified with EMG rehabilitation to optimize muscle function but other augmentative procedures such as muscle grafts may be required.[14,30]

Reinnervating Nerve of Preference

Although reinnervation of the distal facial nerve was initially performed with crossover nerve from the spinal accessory, the majority of the literature, especially in the United States, then advocated the hypoglossal nerve as the new motor nerve of choice.[17,25,27] The reasons for this are manifold: closer temporal and functional similarity between chewing, talking, and swallowing in conjunction with facial movement; closer cortical anatomic proximity; minimally visible intraoral dysfunction; avoidance of painful shoulder drop and voluntary facial movement with gross shoulder movement; and embarrassing facial movement with shoulder activity. Poe reported achieving facial reanimation with the spinal accessory nerve without shoulder paralysis by mobilizing the branches to the sternocleidomastoid muscle. These nerve fibers comparatively contain 29% of the axon volume seen in the facial nerve, which was still thought to be sufficient for reinnervation.[89,90] Burgess and Goode,[59] however, "suspect" that a higher number of axons should be used. There have been early, scattered reports of reinnervation with glossopharyngeal and phrenic nerves, which have been abandoned in favor of hypoglossal nerve crossover.[17,22,23,26]

In addition to the phylogenetic synergy of the facial trigeminal and hypoglossal cranial nerves for sucking, chewing, and swallowing, Stennert[2] cited anatomic reports of rat neuronal projections from the bulbar lateral tegmental field traversing to the motor nuclei of trigemenal, facial, and hypoglossal nerves. These anatomic connections were bilaterally organized.

Electrophysiologic recordings from patients after HFA produced trigeminal-hypoglossal reflexes with latencies and amplitudes comparable to trigeminal-facial reflex ("blink reflex"). Moreover, 63% of the subjects had demonstrable R1 components recorded from ipsilateral tongue suggesting reflex arc connections between the trigeminal and hypoglossal nerves. Postsynaptic inhibition recordings from the tongue following a maximal R2 reflex response from the orbicularis oculi muscles support the assumption that facial hypoglossal motorneurons have common interneurons that are activated by trigeminal afferents.[91] It is interesting to reflect how Korte,[10] almost 100 years ago, anticipated these phylogenetic anatomic and physiologic observations when contemplating possible intracranial communications between hypoglossal, facial, and trigeminal nuclei:

> It is a most remarkable physiologic fact that ganglion cells of the hypoglossal or the accessory nerve nucleus can acquire under voluntary influence the ability to induce contractions of muscles originally innervated from other nerve centers. We cannot yet decide whether there are connections between the two areas of nerve ganglia, or whether by will-power the nerve center can adapt or become accustomed to stimulating a newly acquired muscle system [translated by Stennert and Limberg[91]].

Electromyographic Rehabilitation

Electromyographic rehabilitation, sometimes called neuromuscular retraining (NMR), has been used at several centers to enhance facial reanimation.[30,92,93] Ross and others have demonstrated the efficacy of neuromuscular feedback training in long-standing paralysis and following facial reinnervation surgery.[94]

Individual facial rehabilitation strategies are varied but are specific and learned for a given problem area. Typically, facial muscle activity is monitored with visual and auditory feedback signals from surface EMG electrodes recording from normal contralateral and ipsilateral facial

muscle groups (Fig. 1). With slow execution of discrete facial muscle movements, new motor strategies are developed and learned. Small muscle excursions are used to preserve isolated regional movements and to control recruitment of adjacent muscle groups from neuronal overflow associated with large facial movements. This approach leads to enhanced coordination of small facial movements and reduction of synkinesis. Practiced concomitant control of offending recruiting muscle groups also contributes to inhibition of synkinesis. Ultimately these and similar strategies lead to improvement of muscle activity in weaker muscle groups to reduce hypotonia, normalization of muscle resting tone for decreasing hypertonia and synkinesis, more precise control of muscle group activity to facilitate regionalization, and better symmetric activity and augmentation of emotional expression.

In patients following HFA reinnervation, facial movements are initiated with lingual activity only until the patient can make minimal facial movements without tongue movement. After this milestone, the patient learns to inhibit facial movements during tongue activity, which reduces aberrant facial movement seen during speech and chewing. Ultimately there is a dissociation of inappropriate facial activity from lingual movements.

Diels has hypothesized that neuromuscular rehabilitation may "allow use of emotional input to establish more natural motor control of paralysis" based on the concept of neural "plasticity" and that the extrapyramidal motor system for emotional expressive movements is a different upper motor pathway than the pyramidal pathway for voluntary facial activity. It remains to be seen if this extrapyramidal pathway for emotional expression can be recruited or facilitated with NMR.[93] Although objective reports about emotional facial expression following NMR are not available, there is a definite appreciation of the need for sufficient mimetic restoration, which is still lacking with all current methods of facial reanimation.

JUMP INTERPOSITIONAL GRAFT HYPOGLOSSAL FACIAL ANASTOMOSIS

Because of difficulties with speech, chewing, and swallowing from hemilingual deinnervation and inability to use HFA in patients with concomitant lower cranial nerve deficits or bilateral facial paralysis, May[15] developed an alternative procedure with an interpositional graft for facial reanimation. It is derived from the classic HFA and Terzis's "babysitting" technique[62] for maintaining maximum number of distal facial motor neuromuscular structures viable with a temporary nerve supply until the anticipated crossover facial-facial anastomosis or free muscle implant is ready. The procedure entails placing an interpositional nerve graft between a partially transected hypoglossal nerve trunk, distal to the hypoglossal descendens, and the distal facial nerve trunk or its branches. May frequently combined this procedure with other reanimation techniques depending on the specific areas of weakness.

All 23 patients achieved facial tone and symmetry at rest; excellent facial movement was present in 79% of the cases in which JIGHFA was performed within 12 months following the onset of paralysis and in 38% of those undergoing surgery between 13 and 48 months.[15] Regionalization of movement (separate eye and mouth movement) was noted in 10%. Significant mass facial movements were not encountered, in contrast to the classic HFA. Hypertonia was not observed. Supplementary procedures involved regional reanimation methods such as cross facial grafting, eyelid weight implantation, spring or cartilage implantation in 30 patients, and temporalis muscle transposition in 21 patients.[14,95]

In May's series,[15] there were 3 cases of hemilingual atrophy in 23 cases, with swallowing deficit in 1 patient, mastication problem in 1, and speech difficulty in 1. Patients with swallowing, masticatory, and speaking problems had deficits with lower cranial nerves III to VII, IX, and X before facial reanimation. Hemilingual atrophy in these three patients was believed to be from excessive transection of the hypoglossal nerve proximal to the hypoglossal descendens take-off because its diameter was assumed to be larger due to the unappreciated contribution from the fibers going to the hypoglossal descendens branch. When the partial transection was distal to the hypoglossal descendens take-off, the ipsilateral tongue function was preserved without atrophy.

