Otology & Neurotology
33:1439–1444 © 2012, Otology & Neurotology, Inc.

Rehabilitation of Central Facial Paralysis With Hypoglossal-Facial Anastomosis

C. Eduardo Corrales, Richard K. Gurgel, and Robert K. Jackler

Department of Otolaryngology–Head and Neck Surgery, Stanford University School of Medicine, Stanford, California, U.S.A

Objective: To evaluate the ability of hypoglossal-facial nerve anastomosis to reanimate the face in patients with complete nuclear (central) facial nerve palsy.
Study Design: Retrospective case series.
Setting: Tertiary academic medical center.
Patients: Four patients with complete facial nerve paralysis due to lesions of the facial nucleus in the pons caused by hemorrhage due to arteriovenous or cavernous venous malformations, stroke, or injury after tumor resection.
Intervention: All patients underwent end-to-end hypoglossal-facial nerve anastomosis.
Main Outcome Measures: Facial nerve function using the House-Brackmann (HB) scale and physical and social/well-being function using the facial disability index.
Results: The mean age of the patients was 53.3 years (range, 32–73). There were 3 female and 1 male patients. All patients had preoperative facial function HB VI/VI. With a minimum of 12 months' follow-up after end-to-end hypoglossal-facial anastomosis, 75% of patients regained function to HB grade III/VI, and 25% had HB grade IV/VI. Average facial disability index scores were 61.25 for physical function and 78 for social/well-being, comparable to results from complete hypoglossal-facial anastomosis after peripheral facial nerve palsy after acoustic neuroma resection.
Conclusion: Patients with nuclear facial paralysis who undergo end-to-end hypoglossal-facial nerve anastomosis achieve similar degrees of reanimation compared with those with peripheral facial nerve palsies. This raises the intriguing possibility that reinnervation may also be of benefit in patients with the vastly more common facial dysfunction because of cortical stroke or injury. **Key Words:** Facial nucleus—Facial paralysis—Facial reanimation—Hypoglossal-facial anastomosis—Stroke.

Otol Neurotol **33:**1439–1444, 2012.

Facial paralysis is a challenging problem for both patients and the physicians who seek to help them (1). It has functional, aesthetic, psychological, and social consequences. Paralysis of the muscles of facial expression may result from injury to the facial nerve peripherally, the facial nucleus in the brainstem, or the motor cortex. Most causes of peripheral facial paralysis are due to nerve damage from trauma, infection, metabolic disorders, birth trauma, neoplasms, iatrogenic and idiopathic causes (1). Cortical paralysis is most often due to trauma or stroke (2). In the population of those with facial paralysis, peripheral and cortical facial palsies predominate, whereas brainstem lesions are relatively uncommon. Nuclear facial palsy may come from stroke, vascular malformation, or tumor (3).

As motor nuclei for the VIth and VIIth cranial nerves and descending cortical motor tracts cluster in proximity, most brainstem lesions which result in nuclear facial palsy involve numerous accompanying deficits (Fig. 1). Causes of brainstem ischemia include hemorrhage at the facial nucleus region secondary to arteriovenous malformations, central (lacunar) infarct secondary to small vessel disease (microangiopathic occlusion of an arteriole), and injury to the lateral pontine region from pontine-cavernous and petroclival tumors (Fig. 2, A and B) (3–5).

Anastomosis of adjacent cranial nerves has been a method of restoring facial function since the late 19th century (6). Although techniques have been described grafting Cranial Nerves V, IX, X, XI, and XII, the hypoglossal nerve (XII) is the most frequently used today. Technical variations have been developed including end-to-end, end-to side, and partial nerve crossover (7,8).

There has been substantial progress in surgical techniques to improve the paralyzed face (8). Nearly the entire published experience reported in the literature relates to patients with facial paralysis because of dysfunction of facial nerve itself. The goal of this study was to evaluate the functional outcomes after hypoglossal-facial (XII-VII) nerve anastomosis in patients with facial palsies because of damage of the facial mid-pontine nucleus. Typically,

Address correspondence and reprint requests to Robert K. Jackler, M.D., 801 Welch Road Stanford, CA. 94305-5739; E-mail: rjackler@ohns.stanford.edu
The authors disclose no conflicts of interest.