Three patients had bilateral facial paralysis, two from pediatric brainstem tumors and one from bilateral temporal bone fractures.

May believed that best results were obtained following reanimation surgery when performed within 12 months of facial paralysis, although this "time window is not absolute." May emphasized that ancillary reanimation techniques significantly contributed to the excellent results, if not more than the effects of the interposition grafts. His series did not comment on or include EMG feedback rehabilitation.

Others subsequently reported successful facial reinnervation with this technique of jump interpositional graft along with the management of the eyelid with gold weight upper lid implantation. In one series of six patients, the gold weight was rejected in one patient and it was not replaced due to sufficient reinnervation of the *orbicularis* oculi muscles.[66]

To reduce synkinesis, Kartush[66] further modified this approach with reinnervation of only the inferior facial nerve branches with the jump interpositional graft anastomosed to two thirds of the hypoglossal nerve diameter along with the gold weight lid insertion for lagophthalmos.[66]

OBJECTIVES

The purpose of this study was to confirm and assess the reliability and degree of facial reanimation that can be achieved with JIGHFA along with gold weight eyelid implantation. EMG rehabilitation was used by the majority (66.7%) of the JIGHFA patients in this study. The degree of facial reanimation was measured by trained observers using the modification of the House-Brackmann scale (Table I) of Brudny et al.[30]

The outcome results were compared with Brudny's series of patients managed with the classic HFA and EMG rehabilitation. The utility of JIGHFA for patients with concomitant ipsilateral lower cranial nerve deficits was also evaluated. Particular attention was directed to noting the extent of facial symmetry, regionalization, synkinesis, hypertonia, and dissociated movements. Potential prob-

lems with lagophthalmos, speech, chewing, and swallowing were examined. Information was also included from a retrospective review by subjective questionnaire and/or chart analysis of 22 patients from a series of 48 patients managed with classic HFA and lateral canthoplasty.

PATIENTS AND METHODS

Patients

Facial paralysis was secondary to acoustic neuroma resection in 13 patients, all of whom had tumors ranging in size from 3 to 7.5 cm. One patient lost facial function during surgery of recurent acoustic neuroma. In the remaining five patients, their facial paralyses were caused by hemangioma multiform in the cerebellopontine angle, cerebellar astrocytoma, medulloblastoma, facial neuroma, and herpes zoster oticus (Table IV).

Facial paralysis was complete in all patients. With one exception, all patients with facial paralysis following acoustic neuromma surgery had transected facial nerves, and electrophysiologic tests were not obtained. The remaining patients had EMG findings consistent with severe facial nerve degeneration or denervation.

There were nine males and nine females who underwent JIGHFA over a 5-year period (1990–1995). Their ages ranged from 9 to 73 years. The intervals between the onset of facial paralysis and JIGHFA surgery ranged from 0 to 24 months with almost half (8) of the patients having facial reanimation surgery within 1 month and the majority (14) within 6 months. The longest interval was 24 months (Table IV).

Brudny's series of 30 patients, operated on by several different surgeons, with classic HFA and EMG feedback, was used as a comparative group for the patients who underwent reanimation surgery (Fig. 1).

A series of 48 patients with standard hypoglossal facial nerve anastomosis and lateral canthoplasty retrospectively reviewed with anonymous questionnaire or chart analysis when identified was made available. In this group of patients operated on between 1981 and 1990, the origins of the facial paralysis were cerebellopontine angle lesions, 45 acoustic neuromas, 1 facial neuroma, 1 meningioma, and 1 glomus jugulare. Twenty-two patients returned the questionnaire. The respondents were anonymous for the purpose of encouraging their participation in completing the questionnaire. Therefore specific data (age, sex, time profile with respect to duration of paralysis and rehabilitation) were not available. Information and comments from respondents were uniquely subjective from the patients' viewpoints, which are frequently overlooked in "objective" databases. This questionnaire was formulated before Brudny's modified classification of House-Brackmann grading scale became available (Fig. 2).

Methods

In the JIGHFA group, the ipsilateral hypoglossal nerve trunk was partially and obliquely transected up to half its diameter just distal to the hypoglossal descendens branch. An interpositional jump graft, either ipsilateral greater auricular (15) or sural nerve (4), was placed between the partially transected proximal hypoglossal nerve and the transected distal facial nerve trunk. The nerves were anastomosed without tension with interrupted perineural 9-0 or 10-0 nylon suture (Fig. 3). The JIGHFA reanimation surgery in this series was performed by the author.

In 17 of 18 patients, the eye was managed with a gold weight placed in the upper lid and if indicated, a lower lid lateral canthoplasty. Artificial tears and/or ophthalmic lubricants were used as needed. Some patients were fitted with windshields attached to the temples of eyeglasses. All patients but one had their reanima-

TABLE IV.
Patient Information.

Patient No.	Age (y)	Duration of Paralysis (mo)	Time to Reinnervation (mo)	Follow-up (mo)	EMG Sessions (n)	Class*	Etiology	Comments
01	16	2	4	24	6	III	AN	X†, SN
02	50	0	8	36	29	II	AN	Au WT removed
03	53	0	5	48	26	II	AN	Au WT removed
04	16	0	8	24	15	III	AN	Hypertonus, transient corneal abrasion
05	42	2	5	30	9	III	AN	Hypertonus, X†, Au WT removed
06	65	3	10	36	23	III	AN	Hypertonus, recurrent AN
07	15	0	5	24	3	IV	AN	
08	17	4	6	48	0	III/IV	AN	
09	71	15	8	36	0	III	AN	SN
10	40	0	6	6	0	N/A	AN	Lost to follow-up
11	51	0	5	36	22	II	AN	No Au WT
12	43	0	10	18	0	III	AN	
13	43	0	5	8	0	III	AN	
14	40	0	5	8	2	III	HM	
15	12	24	6	0	0	II	CA	V, IX, X†, SN
16	73	18	12	24	8	III	HZO	SN
17	9	16	12	8	15	III	MB	X†
18	68	0	5	24	15	III	FN	

*Facial functional classification based on Brudny modification of House-Brackman scale for facial paralysis grading through I to VI.
†Cranial nerve deficit.
AN = acoustic neuroma; HM = hemangioma multiforme; CA = cerebellar astrocytoma; HZO = herpes zoster oticus; MB = medulloblastoma; FN = facial neuroma; SN = sural nerve graft; Au WT = gold weight.

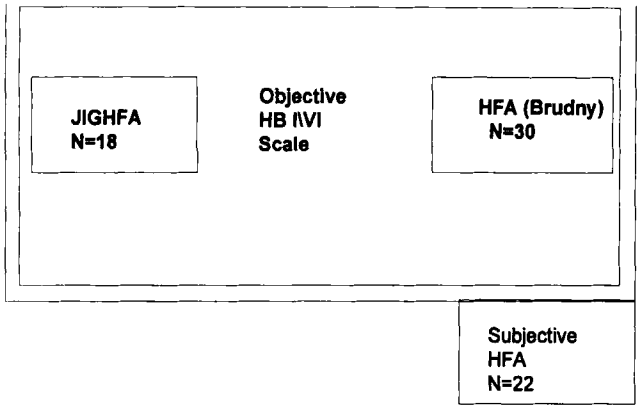

Fig. 2. Schematic rendition of patient populations.

tion surgery performed within 4 months after the advent of facial paralysis, except in patients with causes or origins other than schwannoma, as follows: herpes zoster oticus (11 mo), medulloblastoma (16 mo), and cerebellar astrocytoma (24 mo).

After facial reinnervation was evident with lingual activity, an intensive, structured EMG rehabilitation program was instituted for nine patients who were able to participate in such therapy after JIGHFA surgery (Fig. 4A–C). In the HFA surgery respondent group, 18 of 22 patients had EMG therapy. Geographic distance, logistics, and finances were the limiting factors precluding EMG rehabilitation for the remaining patients.[30,92,93]

All but three patients were followed for at least 24 months. One patient was transferred to a rehabilitation facility where satisfactory follow-up information was not obtainable. The 9-year-old girl with medulloblastoma was unaccessible while living in Israel and then in Australia. The 40-year-old patient with hemangioma multiforme was included because sufficient information concerning reinnervation was available within this short time frame of 8 months.

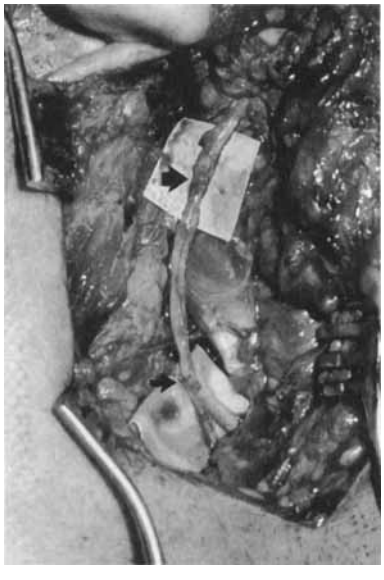

Fig. 3. Intraoperative view of jump interpositional graft (greater auricular nerve) anastomosed between main trunk of facial nerve (large arrowhead) and partially transected hypoglossal nerve (small arrowhead) distal to descendens branch.

RESULTS

Following JIGHFA surgery, facial reinnervation was visible within 10 months for all patients with paralysis from acoustic neuroma (Table IV). In 11 of 13 patients (84.6%), reinnervation was present by 8 months. Facial reinnervation was present within 5 months for the patient with facial neuroma, within 6 months for the patient with cerebellar astrocytoma, within 10 months for the patient with glomus jugulare, within 13 months for the patient with medulloblastoma, and within 18 months for the patient with herpes zoster oticus (Table IV). Facial reinnervation presented at 5 to 18 months in all patients with the majority of acoustic neuroma patients presenting within 9 months.

Respondents to the HFA questionnaire reported facial reinnervation occurring in all patients within 36 months (Table V). In 11 of 22 patients (50%), reinnervation presented within 6 months, and in 16 of 22 patients (73%) within 12 months. In the JIGHFA group, 9 of 18 patients (50%) were reinnervated within 6 months, and 17 of 18 patients (94.4%) within 12 months. Although in both groups of patients nearly all the patients appeared to eventually demonstrate facial reinnervation, there was a significantly greater proportion of JIGHFA patients with reinnervation at 12 months than in the HFA group (two-sided Fisher's Exact Test; $P = .046$). There is no significant difference at 6 months or at a minimum of 2 years.

Facial reanimation function was rated with the Brudny modification of the House-Brackmann grading scale.[30] For the JIGHFA series, grade II/VI was obtained by 4 patients (22%) followed by 11 patients (61.1%) with grade III/VI rating (Table IV). There were two patients (11.0%) with grade IV/VI, and no information about one (5.5%) patient. EMG rehabilitation was used by 2 of the 4 patients who were rated grade II/VI and by 8 of 10 patients with grade III/VI scores. The two patients with the poorest (grade IV/VI) ratings had the least number of EMG sessions: three and two sessions, respectively (Table IV).

In the HFA series, the responses to the questionnaire demonstrates a rough correlation between facial function (symmetry) with movement and percentage comparison with normal contralateral side (Table VI). For example, the extreme ends of the scale have similar numbers: five patients with "good facial symmetry" correspond to five patients in the 76% to 100% range, and six with poor facial symmetry to the five in the 0% to 25% range (Table VI; Fig. 5). A rough correlation between the two subjective evaluations (adjectival and percentile scale) is demonstrated except for the highest ranks (superb [normal]). Since normal (superb) function was not anticipated, the "good symmetry" group was not subdivided for excellent/superb categories, which makes it difficult to correlate with further precision the adjectival best functional results within the top percentile group.