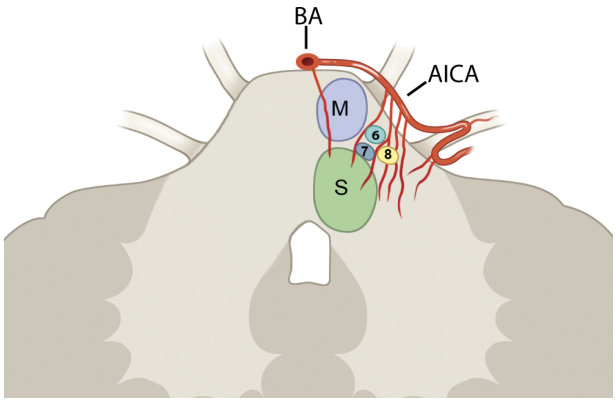

FIG. 1. Cross-sectional view of the mid-pons showing the brainstem nuclei for the facial (7), audiovestibular (8), and abducens nerves (6) as well as motor (M) and sensory (S) tracts. AICA indicates anterior inferior cerebellar artery; BA, basilar artery. Note: The authors grant permission to reproduce this illustration in the journal in all of its versions (paper and electronic) but reserve copyright ownership privileges for future use.

patients with nuclear facial palsy have other cranial nerve neuropathies. This raises the question of optimal treatment and rehabilitation for complete nuclear versus peripheral facial nerve palsy.

METHODS

Patients with complete nuclear facial palsies were retrospectively identified from May 2004 to October 2011. Institutional review board was formally waived according to our institutional guidelines because of the limited number of involved patients. Severity of facial nerve paralysis was evaluated with the House-Brackmann (HB) scale (9). Demographic and clinical data were collected for each patient, including age, length of time from event to reinnervation procedure, preoperative and postoperative HB scores, follow-up period, and other cranial nerve deficits at initial presentation. We also noted adjunct procedures performed including gold weight placement, canthoplasty (medial and lateral), vocal cord medialization procedures, and need for feeding tube.

All patients underwent end-to-end XII to VII anastomosis as their primary reinnervation procedure. All patients completed the facial disability index (FDI) questionnaire (10). The FDI is a subjective self-reported, disease-specific questionnaire that gauges the patients' mastication, deglutition, communication, labial and lip mobility, emotional adjustments, and social integrations. The FDI measures physical disability and related social and emotional health of patients with facial nerve disorders. It is scored using a 100-point scale, with higher scores representing less impairment and handicap. Additionally, all patients were asked at 12 months' follow-up to subjectively evaluate whether facial function was worth the associated partial tongue weakness.

RESULTS

Four patients with nuclear facial paralysis were identified, all of who had HB VI/VI facial function after their central insult. The mean age of the patients at the time of XII to VII anastomosis was 53 with a range of 32 to 73 years. There were 3 female and 1 male patients. The mean time (in months) from brainstem event to reinnervation procedure was 12.5 months (range, 8–15 mo). HB scores were taken at every clinic visit during the follow-up period. Patient characteristics, cause of the nuclear facial

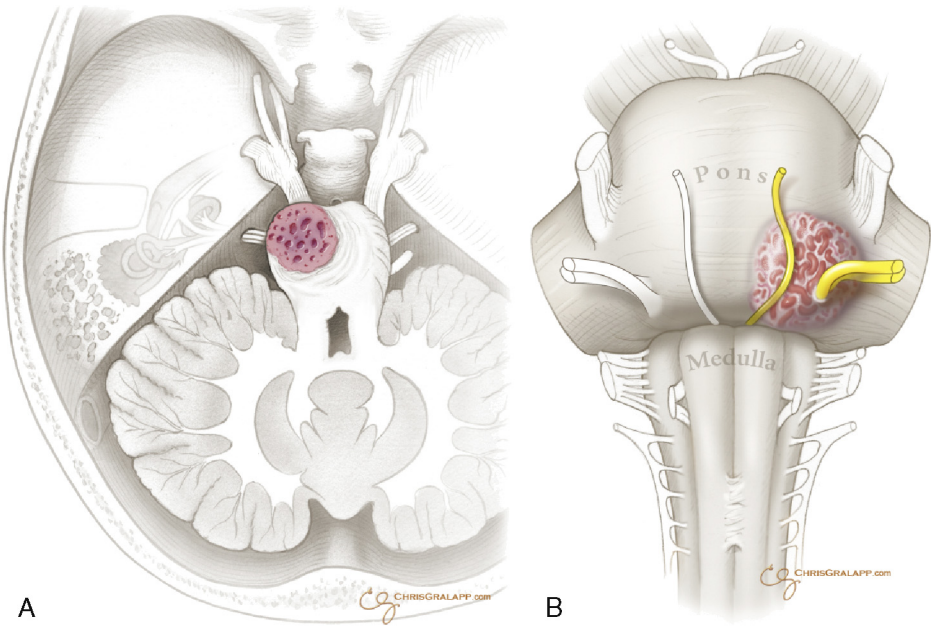

FIG. 2. *A*, Illustration of a large pontine vascular malformation on axial view. *B*, Ventral view demonstrating a pontine vascular malformation at the cerebellopontine angle and its relationship to Cranial Nerves VI, VII, and VIII. Note: The authors grant permission to reproduce this illustration in the journal in all of its versions (paper and electronic) but reserve copyright ownership privileges for future use.

TABLE 1. *Patient's age at time of nerve graft, cause of nuclear facial palsy, and other cranial nerve deficits*

Patient	Age (yr) at time of nerve graft	Cause	Cranial nerve deficits
1	32	Cystic cavernous malformation in the central-lateral pons	V, VI, VII, VIII
2	65	Vascular malformation with hemorrhage in the posterolateral pons and middle cerebellar peduncle	VI, VII, VIII, X
3	73	Vascular malformation with hemorrhage into the lateral pons	V, VI, VII, VIII, IX
4	42	Hemorrhage into the lateral pons secondary to infarct.	V, VI, VII, VIII, IX, X

palsy, and comorbid cranial nerve deficits are summarized in Table 1. Average follow-up for all patients was 36.5 months (range, 18–80 mo).

All patients underwent gold weight placement during their rehabilitation period. Two patients (Patients 1 and 3) had additional ophthalmic procedures, including superior and inferior rectus lateral transposition and lateral and medial canthoplasty. Patient 4 had severe cerebellar edema with herniation after removal of the petroclival meningioma requiring ventilator support. Table 2 summarizes additional surgical procedures performed for other cranial nerve neuropathies.

Table 3 summarizes the preoperative and postoperative HB score and the FDI. The outcomes of end-to-end XII to VII nerve anastomosis were as follows: 75% (3 patients) HB grade of III and 25% (1 patient) HB grade of IV.

The average for the physical function score of the FDI was 61.25 (±11.08). The social/well-being aspect was 78 (±11.54). There was no appreciable difference in the reported FDI scores compared with patients who had complete facial palsy and XII to VII anastomosis after acoustic neuroma excision (8). Additionally, all 4 patients were asked at their 12-month follow-up if the tradeoff of tongue weakness for improved facial function was worth it to them. Each stated that they were glad they had undergone the procedure.

Patient 1

Patient 1 is a 29-year-old man who presented to the neurosurgery clinic after 6 months of progressive dysphagia and left arm and leg weakness and hypesthesia. His neurologic examination was significant for decreased sensation of the left face, arm, and leg. He had no cranial neuropathies. Magnetic resonance imaging (MRI) revealed a right-sided, cystic pontine vascular malformation (Fig. 3A). Because of signs of recent hemorrhage, the patient was taken to the operating room for a subtemporal approach, cyst decompression, and partial resection of the pontine malformation.

Postoperatively, the patient had right frontal branch weakness but otherwise recovered well until 2 months after his surgery, at which time his symptoms of left-sided numbness rapidly progressed as well as new ataxia and dysarthria. Repeat imaging showed new hemorrhage into the pons from the vascular malformation, and the patient returned to the operating room for a suboccipital craniotomy with resection of the vascular malformation. A 2.5-cm vascular malformation was found in the right pons extending from the floor of the fourth ventricle to the ventral surface of the pons. The malformation was composed of friable, thin-walled vascular channels, and there was hemosiderin staining of the surrounding brainstem. Postoperatively, the patient was hemiparetic on the left and had a complete right facial palsy.