The Brudny modification of the House-Brackmann scale for grading facial paralysis was applied to the respondents' stratification percentile with grade I being normal, grade II corresponded to the 76% to 100% class, grade III applied to the 51% to 75% level, grade IV matched with the 26% to 50% level, and grades V and VI approximated to the 0% to 25% group (Table VII). Such

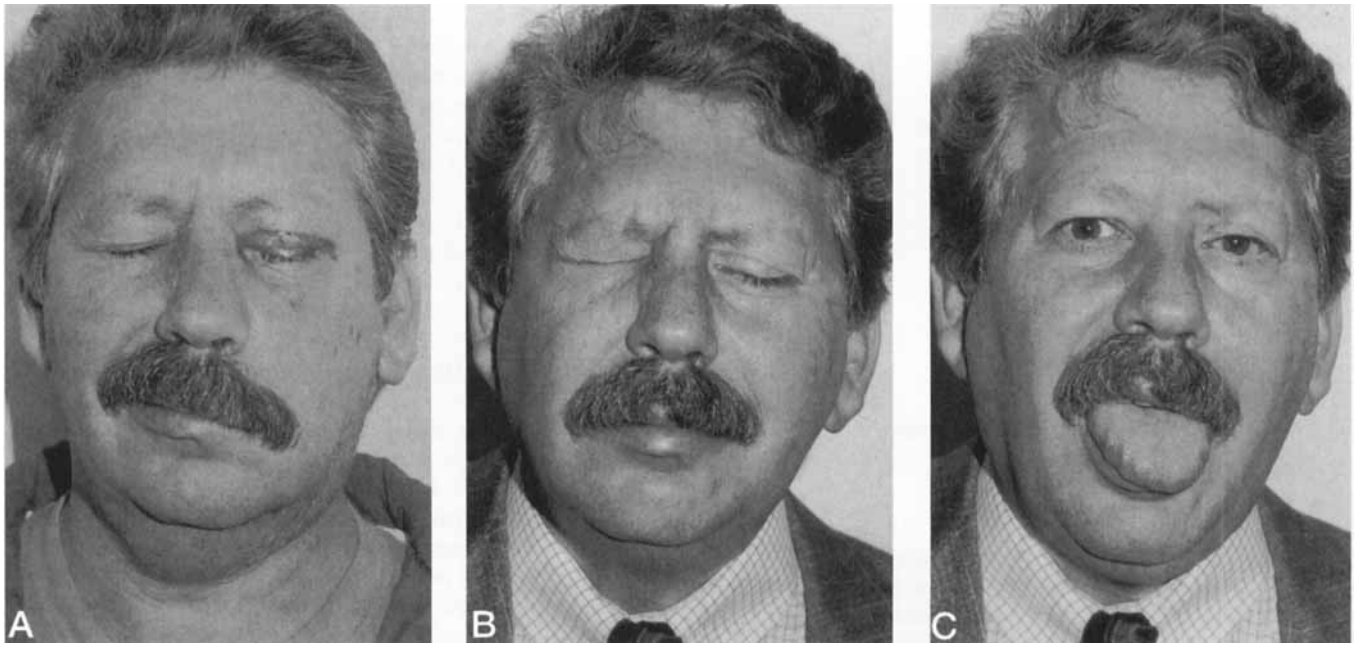

Fig. 4. Before **(A)** and 12 months after **(B)** jump interpositional graft hypoglossal facial anastomosis (JIGHFA) facial reanimation with EMG feedback rehabilitation. Patient has ointment and suture following insertion of gold weight in upper lid. **B.** There is good eye closure (following removal of gold weight), facial tone, and nasolabial fold symmetry. The balanced smile is symmetric but limited. **C.** Intact left side of tongue without hemiatrophy or eye closure with lingual protrusion. Mild asymmetry of nasolabial fold is evident.

transposition of facial paralysis grading systems between subjective evaluation and objective ratings have been used in other studies.[35] However, in our HFA series this extrapolation is based only on subjective responses using different criteria. The Brudny, House-Brackmann scale is only for the purpose of applying a "relative" relationship and place for descriptive purposes. It is not to suggest that these categorizations truly meet the criteria listed in Table I. The subjective HFA series show five patients achieving a rating of grade II (23%), seven attaining grade III classification (32%), five classified on IV (23%), and five stratified in the least successful group, grades V to VI (23%).

In the JIGHFA series, the gold weight was rejected by one patient and electively removed at the patients' request in two. Since the orbicularis oculi muscles were sufficiently reinnervated, further oculoplastic rehabilitation was not warranted (Fig. 4B). One patient did not have a gold weight placed. There were at least two patients (11.1%) with eye discomfort, one of whom also had transient corneal abrasion (Table IV).

Respondents following HFA surgery with lateral canthoplasty reported satisfactory voluntary eye closure in 17 of 22 patients (Table VIII). Synkinesis with eye closure was bothersome for six patients. Vision was noted to be poor in 2 patients (9%), not usable in 2 (9%), and fair to good in 18 (82%). Artificial tears were used by 11 patients, and lubricants were administered by 13. Five patients underwent tarsorrhaphy, and five reported corneal anesthesia. Three respondents had epiphora, and one noted gustatory tearing associated with eating grapes. Visual problems from drooping eyebrow were reported by three, and a sagging upper eyelid affected two respondents. In summary, while 17 (77.3%) patients reported satisfactory eye closure, severe visual difficulties occurred in at least 4 (18.2%) patients—all of whom had corneal anesthesia (Table VIII).

Hemifacial hypertonia was observed in 3 of the 18 patients (16.7%) who underwent JIGHFA surgery, and in 8 of 22 patients (36.7%) from the HFA questionnaire group (Table IX). A statistical significance is not observed between the two groups (HFA questionnaire and JIGHFA) re-

TABLE V.
Facial Reinnervation Time.

	≤6 Mo	≤12 Mo	≤24 Mo	≤36 Mo	Total
HFA (N = 22)	11 (50%)	16 (73%)	21 (95.4%)	22 (100%)	22
JIGHFA (N = 18)	9 (50%)	17 (94.4%)	17	17	17

The hypoglossal facial anastomosis (HFA) information is based on patient response to questionnaire in contrast to the information from chart review for the jump interpositional graft hypoglossal facial anastomosis (JIGHFA) patients. There is a significantly greater proportion of JIGHFA patients with reinnervation at 12 months than in the HFA group (two-sided Fisher exact test; $P = .046$). There is no significant difference at 6 months or 2 years. Note that with less than 50% response rate in the HFA group, the time profile of reinnervation in this group may not be accurately depicted for the entire HFA group.

TABLE VI.
Patient Response to Questionnaire After Hypoglossal Facial Anastomosis Surgery (N = 22).

	Yes	No
Facial symmetry in repose	21	1
With voluntary movement		
Good facial symmetry	5	17
Fair facial symmetry	14	8
Poor facial symmetry	6	16
Is there any movement on the paralyzed side?	18	4
Overall, has your facial function improved compared with prior to HFA?	20	2
Compared with the normal side, how much facial function has been obtained with your HFA (normal side = 100%)?		
0%–25% 5		
26%–50% 5		
51%–75% 7		
76%–100% 5		
Total 22		
Do you have facial hypertonus (spasticity) or excessive muscle tightness on the operated side?	8	14

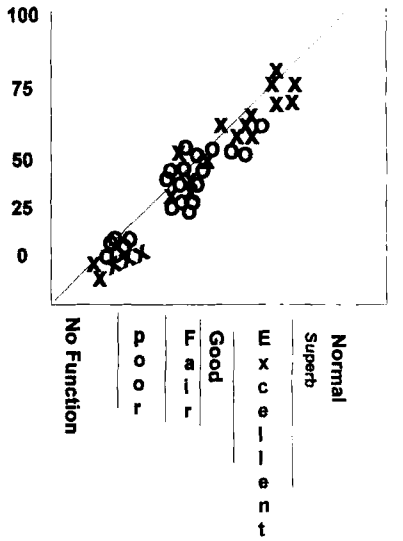

Fig. 5. Relationship between percentile subjective rating and adjectival rating for hypoglossal facial anastomosis (n = 22) group of patient respondents.

lated to hypertonia. The incidence of hypertonia was not noted in Brudny's series. Those performing EMG rehabilitation for our patients emphasized that they found hypertonia following JIGHFA markedly less severe and considerably easier to ameliorate compared with the hypertonia associated with HFA surgery (Brudny J, personal communication, December 1994). Dissociated facial grimacing was not noted in either group (HFA respondents or the JIGHFA series).

Initial hypotonia was observed in at least 10 of the 19 patients following JIGHFA surgery after reinnervation became visible. While the hypotonia was not quantified, subjectively it was more noticeable by this viewer and the EMG rehabilitation physicians in terms of frequency and intensity when compared with the HFA patients. During the several months after the onset of facial reinnervation, this hypotonia in the JIGHFA patients became less obvious, especially after EMG rehabilitation.

Symptomatic synkinesis was noted in 27.3% HFA respondents and in 44.4% from the JIGHFA series (Table IX). There is no statistically significant difference between the two groups. To this viewer and trained EMG rehabilitation and professionals, the synkinesis was definitely less intense in the patients after JIGHFA when compared with those with HFA. In contrast to the subjective respondents' replies, this viewer and EMG rehabilitation associates recall that all HFA patients initially had some degree of synkinesis which was more difficult to control with EMG rehabilitation when compared with the JIGHFA patients (Brudny J, personal communication, December 1994).

Ten of 22 (45%) HFA respondents indicated that they had difficulty with speech, and two had problems with biting their lip or tongue (Table IX). Transient drooling was noted by 14 patients, followed by 2 with long-term drooling. Swallowing difficulties were reported by 10 (45%) of the HFA patients (Table IX). One respondent who rated her facial function as 75% to 100% compared with the normal side, nevertheless, commented as follows:

> Sometimes one wonders if the loss of the use of half the tongue and the ability to chew on one side, both of which contribute to a difficulty in eating, chewing food sufficiently and some speech difficulty was worth the gain. Perhaps, there is a better way . . .

In contrast, none of the JIGHFA patients noted problems with speech, mastication, and swallowing (Table IX) (Fig. 6). The difference is statistically significant (P = .001). In the JIGHFA group, there were four patients with lower cranial nerve deficits, including three with ipsilateral nerve X deficits and one with nerve IX dysfunction (Table IV). Hemilingual atrophy was absent in the JIGHFA group.

While 20 HFA respondents noted good oral commissure symmetry at rest, only 8 (40.0%) believed there was good symmetry with voluntary movement (Fig. 6). With involuntary smiling/laughter, none of the respondents believed that they had symmetry of movement seen with emotional expression (Table X). In the JIGHFA series, similar limited spontaneity occurred in 4 of 18 patients (22.2%). In actuality, none of the reinnervated patients, regardless of type of surgery, had symmetry concurrent with deep spontaneous facial expression; there was, however, some movement suggestive of a sense of emotion in synchrony with contralateral emotional expression.

DISCUSSION

Our experience with JIGHFA demonstrates that it is a reliable technique to achieve facial reinnervation in properly selected cases. Of the patients in this series, 83.3% (22% 61.1%) attained (Brudny modification of House-Brackmann scale) grade III or better, which may be better than the HFA results (64.5%) cited earlier from the literature (Table II).

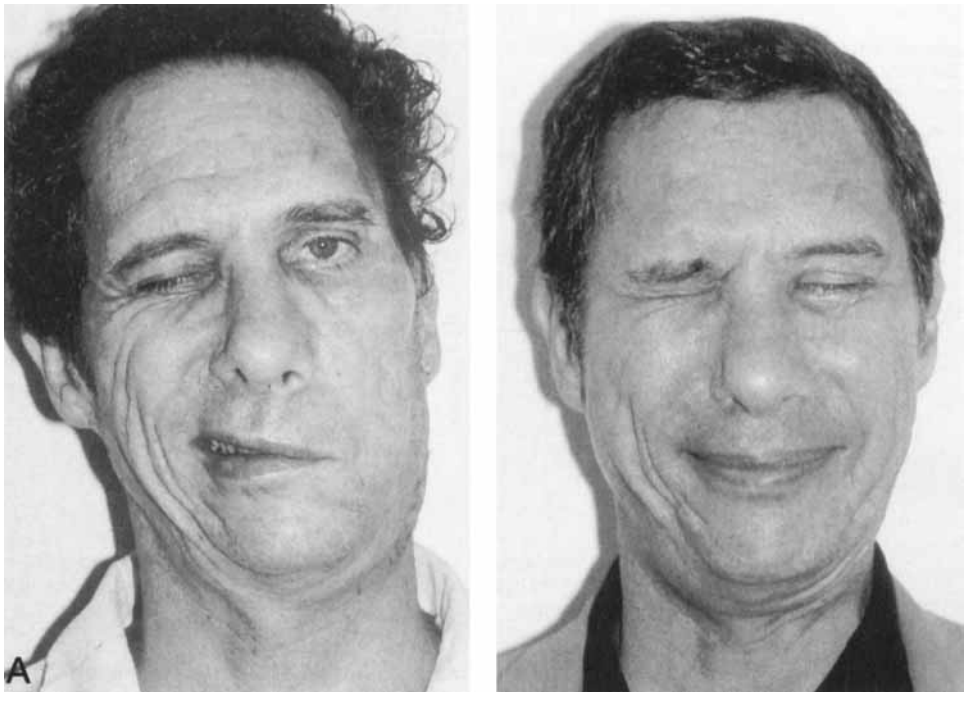

Fig. 6. Before **(A)** and after **(B)** facial reanimation with hypoglossal facial anastomosis (HFA), lateral canthoplasty, and electromyographic rehabilitation. Twelve months after facial reanimation surgery. Observe forceful contraction in **B**, and compare it with less powerful facial contracture in Figure 4 after reanimation with jump interpositional graft hypoglossal facial anastomosis (JIGHFA). This stronger contraction activity is more commonly observed in patients with HFA compared with the JIGHFA reanimation procedure.