After his acute recovery, the patient was referred to our neurotology clinic for facial reanimation. The patient had a HB VI/VI facial paralysis but intact lower nerve function and normal swallowing. The patient was given 15 months postoperatively to evaluate for spontaneous facial recovery, after which time, he underwent a XII to VII end-to-end anastomosis. At the patient's last clinic visit 19 months after surgery, his facial function had progressed to a HB III/VI.

Patient 2

Patient 2 is a 63-year-old woman who presented to the emergency department with sudden onset left facial paralysis and left-sided hearing loss. The patient had a past medical history significant for hypertension with systolic blood pressures typically in the 150s, although in the 190s on admission to the emergency room. MRI revealed a hemorrhagic lesion in the left posterolateral pons with associated vasogenic edema (Fig. 3B).

After remaining neurologically stable, the patient was taken to the operating room for a suboccipital craniotomy with resection of vascular malformation and evacuation of brainstem hematoma. A 2.4-cm area of subacute hematoma and associated vascular malformation were found in the left mid pons, extending to the left middle cerebellar peduncle. The vascular malformation itself measured approximately 1.0 cm and was composed of thin-walled vascular channels that were partly thrombosed.

TABLE 2. *Adjunct surgical procedures for each patient*

Pt	Gold weight	Adjunct procedures
1	Yes	Superior rectus lateral transposition Inferior rectus lateral transposition Botox medial rectus muscle
2	Yes	None
3	Yes	Lateral and medial canthoplasty with lateral tarsal strip
4	Yes	Tracheostomy Gastrostomy tube Bilateral TVC medialization injections Coronoidectomy

Patients 1 and 3 had additional ophthalmic procedures including superior and inferior rectus lateral transposition and lateral and medial canthoplasty. Patient 4 had severe cerebellar edema with herniation after removal of the petroclival meningioma requiring ventilator support followed by tracheostomy and gastrostomy tube placement.

TABLE 3. *Preoperative and postoperative facial nerve function and facial disability index*

Pt	Preoperative House-Brackmann	Postoperative House-Brackmann (months after surgery)	Facial disability index	
			Physical function	Social/well-being function
1	VI/VI	III/VI (18)	75	80
2	VI/VI	IV/VI (18)	65	64
3	VI/VI	III/VI (80)	50	92
4	VI/VI	III/VI (30)	55	76

Three patients had House-Brackmann Grade III, 1 patient had House-Brackmann Grade IV. The average for the physical function part of the facial disability index was 61.25 (range, 50–75). The social/well-being aspect was 78 (range, ± 11.54).