This outcome of JIGHFA surgery is also comparable to Brudny's series of 30 patients in whom 87% attained grade III with better facial reanimation. There is no statistical significance between the results of the JIGHFA group and Brudny's HFA in terms of attaining House-Brackmann grade III or better (using Fisher's Exact Test; $P = .74$) (Table XI; Fig. 7) It should be recalled that Brudny's series include only patients who have demonstrated facial reinnervation and who have participated in intensive EMG rehabilitation compared with the 67% of those JIGHFA patients who underwent EMG-monitored training. It is also unclear whether the different percentages of patients who underwent EMG rehabilitation therapy significantly affected the outcome of these groups, potentially unfounding the study results. The best way to clarify this problem will be with a future study comparing two groups of JIGHFA patients, one with and one without EMG rehabilitation.

Our JIGHFA results did not achieve "superb" results that May[15] reported in his series with 3 of 20 patients (15%), along with 13 patients within the "excellent" category (grade II; Table XI; Fig. 7). In his series, there were none rated good (III) and 4 patients (20%) were classified as fair (IV) or poor (V). In our JIGHFA series we were reluctant to categorize any of the grade II rated patients as superb (grade I) because we consider this to equal normal function, which includes vigorous emotional facial expression and forehead movement, whereas May 's "superb" patients have "some" emotional expression.

TABLE VII.
Application of Brudny Modification to HFA Subjective Percentile Rating of Normal Facial Function.

Subjective HFA Facial Function (% of normal side)	N (%)	Brudny-House-Brackmann Scale I–VI
0–25	5 (23)	V–VI
26–50	5 (23)	IV
51–75	7 (32)	III
76–100	5 (23)	II

TABLE VIII.
Ophthalmic Status After Hypoglossal Facial Anastomosis and Lateral Canthoplasty (N = 22).

	Yes	No
Satisfactory eye closure	17	5
Drooping eyebrow	7	14
Cosmetic problem	6	
Visual impairment result	3	
Drooping eyelid	5	17
Cosmetic problem	4	
Visual impairment result	2	
Synkinesis with eye closure	6	15
Ophthalmic irritation	10	11
Lubricant used	13	
Artificial tears used	11	
Protective windshield	2	
Corneal anesthesia	5	
Vision in ipsilateral eye		
Good	9	
Fair	9	
Poor	2	
Not usable	2	
Epiphora	3	19
Gustatory tearing	1	

Note the presence of severe (not usable or poor) visual difficulties in four patients (18.2%), all of whom had corneal anesthesia.

May used the JIGHFA with various supplementary procedures such as facial-facial cross grafting in 3 patients and temporalis muscle transposition in 21 patients, which may be sufficient augmentation to elevate the patients into a higher class. The facial-facial cross graft may be the only procedure that can account for improvement in appropriate synchrony of spontaneous emotional expression. Parenthetically, it should be recalled that many authors over the years have observed that ipsilateral primary reconstruction facial or interpositional facial nerve graft anastomosis has not been associated with successful facial movement with emotional expression.[2,17] May's report did not specify which ancillary procedure was used in the three patients with the superb results. Nevertheless, May emphasized that these "excellent results were due more to ancillary reanimation procedures than the effects of the interposition graft" in at least one of the cases of superb results. If we add together the number of the grade III and better results from our JIGHFA series (Table XI; Fig. 7), we observe that 83.3% (22% 61.1%) slightly exceeded May's total of 80% of grade III or better (15% 65%). As an alternative, it is possible that May's technique with supplementary procedures has elevated 80% of his patients to a full grade higher (i.e., to grades I and II as opposed to II and III in our JIGHFA series). Nevertheless, May's superb results (15%) certainly suggest that we need to be more supportive and encouraging of our patients with JIGHFA to consider supplementary procedures to enhance their facial reanimation.

In contrast to the HFA patients (from the above-cited series in Tables II and XI), there were three JIGHFA patients who had successful facial reinnervation despite lower cranial nerve deficits. Therefore it can be said that, except for hypoglossal paralysis, the JIGHFA technique can be used by patients with lower cranial nerve deficits, unlike the HFA procedure, which is contraindicated in those with such lower cranial nerve dysfunction. May[15] also reported that bilateral facial paralysis may be managed with JIGHFA, whereas a bilateral classical HFA procedure would induce bilateral lingual dysfunction. Our JIGHFA series did not have any patients with bilateral facial paralysis.

Hypertonia was present in most of the HFA series reviewed in the introduction, in 36.7% of our HFA respondents, and in 16.6% of our JIGHFA patients (Table IX), in contrast to none in May's series. Perhaps the patients in May's series had less axonal supply for reinnervation, since he also relied on ancillary reanimation procedures. It is believed that this reduced axonal supply in our JIGHFA patients also facilitated the EMG management of hypertonia compared with the replete axonal load from the completely transected hypoglossal nerve in HFA. Considerably more effort and EMG rehabilitation was required to subdue the hypertonia associated with HFA when compared with JIGHFA.

Hypotonia seemed to be more noticeable during the early period after dynamic facial reinnervation became visible in the JIGHFA patients compared with HFA patients. This weakness diminished with EMG feedback rehabilitation and time. While the author found that the best JIGHFA results equaled the best results observed in the HFA patients, several JIGHFA patients in grade III still had persistent mild asymmetry and hypotonia (Fig. 4B and C), which was not seen in May's series. Again, this may suggest the need for ancillary procedures to augment facial reanimation.

TABLE IX.
Problems Following Facial Reanimation Surgery.

	Hypertonia (%)	Synkinesis (%)
JIGHFA (N = 18)	3 (16.6)	8 (44.4)
HFA (N = 22)	8 (36.7)	6 (27.3)
	Speech Problems (%)	**Swallowing (%)**
JIGHFA (N = 18)	0	0
HFA (N = 22)	10 (45)	10 (45)

There is a significant difference between the two groups (HFA and JIGHFA) related to speech and swallowing problems (two-sided Fisher exact test: $P = .001$). Since the numbers are the same, the P value for each outcome is the same.

JIGHFA = jump interpositional graft hypoglossal facial anastomosis; HFA = hypoglossal facial anastomosis.

Synkinesis was present in 27.3% (6/22) of the patients responding to HFA questionnaire and 44.4% (8/18) of our JIGHFA patients, contrasted to May's series, which reported no patients with significant mass movement (Table IX). All of the prior HFA reports previously cited encountered synkinesis, especially with strong lingual activation.[16,17,22–30,34,35] Both techniques, HFA and JIGHFA, have the fundamental difficulty with axonal misdirection along distal facial tubules, which makes it difficult to account for May's absence of synkinesis in his JIGHFA series. Synkinesis certainly can be reduced in relation to the smaller axonal supply available for reinnervation in the JIGHFA patients, but it is probable that mass action of some extent will always be visible to the trained observer. As noted earlier, EMG rehabilitation can control synkinesis more readily in the JIGHFA patients, again, because of weaker or fewer areas of competing of muscle activity. Therefore it is likely that mass action will be less in the JIGHFA series but present, if not controlled with proper rehabilitation, or present when the patient is fatigued or inattentive.[30,93]

With the reduction of synkinesis, there should be more regionalization, movement of different areas of the face independently of each other. Again, this may be due to

TABLE X.
Hypoglossal Facial Anastomosis Questionnaire Response on Oral Symmetry (N = 22).

	Yes	No
At rest, is there good symmetry of the lips?	20	2
With movement is there good symmetry of the lips?	8	14
With smiling/laughter is there:		
Good symmetry?	0	
Fair symmetry?	15	
No symmetry?	7	
Is the lower lip/corner of the mouth:		
Weak?	15	
Flaccid?	3	

TABLE XI.
Facial Reanimation Results Comparing Hypoglossal Facial Anastomosis (HFA) by Questionnaire, and Brudny's HFA with Electromyographic (EMG) Rehabilitation, Jump Interpositional Graft Hypoglossal Facial Anastomosis (JIGHFA), May's Series of JIGHFA Using Brudny's Modification of House Brackmann Scale of Facial Paralysis.[15,30]

Class	HFA Questionnaire (N = 22) (%)	Brudny[30] EMG HFA (N = 30) (%)	JIGHFA (N = 18) (%)	May's[15] JIGHFA (N = 20) (%)
I				3 (15)
II	5 (23)	10 (30)	4 (22)	13 (65)
III	7 (32)	17 (57)	11 (61.1)	0
IV	5 (23)	3 (23)	2 (11.1)	3 (15)
V	5 (23)			1 (5)
VI				
Total	22	30	17	20

Note that May's JIGHFA results achieved highest rating with three patients. Nevertheless, when class III or better results are combined, Brudny's HFA series with EMG rehabilitation and our JIGHFA series followed a similar trend: 87% and 83.3%, which are comparable to May's results of 80%. The poorest results were with the HFA questionnaire patients in whom only 55% attained class IV or better. There is no statistically significant difference between the Brudny HFA and JIGHFA groups with regard to attaining House-Brackmann class III or better (Fisher exact test; $P = .74$ and chi-square).

the lower axonal volume available for misdirection to innervate competing muscle groups. EMG rehabilitation has been more successful in obtaining regional control and facial movement in the JIGHFA patients compared with the HFA patients. Regionalization can be enhanced with separate reanimating subsystems providing movement to discrete areas of the face (e.g., gold weight for implantation and neural reinnervation of only lower facial nerve branches).[66]

Ophthalmic problems were markedly less frequent in our JIGHFA (11.1%) and May's series (0%) when compared with the reports from our HFA questionnaire and the Pensak's HFA study (29%)[15,29] (Tables III and IV). Although there was eye discomfort in two patients and one patient had corneal abrasion in the JIGHFA series, no one had visual impairment. Poor vision and loss of vision were noted by 18.2% of the respondents in our HFA series, which exceeded the 11% Pensak identified in Glasscock's HFA series. Because the response rate from the HFA questionnaire group was less than 50% (22 of 48), there is a potential for bias which may preclude meaningful difference between this HFA group and Pensak's concerning visual impairment (Tables III and VIII). The fewer ophthalmic complications in patients reinnervated with JIGHFA may be due more to ancillary procedures (i.e., gold weight implantation) specifically dedicated to management of lagophthalmos and fewer patients with corneal anesthesia in our series. It should be recalled that there was a potential problem with the gold weight upper lid implantation when it was rejected in three patients. However, orbicularis oculi reinnervation was established by then, so further ophthalmic procedures were not required.

Limited mimetic movement (facial expressions) was present in our JIGHFA (22.2%) and HFA (0%) series, which was certainly similar to the reports reviewed earlier. Spontaneity of expression, albeit minimal with some asymmetry, was observed by Brudny[30] in 30% of his series of HFA patients after EMG rehabilitation. It is acknowledged that there is certainly a sense of reduced movement with lingual activity associated with labile changes, but marked asymmetry still accompanies vigorous deep involuntary mimetic activity. This is particularly noticeable around the mouth, which has a bilateral muscle supply that normally functions synergistically and symmetrically. With only one side of the mouth undergoing spontaneous dynamic movement, there will be contralateral displacement of this "axial" structure highlighting the incomplete mimetic activity of the reanimated side. As noted earlier, May's JIGHFA series with successful mimetic movement may be partial at best because he defines superb as "some mimetic (spontaneous expression) movement" which undoubtedly has an element of asymmetry when compared with contralateral normal facial excursion.

Lack of mimetic movement following direct primary facial anastomous or facial interpositional grafts have been reported by earlier authors such as Ballance,[17] Coleman,[22] and, later, by Stennart.[2] Alexander[16] recalled that his "best" hypoglossal facial function occurred in two blind patients who had minimal or no contralateral movement

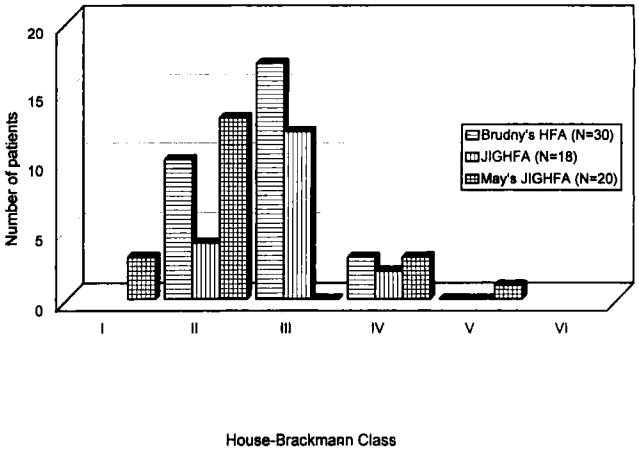

Fig. 7. Facial reanimation results by House-Brackmann scale after hypoglossal facial anastomosis.

which was thought to be associated with blindness. It is possible that some mimetic movements are learned as part of nonverbal human social interaction and that other "lower reflex" mimetic activity is also a function of some type of unappreciated afferent loop via facial nerve and/or trigeminal pathways which are disrupted by facial nerve injury. Further investigation of these concepts obviously is needed before mimetic dysfunction can be understood.