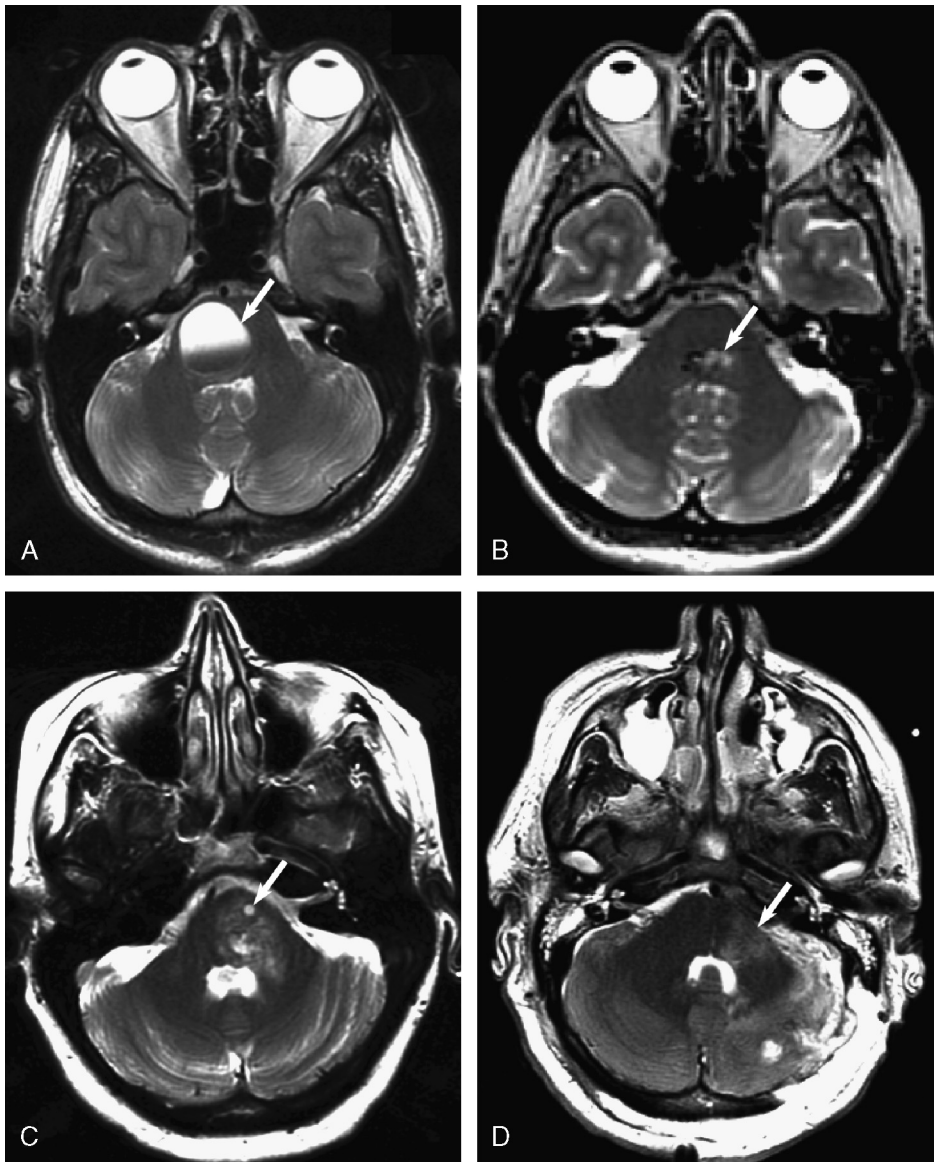

FIG. 3. Axial MRIs from all 4 patients. *A*, T2-weighted MRI showing arteriovenous malformation with hemorrhage seen as a blood-fluid level. *B*, T2-weighted MRI showing a vascular malformation with hemorrhage in the posterolateral pons and middle cerebellar peduncle. *C*, T2-weighted MRI showing a vascular malformation with hemorrhage into the lateral pons. *D*, T2-weighted MRI showing a large peduncular infarct extending into the mid pons.

The patient was subsequently referred to the neurotology clinic, and a HB VI/VI facial palsy was confirmed. After 12 months of observation for spontaneous return of facial function, the patient underwent a XII to VII end-to-end anastomosis. At the patient's last clinic visit 18 months after surgery, she was regaining facial tone and had progressed to a HB IV/VI.

Patient 3

Patient 3 is a 67-year-old woman who presented to the neurosurgical clinic with diplopia and was found to have a right-sided abducens palsy. MRI showed a pontine vascular malformation with a brainstem hemorrhage (Fig. 3C). She was followed for 6 months but developed a second pontine hemorrhage with patchy left-sided numbness. Because of the recurrent bleeding episodes, the patient underwent a left suboccipital craniotomy and resection of her vascular malformation. Postoperatively, the patient had bilateral abducens palsy and a left HB VI/VI facial palsy.

In the absence of any signs of spontaneous recovery of facial nerve function, the patient underwent XII to VII end-to-end anastomosis 10 months after the initial facial paralysis. One year postoperatively, the patient had progressed to a HB III/VI that remained stable for many years. At 5 years postoperatively; however, she had progressive bilateral motor and sensory decline in the trigeminal and facial distribution because of a bulbar palsy related to her brainstem vascular disorder.