This retrospective study involving subjective questionnaire response from one series of patients with HFA partially represents a unique patient group from a larger series of 48 patients. This subjective series offers information from the perspective of the anonymous patient. Some of this material might not be easily accessible because of patient bias toward pleasing the physician. On the other hand, there was less than 50% response (22/48) in this subjective series, which can allow the potential for a bias. Patients with particularly good results might be particularly inclined to return the questionnaire, or alternatively, those particularly unsatisfied might be more eager to respond to the questionnaire. The direction of the bias cannot be determined with this study. Limitations and shortcomings (vision problems, dysarthria, dysphagia) of the HFA procedure may be revealed instead of being overlooked by treating physicians, who may be most focused on evidence of reanimation movement at the exclusion of unsolicited information about potential problems.

This subjective series alone cannot be compared with the JIGHFA series in which results were evaluated with a more objective criteria (modification of House-Brackmann scale to grade facial paralysis (Table I). Instead. we attempted to determine whether the subjective HFA series had results comparable to the prior HFA literature and to Brudny's series, whose HFA results were stratified by objective criteria that were also the same for our JIGHFA subjects. If our HFA results were comparable to Brudny's series, then it might be possible to compare with some validity some of our HFA results with those of the JIGHFA series. We also retrospectively attempted to apply an internal check of answer consistency from the respondents by comparing two sets of answers to roughly the same question: adjectival categorization of facial reanimation movement "good, fair, poor and none" to quarterly percentage score relative to normal contralateral side. As noted, there was a rough correlation suggesting a consistency in the respondents' reports (Tables VI and VII; Fig. 5). After extrapolation of percentage quartile data from the subjective questionnaire group to House-Brackmann grading scale, our HFA series was also compared with the Brudny's HFA series with results graded along by House-Brackmann scale (Table XII). The HFA questionnaire group had its results more scattered, particularly in grade IV and V, which may be explained by the reduced number of EMG participants.

The JIGHFA series with the House-Brackmann grading was compared with the Brudny HFA series, which used the same House-Brackmann scale (Table XII). There is a reasonableness in comparing these results based on the same criteria. After examining May's criteria in his JIGHFA report, we can see that it is most likely based on grading scale similar to the House-Brackmann system (I = superb to VI = no function). Therefore it was concluded that reanimation results from Brudny's HFA series and the two JIGHFA series may be compared, since they are, indeed, using similar criteria from the House-Brackmann grading system (Table XI; Fig. 7).

While we attempted to stratify our observations along discernable objective criteria, there are significant limitations in the rigor of these data because of the informality of experimental design in this retrospective study. For example, the patients were not randomly chosen, nor was their surgical procedure of facial reanimation (HFA vs. JIGHFA) randomized. Most patients preferred to retain lingual function and avoid hypertonia, especially after talking with someone suffering from severe hypertonia and contractures following the classic HFA. The patients were not matched in terms of age, tumor size, disease, presence of lower cranial nerve deficits, or EMG rehabilitation therapy. Since the trained observer knew which surgical procedure was performed, these ratings did not occur in an unbiased double-blind setting.

CONCLUSION

We presented our experiences with 22 patients with HFA and its derivative facial reanimation technique, JIGHFA with gold weight upper lid implantation, in 18 patients, 12 of whom also had EMG feedback rehabilitation. We found that in properly selected patients, the JIGHFA technique is capable of achieving substantial facial reinnervation (grade III or better) in 83.3% of the patients, not unlike the success associated with its predecessor, HFA. In contrast to HFA, JIGHFA can be used in patients with concomitant lower cranial nerve dysfunction (except nerve XII) and with bilateral facial paralysis. Three of our patients with lower cranial nerve impairment had no oropharyngeal symptoms after JIGHFA surgery. While May found that best results with JIGHFA occurred when the duration of facial paralysis was less than 12 months, we did not have enough patients to establish an optimal time period beyond which JIGHFA reanimation would not be effective.

TABLE XII.
Comparison of Hypoglossal Facial Anastomosis (HFA) by Questionnaire to Jump Interpositional Graft Hypoglossal Facial Anastomosis (JIGHFA) and HFA Results by Trained Observers Using Brudny Modification of House-Brackmann Scale for Facial Paralysis.

Class	HFA Questionnaire (N = 22) (%)	JIGHFA (N = 18)	HFA (Brudny) (N = 30)
I	0		
II	5 (23)	4 (22)	10 (30)
III	7 (32)	11 (61.1)	17 (57)
IV	5 (23)	2 (11.1)	3 (3)
V	5 (23)		
VI			
Total	22	17	30

Note that all of Brudny patients underwent EMG biofeedback compared with 82% from the subjective HFA group and 67% from the JIGHFA group. Note that the subjective group rated itself throughout the two lower classes IV and V in contrast to other groups, which had very few or no patients classified in these categories. One patient in the JIGHFA group is lost to follow-up, causing the total to be 17.

We found that hypertonia occurring in our JIGHFA patients was considerably milder and far easier to manage with EMG feedback when compared with hypertonia after HFA. Synkinesis was less severe (but not necessarily less frequent) in patients with JIGHFA because of fewer axons being misdirected. Regionalization was also better because of fewer (misdirected) axons innervating conflicting or simultaneous muscle groups. Separate subsystems of reanimation should be able to enhance regeneralization.

In our JIGHFA series, mimetic activity was not more symmetric during vigorous activity of the contralateral normal side when compared with HFA. No JIGHFA patients had problems of speech, chewing, or swallowing compared with (45%) of patients who complained of these difficulties following HFA.

Lagophthalmos was well managed in our JIGHFA series. However, there were no patients with corneal anesthesia, unlike our series with the HFA group. Interestingly, three patients had no ocular problems after removal of gold weight because of successful dynamic facial reinnervation from the JIGHFA technique.

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