Patient 4

A 38-year-old woman presented to the neurosurgical service with severe headache, left-sided hearing loss, and progressive ataxia. MRI revealed a 3.5-cm meningioma of the left petrous face. The patient also had a Chiari malformation with 1.8 cm of tonsillar descent and severe cervical syringomyelia. The patient underwent a bilateral suboccipital craniectomy with C1 laminectomy and a subtotal resection of the petrous face meningioma. Because of the crowding of the posterior fossa from both the meningioma and Chiari, the surgery was complicated by significant cerebellar swelling, requiring partial resection of the cerebellar hemisphere. Intact baseline somatosensory and motor evoked potentials were present at the conclusion of the case. The patient awoke with a HB VI/VI facial palsy. Postoperative imaging revealed a cerebellar peduncular infarct with extension into the pons (Fig. 3D) in the territory of the facial nucleus.

The patient was referred to the neurotology clinic for facial reanimation, and after 14 months of observation for return of function, the patient underwent a XII to VII end-to-end anastomosis. At her last follow-up visit nearly 3 years after surgery, her facial nerve function had progressed and remained at a HB III/VI.

DISCUSSION

Facial nerve injury and paralysis have significant functional and cosmetic ramifications. The face is a focal point for communicating and expressing emotion. Facial nerve function is necessary for eye protection, chewing, swallowing, and speech articulation. The most common cause of peripheral FN paralysis is mononeuritis, which recovers at least partially in the vast majority of cases. Repair is most often indicated in trauma, followed by neoplasms and iatrogenic injury (11). Current treatment strategies for repairing the injured FN include direct surgical repair and interposition nerve grafting. When the proximal facial nerve is not available for reconstructive use, facial reanimation procedures become necessary. Although a variety of strategies are in use, crossover anastomosis of an adjacent cranial nerve, most commonly the hypoglossal, is a mainstay.

Complete facial paralysis because of central injury to the facial motor nucleus is a relatively rare condition. It is associated with intracerebral hemorrhage, arteriovenous malformations, stroke, or brainstem tumors. Such lesions involving the mid pons have a high mortality. For example, intracerebral hemorrhage has the highest mortality of all cerebrovascular events (3). Mortality rates have been reported between 35% and 52%, with half of the patients dying within the first 48 hours. An acute ischemic pontine infarction has been shown to be an independent risk factor for deteriorating neurological function (12). Because of this high morbidity and mortality of brainstem infarcts, it is rare to treat patients who survive the insult and are left with nuclear facial palsies.

The key outcomes of reinnervation procedures in facial nerve palsy include symmetric resting muscle tone, adequate eye closure for corneal protection, and oral competence. When considered in aggregate, these indicators would be equivalent to a HB score of III/VI. Although this degree of facial function is certainly imperfect and leaves a clearly discernable cosmetic deficit, it still represents a highly consequential improvement compared with flaccid paralysis and is much appreciated by patients. In a comprehensive, systematic search of the English language, Medline literature since 1966, we were unable to identify facial nerve outcomes pertaining to patients with complete nuclear facial nerve palsies.

Patients presenting with nuclear facial palsy also may have other cranial nerve neuropathies including ophthalmoplegia, hearing impairment, facial sensory disturbance, dysarthria, dysphonia, dysphagia, and aspiration. The patients in this study had a high rate of comorbid cranial neuropathies (Table 1). Three (75%) of 4 patients had concurrent lower cranial nerve palsies at presentation. Repair of facial nerve palsies consists of anastomosing the facial nerve end to a functional motor donor nerve, usually the hypoglossal nerve. If the ipsilateral hypoglossal nerve, nucleus, or controlling cortex is injured, then XII to VII anastomosis will fail because of insufficient neuronal contribution. Moreover, for patients with impaired swallowing because of lower cranial neuropathies, hemiplegia of the tongue because of a hypoglossal-facial procedure may worsen their dysphagia. In this scenario, success has been reported in using other available motor nerves, such as the masseteric-facial anastomosis (13). Partial hypoglossal crossover, which may spare tongue

movement, has also been recommended by some (8). More importantly, in contrast to isolated facial paralysis, patients with pontine lesions typically have a number of other associated disabilities accompanying their palsy. The improved facial tone and movement can have an important psychological benefit in patients coping with a number of serious and irremediable neurologic deficits.

In a 20-year study by Catli et al. (14), outcomes with the hypoglossal-facial anastomosis showed 71.4% of patients reaching a HB score of III and 28.6% reaching a score of IV at 3 years. These results are comparable to this study, with 75% of patients reaching a HB grade III and 25% a HB IV.

The FDI gives the clinician input about the disability and social and emotional well-being of patients (10). Our patient's physical function and social/well-being scores of the FDI were comparable to other published reports (8). Our results demonstrate similar outcomes for end-to-end XII to VII anastomosis in patients with nuclear facial palsy compared with peripheral facial nerve paralysis. This also corroborates that all 4 patients felt it worthwhile to have had undergone the procedure.

In summary, because of the high morbidity and mortality associated with pontine infarcts and nuclear injury, it is rare to treat patients with complete nuclear facial palsies. Hypoglossal-facial nerve anastomosis can partially restore facial motor tone and function. Patients with complete nuclear facial palsy who undergo hypoglossal-facial nerve anastomosis have similar HB scores compared with those with peripheral facial nerve palsies who undergo the same reanimation procedure. Furthermore, in patients presenting with complete nuclear facial palsy with other associated cranial nerve deficits, their social and emotional health is comparable to patients with peripheral facial nerve palsies after repair.

The observation that hypoglossal-facial anastomosis is of substantial benefit in brainstem nuclear lesions raises the intriguing possibility that it might be of value in the much more sizeable population of patients who experience central facial palsy because of cortical lesions such as those associated with stroke in the territory of the middle cerebral artery (MCA) or anterior cerebral artery (ACA). A clinical-radiologic study by Cattaneo et al. (2) describes the incidence of facial palsy caused by strokes to the primary motor cortex, the anterior cingulate and mesial frontal cortex in both MCA and ACA territories, clearly demonstrating a higher rate of cortical facial palsies compared with nuclear facial palsies. However, the article did not comment on how these patients with cortical facial palsies were rehabilitated.

REFERENCES

1. Benecke JE Jr. Facial paralysis. *Otolaryngol Clin North Am* 2002; 35:357–65.
2. Cattaneo L, Saccani E, De Giampaulis P, et al. Central facial palsy revisited: a clinical-radiological study. *Ann Neurol* 2010;68:404–8.
3. Santalucia P. Intracerebral hemorrhage: medical treatment. *Neurol Sci* 2008;29:S271–3.
4. Novy J, Michel P, Poncioni L, et al. Isolated nuclear facial palsy, a rare variant of pure motor lacunar stroke. *Clin Neurol Neurosurg* 2008;110:420–1.
5. Nanda A, Javalkar V, Banerjee AD. Petroclival meningiomas: study on outcomes, complications and recurrence rates. *J Neurosurg* 2011; 114:1268–77.
6. Shah SB, Jackler RK. Facial nerve surgery in the 19th and early 20th centuries: the evolution from crossover anastomosis to direct nerve repair. *Am J Otol* 1998;19:236–45.
7. Campero A, Socolovsky M. Facial reanimation by means of the hypoglossal nerve: anatomic comparison of different techniques. *Neurosurgery* 2007;61:41–9; discussion 9–50.
8. Lin V, Jacobson M, Dorion J, et al. Global assessment of outcomes after varying reinnervation techniques for patients with facial paralysis subsequent to acoustic neuroma excision. *Otol Neurotol* 2009;30:408–13.
9. House JW, Brackmann DE. Facial nerve grading system. *Otolaryngol Head Neck Surg* 1985;93:146–7.
10. VanSwearingen JM, Brach JS. The facial disability index: reliability and validity of a disability assessment instrument for disorders of the facial neuromuscular system. *Phys Ther* 1996;76:1288–98; discussion 98–300.
11. Vlastou C. Facial paralysis. *Microsurgery* 2006;26:278–87.
12. Miyamoto N, Tanaka Y, Ueno Y, et al. Demographic, clinical, and radiologic predictors of neurologic deterioration in patients with acute ischemic stroke [published online ahead of print September 7, 2011]. *J Stroke Cerebrovasc Dis*.
13. Biglioli F, Frigerio A, Colombo V, et al. Masseteric-facial nerve anastomosis for early facial reanimation. *J Craniomaxillofac Surg* 2012;40:49–55.
14. Catli T, Bayazit YA, Gokdogan O, et al. Facial reanimation with end-to-end hypoglossofacial anastomosis: 20 years' experience. *J Laryngol Otol* 2010;124:23–5.